3rd INTERNATIONAL RENAL PATHOLOGY CONFERENCE
10-12th February 2017

ABSTRACT BOOK

Jawaharlal Nehru Auditorium, All India Institute of Medical Sciences, New Delhi
Dr Lal PathLabs is now the 1st private laboratory in India to introduce Electron Microscopy.

**DIAGNOSTIC APPLICATIONS IN RENAL PATHOLOGY**

- Proteinaceous deposits
- Immune complexes (seen as electron dense deposits)
- Subendothelial granular deposits in kappa light chain disease
- Amyloid fibrils · Cryoglobulins · Other fibrillary deposits (non-amyloidotic fibrillary glomerulopathy)

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MESSAGE

I am happy to learn that Department of Pathology, All India Institute of Medical Sciences is organizing the 3rd International Renal Pathology Conference and XIIth Annual Conference of Indian Society of Renal & Transplant Pathology from 09-12 February, 2017 at All India Institute of Medical Sciences, New Delhi. It is a great pleasure for me to welcome the leading experts from around the world and India to this premier medical institute who are participating in this event. The field of Renal and Transplant Pathology is advancing in a rapid pace resulting in change in the diagnostic practice. Understanding of recent advances and updating of the knowledge is extremely important for early accurate diagnosis and patient management. This conference will create a common discussion forum for experts from pathology, nephrology, transplant surgery, immunology and allied fields to communicate and exchange knowledge and views resulting in development of awareness and collaboration in this field.

I sincerely hope that this conference will be of great success and pave the pathway for improvement of practice of renal pathology.

(Prof. Balram Airan)
Director (Officiating)
Message

It gives me immense pleasure and joy to welcome all the speakers and delegates to the 3rd International Renal Pathology Conference and 12th Annual Conference of Indian Society of Renal & Transplant Pathology, 9 – 12 February 2017 at All India Institute of Medical Sciences, New Delhi. I congratulate Renal Pathology Society (RPS) and Indian Society of Renal & Transplant Pathology (ISRTP) for their joint effort in organizing this event. It is heartening to know that this event has generated an overwhelming response with delegates participating from 15 countries making it a truly global event. It will be a memorable event for renal pathologists, nephrologists, transplant surgeons, immunologists and all delegates from the allied fields.

This International conference and Workshop will provide an excellent opportunity for acquiring an overview of recent advances in Renal and Transplant Pathology. I wish to thank all the faculty members and staff of my Department for their dedicated efforts in organizing this event.

I hope all the delegates will have an enriching experience. I urge all the members to have fruitful discussions and wish the conference every success.

(Dr. S.K. Panda)

Prof. S.K. PANDA
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Ansari Nagar, New Delhi-110029
As the chairperson of the local organizing committee of 3rd International Renal Pathology Conference (IRPC2017) and XII Annual Conference of Indian Society of Renal Transplant Pathology (ISRTPCON2017), I welcome all the delegates, faculties and chairpersons who are participating in this great event. I take this opportunity to thank all the delegates and faculties coming from 24 countries across the world for this conference. I hope you will have a pleasant stay at Delhi, the capital city of India with a glorious past which traces its history of Mahabharata and journey through the Mughal and British Rules.

I sincerely hope this two and a half days of academic event and the preconference workshop will help all of us to learn, share and rejuvenate our ideas in the area of diagnostic renal and transplant pathology. As the pathology diagnosis is the foundation of Clinical Nephrology practice, we have planned the sessions with an emphasis on clinico-pathologic correlation. We are grateful for continuous guidance and help of Renal Pathology Society, specially Dr. Anthony Chang and Dr. Cynthia Nast for planning and execution of this international event. We expect this event with faculties from different parts of the world will encourage us to introduce the latest technologies and molecular markers in our diagnostic practices as well as enhance the collaborations across different centers, strengthening the diagnostic renal pathology practice in the global scenario.
Message from the Renal Pathology Society President

On behalf of the Renal Pathology Society, I welcome you to the 3rd International Renal Pathology Conference in New Delhi. The 1st and 2nd International Renal Pathology Conferences were held in La Coruna, Spain (2010) and Tsukuba, Japan (2015) and we are pleased to join efforts with the Indian Society of Renal and Transplantation Pathology to return the 3rd conference to the Asian continent, especially for our international members.

The meeting will deliver state-of-the-art presentations on native and transplant kidney diseases by internationally known speakers. We hope that this conference will inspire, create, and renew collaborations and friendships among the renal pathologists, nephrologists, and kidney researchers in attendance, which will flourish for decades.

We particularly wish to thank our local hosts and organizing team, as this 2.5 day conference represents the preparation and tremendous efforts of many individuals over several years. Finally, we also thank you as the participant, as your enthusiasm, participation, and intellectual curiosity are essential for the success of this conference.

Anthony Chang MD
Professor of Pathology,
Cedars Sinai Medical Centre,
Los Angeles, CA, USA
XIIth Annual Conference of Indian Society of Renal & Transplant Pathology (ISRTPCON 2017) and Pre conference workshop

Venue - Jawaharlal Nehru Auditorium, AIIMS, New Delhi

Thursday, 9th February, 2017

8.00 AM - Registration
8.45 AM - Opening remarks by the past and present Presidents of ISRTP

Session I. Basics of Renal Biopsy Processing & Interpretation

Moderators: Dr. R.K.Gupta, Dr. Arvind Bagga & Dr. Sudipta Roy

09.00 – 09.30 AM – Image guided renal biopsy: Procedure and handling
Dr. Soumita Bagchi & Dr. Rajesh NG

09.30 – 10.00 AM – Pattern recognition in renal biopsy interpretation
Dr. Swarnalata Gowrishankar

10.00 – 10.30 AM – Immunofluorescence and masked antigens: A changing scenario
Dr. Geetika Singh

10.30 – 11.00 AM – Ultrastructural basis of renal biopsy interpretation
Dr. Vinita Agarwal

11.00 - 11.15 AM – Panel Discussion, Q&A

11.15 – 11.45 AM – Coffee break

Session II. Case based discussion – Native kidney diseases
Panelists: Dr. N Jayaram, Dr. Vijay Kher

11.45AM– 01.30PM - Case presentation, quiz and panel discussion

01.30 – 02-15 PM - Lunch Break

Session III. Case based discussion – Renal Transplant Pathology

Panelists: Dr. Manoj Jain, Dr. Sanjiv Saxena

02.15PM – 4 PM – Case presentation, quiz and panel discussion

(Quiz moderators: Dr. Minakshi Bhardwaj, Dr. Ronica Baruah & Dr. SK Verma)

04.00 – 04.30PM – Coffee break/ Executive Committee Meeting of the ISRTP

04.30 – 04.45 PM – Declaration of the result of quiz competition

04.45 – 05.30 PM – General body meeting of the ISRTP
3rd International Renal Pathology Conference

Co-Directors: Amit Dinda; Anthony Chang; Cynthia Nast

Venue – Jawaharlal Nehru Auditorium, AIIMS, New Delhi

Friday, February 10, 2017

8:00 - Registration
8:30 - 9 am - Introduction and welcome remarks
  Director ( AIIMS), HOD Pathology, Amit Dinda, Anthony Chang

Moderators: Anthony Chang (USA), Ian Roberts (UK)

9 – 9:30 am - Molecular Pathology of MCD/Focal and Segmental Glomerulosclerosis
  Jeffrey Hodgkin (USA)

9:30 – 10 am - Podocytropathies: A Nephrologist’s Perspective
  John Feehally (UK)

10 -10:30 am - Foam cells and the glomerulus
  Charles Alpers (USA)

10:30 – 11 am - Coffee break & Poster viewing

Moderators: Surya Seshan (USA), Amit Dinda (India)

11 – 11:30 am - 3D Map of Glomerular Capillary Network: Implications in Health & Disease
  Anthony Chang (USA)

11:30 – 12 pm - Glomerular Microcirculation in Development and Disease
  Michio Nagata (Japan)

12 - 2 pm - Lunch & Poster viewing

Moderators: David Thomas (USA), Charles Alpers (USA)

2.00-2:30 pm - IgA Nephropathy: Insights on pathogenesis and Therapy
  John Feehally (UK)

2:30-3 pm - Histologic Classifications – the Good, Bad and Ugly
  Mark Haas (USA)

3-3:30 pm - GN Classification and Report Standardization
  Sanjeev Sethi (USA)

3:30-4 pm - Coffee break & Poster viewing

Moderators: Cynthia Nast (USA), Alok Sharma (India)

4-4:30 pm - Drug-Induced Glomerulopathies
  Ritambhra Nada (India)

4:30-5:30 pm - Interesting Native Biopsies

7.00 pm onwards - Networking Dinner
Venue – Swimming pool lawn, AIIMS
Saturday, February 11, 2017

Moderators: John Feehally (UK), Anjali Satoskar (USA)

8:30 – 9 am  -  Pediatric Renal Pathology: An Update  
                David Thomas (USA)

9 - 9:30 am  -  21st Century Genomic Advances in Pediatric Renal Diseases  
                Loreto Gesualdo (Italy)

9:30 – 10 am  -  C3 Dominant Glomerulonephritides – the spectrum of complement dysregulation  
                Sanjeev Sethi (USA)

10- 10:30 am  -  Pathology of Thrombotic Microangiopathy  
                Surya Seshan (USA)

10:30 – 11 am  Coffee break & Poster viewing

Moderators: Heinz Regele (Austria), Sanjay Agarwal (India)

11 – 11:30 am  -  Mechanisms of Interstitial Fibrosis  
                Agnes Fogo (USA)

11:30 – 12 pm  -  Investigative and Clinical Digital Renal pathology  
                Alok Sharma (India)

12 - 2 pm  Lunch & Poster viewing

Moderators: Sanjeev Sethi (USA), Ritambhra Nada (India)

2-2:30 pm  -  Tubulo-interstitial diseases  
                Amit Dinda (India)

2:30-3 pm  -  Tropical Renal Infections  
                Vivekanand Jha (India)

3-3:30 pm  Coffee break & Poster viewing

Moderators: Agnes Fogo (USA), Jeffrey Hodgin (USA)

3:30-4 pm  -  HIV and CKD  
                Cynthia Nast (USA)

4-4:30 pm  -  Post-mortem renal pathology  
                Ian S.D. Roberts (UK)

4:30-5:30 pm  -  Ask the Experts: Q&A Session  
                John Feehally, Charles Alpers, Ian Roberts
**Sunday, February 12, 2017**

*Moderators*: Mark Haas (USA), Michio Nagata (Japan)

8:30 – 9 am  - *Borderline rejection: the clinical perspective*
Sanjay Agarwal (India)

9 – 9:30 am  - *C4d Negative Antibody-Mediated Rejection*
Aruna Vanikar (India)

9:30 – 10 am  - *Transplant TMA*
Heinz Regele (Austria)

10-10:30 am  *Coffee break*

*Moderators*: Vivekanand Jha (India), Alok Sharma (India)

10:30 – 11:15 am  - *Transplant Biopsies: Banff Update and Interpretation of Intimal Arteritis*
Mark Haas (USA)

11:15 – 11:45 am  - *Interesting Transplant Biopsies*

**POSTER AWARDS AND CLOSING REMARKS**
OS 01

Amyloidogenic Casts: are they different clinically?
Parimal Agrawal¹, Rajesh Kumar¹, Raja Ramachandran², Ashwani Kumar¹, Varma N³, Varma 4,\nAggarwal 5, Gupta KL², Malhotra P⁴, Nada R⁴

Department of 1Histopathology, 2Nephrology, 3Hematopathology, 4Hematology,\n5Immunopathology PGIMER, Chandigarh.

Background
Amongst the variety of renal lesions associated with plasma cell dyscrasias, Light chain cast\nnephropathy is the commonest. Rarely, casts are composed of amyloid, when it is termed\namyloidogenic light chain cast nephropathy (ALCCN). Even rarer is the presence of intratubular\namyloidosis.

Aims & Objective
To document clinico-pathologic spectrum of amyloidogenic light chain cast nephropathy.

Materials & methods
All cases of light chain cast nephropathy, whether antemortem renal biopsies or postmortem autopsy\nmaterial between 2013 to 2016 were retrieved and evaluated for morphology consistent with\namyloidogenic light chain cast nephropathy. Immunofluorescence and electron microscopy features\nwere reviewed. Clinical data was correlated.

Results
Amyloidogenic casts were reported in 0.2% native kidney biopsies from 2013 to 2016 (6/2995 cases).\nThe study included five male and one female patient with mean age of 52 years (range – 40 to 70\nyears). All cases had multiple myeloma and lambda light chain restriction was present in four cases.\nRenal dysfunction (mean serum creatinine – 5.75 mg/dl, range – 2.2 to 8.8 mg/dl) was noted in all\ncases. In five cases, amyloidogenic casts co-existed with light chain casts and in three cases with\nproximal tubulopathy. On electron microscopy, two cases had presence of amyloid fibrils in the casts\nas well as the tubular epithelial cells, indicating intratubular amyloidosis. None of the cases had\nMonoclonal Immunoglobulin Deposition Disease or extra-tubular amyloidosis. Three cases had\nsevere IFTA component and 2 patients succumbed to their illness.

Conclusions
The clinico-pathologic implications of Amyloidogenic light chain casts (ALCCN) remain an enigma. It\nhas been hypothesized that amyloid is formed by filtered light chains within the tubular lumen. The\nclinical and renal outcome as a result of amyloidogenic light chain cast nephropathy or intratubular\namyloidosis has not been documented. The present series shows severe injury to tubulointerstitial\ncompartment, which probably accelerates the progression to ESRD as compared to simple cast\nnephropathy cases.
Significance of glomerular C4d deposition in Lupus nephritis patients.
Dr. Madan Kumar Solanki, Dr. Deepika Hemrajani, Dr. Ranjana Solanki
SMS Medical college jaipur

Background
Systemic lupus erythematosus is the prototype of autoimmune disease caused by dysregulation of immune system. Immune complex mediated complement activation is the main mechanism in the pathogenesis of lupus nephritis leading to deposition of various complement fractions and immune complexes in the various compartment of glomerulus resulting in glomerulonephritis. C4d is a complement split product produced by breakdown of C4b into C4c and C4d in classical or lectin pathway. Glomerular C4d deposition in lupus nephritis seems to cause glomerular injury in these patients. C4d is also a well known sensitive and specific marker of antibody mediated renal allograft rejection and has been found to be accurate indicator of lupus activity than serum complement levels. The purpose of our study was to assess the role of C4d deposition in lupus nephritis cases.

Aims and objective
The study was conducted with following aims and objectives-
1. To study the pattern of C4d deposition in native renal biopsies of lupus nephritis patients
2. To correlate the C4d deposition pattern with immunofluorescence (IF) pattern of other immune complexes in different histological subtypes of lupus nephritis.
3. To correlate whether glomerular C4d deposition can be a useful marker of disease activity in lupus nephritis.
4. To correlate glomerular C4d deposition with clinicopathological features.

Material and methods
A total of 9 diagnosed cases of lupus nephritis received in our department between Jan 2015 to Oct 2016 were analyzed and studied by light microscopy, immunofluorescence (IF) and the result were compared with the C4d deposition pattern which was performed on paraffin embedded tissue by immunohistochemistry.

Results and conclusion
All 9 cases were positive for antinuclear antibody (ANA) and 7 cases were positive for anti-DsDNA. In one case anti-DsDNA was negative while in one case result of anti-DsDNA was not available. Serum creatinine level was ranging from 0.81 to 7.0 mg/dl. Very high serum creatinine (7.0 mg/dl.) was found in one case. Class IV-G was most common subtype seen in 5 cases (55.55%) There was one case of each class I, class II and class V while one case reported as class I + III-G. All cases were positive for C4d deposition, intensity of which was ranging from +1 to +3. Activity index was ranging from 0/24 to 12/24 and chronicity index was ranging from 0/12 to 5/12. Statistical analysis of C4d deposition with different histological subtypes, disease activity and clinical parameter will be observed and discussed.

Keywords- Lupus nephritis, C4d, immune complexes, serum complement, glomerulus
Spectrum of C4d deposition in Proliferative Glomerulonephritis: Its role in evaluation of native renal biopsies.

Dr Suman Meena, Dr. Ranjana Solanki, Dr. Deepika Hemrajani, Dr. Nidhi Sharma
SMS Medical College Jaipur

Background:
Proliferative glomerulonephritis (GN) encompasses immune-complex mediated glomerulonephritis and complement mediated glomerulopathy. There is glomerular deposition of immune complexes/immunoglobulins (Ig) and C3 in immune-complex mediated GN as a result of infections, autoimmune diseases or monoclonal gammopathy. C3 glomerulopathy is derived from deposition of C3 and other fragments of complement with minimal or no deposition of immune complexes/Ig. The presence of isolated C3 implies that the complement has been activated via the dysregulation of alternative pathway. C4d is derived as a byproduct of activation of classical and lectin pathway.

Aims & Objectives:
The aim of our study is to evaluate whether C4d deposition in glomeruli had any correlation with various immunopathologic variables of proliferative (GN).

Methods:
We conducted a retrospective study of 36 cases of proliferative glomerulonephritis including infection-related glomerulonephritis (IRGN), Membranoproliferative glomerulonephritis (MPGN) with C3 alone, MPGN with immune complex mediated glomerulonephritis, Lupus nephritis and IgA nephropathy. We also evaluated the role of C4d in cases of Membranous Glomerulonephritis.

Results & Conclusions:
Results of C4d staining in proliferative glomerulonephritis with various histological and clinical correlates will be observed and discussed.
Application of novel tumour grading scheme for chromophobe renal cell carcinoma
Ganesh Bahirwade, Santosh Menon, Ganesh Bakshi, Gagan Prakash, Amit Joshi, Sangeeta Desai. Tata Memorial Centre, Mumbai, Maharashtra, India.

BACKGROUND:
Renal cell carcinoma (RCC) is the third commonest urological malignancy. Grading of RCC have prognostic significance and aid in appropriate clinical management decisions. Fuhrman grading system (FNG) is already established grading system for conventional RCC. Due to inherent nuclear atypia Fuhrman nuclear grading system is not appropriate for chromophobe renal cell carcinoma (ChRCC). This study focuses on feasibility of application of a novel grading system and its correlation with various histological parameter and disease free survival.

AIMS & OBJECTIVES:
Comparison of Fuhrman nuclear grading system with a novel tumour grading system for chromophobe RCC and correlation with pathological stage and clinical outcome.

MATERIAL & METHODS:
Eighty cases of ChRCC diagnosed on radical nephrectomy were identified during the study period from 2005 to 2014. The tumour in each case was assigned a Fuhrman nuclear grade as well as the novel proposed grading system (CTG) by Paner et al. Demographic parameters, pathological features including TNM stage and follow-up were studied.

RESULTS:
When both grading system were applied in ChRCC, FNG 3 and CTG 1 was the most common grade assigned. Distribution of grade was: FNG1-4 was 1(1.3%), 23(28.3), 44(55.0%) and 12(15.0%) for CTG1-3 was 48(60.0%), 20(25.0%) and 12(15.0%) respectively.

CTG and FNG were analysed with various parameter, CTG showed more significant correlation with DFS than FNG (p value 0.000003 vs 0.001, respectively). In non sarcomatous cases CTG significantly correlated with disease specific adverse event while FNG did not show any significant correlation (p value 0.001 vs 0.106, respectively). CTG correlated with pathological stage while FNG lacked correlation with pathological stage (p value 0.012 vs 0.59, respectively). CTG had higher predictive accuracy regarding DFS than FNG on ROC curve analysis when whole group was analysed (p value 0.000015 vs 0.001 respectively). In non sarcomatous cases only CTG predicted DFS accurately when compared to FNG (p value 0.001 vs 0.063, respectively).

CONCLUSION:
CTG is feasible to apply in ChRCC. It is a better predictor of DFS and disease specific adverse event than FNG. Hence, we conclude that CTG is more useful than Fuhrman nuclear grading system in grading of ChRCC.
Utility of CD44 immunohistochemistry on renal biopsies with diagnoses of minimal change disease and focal segmental glomerulosclerosis.

Authors:
*: Department of Histopathology, Apollo Hospitals, Jubilee hills, Hyderabad.
**: Department of Nephrology, Apollo Hospitals, Jubilee hills, Hyderabad.

Background:
CD44 is a transmembrane adhesion glycoprotein functioning as a hyaluronan receptor and has been proposed in the kidney as a marker of activated parietal epithelial cells (APECs). These APECs migrate into glomerular tuft, transition to podocytes and participate in the glomerulosclerosis of focal and segmental glomerulosclerosis (FSGS). CD44 has therefore been proposed as a marker to detect very early sclerotic lesions in FSGS.

Aims and Objectives:
1. To study the pattern of parietal epithelial cell activation using CD44 as an immune marker in cases with diagnoses of minimal change disease (MCD), MCD with interstitial fibrosis and tubular atrophy (IFTA), and FSGS (all types).
2. To evolve a semi-quantitative scoring system for CD44 expression.
3. To correlate the scores with the histology in the above mentioned groups and with the clinical follow-up, whereever possible.

Material and Methods:
It is a retrospective observational study of renal biopsies of children below 18 years of age with nephrotic syndrome and a pathologic diagnosis of MCD, MCD with IFTA or FSGS. 20 cases of each group were selected. A CD44 immunohistochemical stain was done in all 60 cases using mouse monoclonal antibody with appropriate controls on the automated Benchmark XT Ventana platform.

Biopsies with a minimum of 10 glomeruli were included. Even a single positive cell per glomerulus was taken as positive and the scoring was done as a percentage of total glomeruli. [Score 0: 75%]. The score was correlated with the findings on light microscopy and the follow-up status whereever available.

Results:
There was progressive increase in the CD44 scores from MCD to MCD with IFTA and FSGS. On a preliminary analysis, the score also seems to correlate with the subsequent course of the disease.

Conclusions:
CD44 immune stain appears to be useful in the spectrum of MCD, MCD with IFTA and FSGS to recognise cases of an early segmental sclerosis and in predicting the course of the disease.
Etiopathological association of glomerular crescents with predictors of renal outcome and end stage renal disease in glomerulonephritis

Dr. Nishika Madi Reddy1, Dr. Mary Mathew2, Dr. Raveendra Prabhu3
Affiliation: Department of Pathology, Kasturba Medical College, Manipal, Manipal University1,2, Department of Nephrology, Kasturba Medical College, Manipal, Manipal University3

BACKGROUND
Glomerulonephritis is the commonest cause of end-stage renal disease (ESRD) in developing countries. It has been subdivided into various categories, based on their clinical presentation, histological analysis, and laboratory data. Formation of glomerular crescents is a response to severe glomerular injury. A higher percentage of crescents is a sign of an aggressive form of glomerulonephritis and is associated with poor prognosis.

AIMS AND OBJECTIVES
This study was undertaken to investigate the etiology, clinical features and prevalence of different types of glomerulonephritis with crescents and to study the association of glomerular crescents with predictors of renal outcome and end-stage renal disease.

MATERIALS AND METHODS
This study included 72 cases of glomerulonephritis with presence of crescents over a period of 7 years. They were divided into three groups: group 1 (50% crescents). The clinical and laboratory parameters were compared among the three groups. The association of clinical and laboratory parameters, and percentage of glomerular crescents with end-stage renal disease was also studied.

RESULTS
There were 32 males and 40 females with a mean age of 37.33 ± 16.54 years. Majority of the patients presented with hypertension and renal failure. Immune complex glomerulonephritis was the most common type of glomerulonephritis, of which IgA nephropathy constituted the majority of the cases (29.2%). Maximum number of cases of Anti-GBM had more than 50% glomerular crescents (85.7%). Older age at presentation, elevated serum creatinine, blood urea levels and decreased eGFR at the time of diagnosis were significantly associated with the percentage of glomerular crescents and ESRD. However, no such association was observed with urine parameters. Significant association was present between the percentage of glomerular crescents and progression to ESRD in all types of glomerulonephritis, except in postinfectious glomerulonephritis.

CONCLUSION
Older age at presentation, hypertension, gross haematuria, nephritic and nephrotic syndromes were the clinical parameters which showed a significant association with the percentage of crescents. Significant association was present between the percentage of glomerular crescents and progression to ESRD in all types of glomerulonephritis, except in postinfectious glomerulonephritis.
Role of associated risk factors in the prevention of chronic kidney disease
Dr. Anjani Bakshi, Dr. Kalyani Singh

Background:
Chronic kidney disease (CKD) is a public health problem and extremely prevalent in developing countries. Risk factors such as obesity, uncontrolled diabetes, hypertension, high intake of sodium and western diet are significantly associated with morbidity, accelerated progression and premature mortality of CKD patients. To reduce the prevalence of CKD and associated increased cardiovascular risk, careful monitoring of risk factors is critical. Family history is an effective and promising way of identifying risk factors for early prevention of disease condition.

Aim and objectives:
Non-dialysed Indian CKD patients were studied to identify the role of associated risk factors in the prevention of chronic kidney disease.

Material and method:
A total of 120 non-dialysed CKD patients from outpatient department of a private hospital, Delhi, India, were analysed on demographic characteristics, educational status, primary and secondary causative factors, case and family history of obesity, diabetes, hypertension, cardiovascular diseases (CVD) and renal stones (RS). Body Mass Index (BMI) was calculated as the ratio of weight (in kilograms) with height (in meter square). Independent t test was used to assess gender differences in BMI and chi square test to associate patient disease with family history were some of the statistical tests.

Results:
Diabetes (57%) and hypertension (79%) was highly prevalent among non-dialysed Indian CKD patients. More than 50% of patients had family history of either diabetes or hypertension. For diabetes, CVD and RS, significant association was observed between family history and presence of similar disease in the patient. 80% of patients who had diabetes had at least two diabetic family members (p = 0.004). However, the association was weak for hypertension. Obesity related risk found to be high in patients with BMI 26.83 ± 5.35 kg/m2.

Conclusion:
The above results indicate that obesity, diabetes and hypertension are strongly associated with CKD. Blood sugar level, if controlled at primary level in those with strong family history, may reduce the chances of renal disease. Blood pressure need to maintain as ≤130/80mmHg to slow down the progression of CKD.
IgA nephropathy: clinical and morphological predictors of end stage renal disease

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BACKGROUND
IgA nephropathy is the most common type of glomerulonephritis in the world. The main criterion of diagnosis is IgA dominant or co-dominant immune deposits within glomeruli. The histological lesions and clinical presentation are variable and influence the renal outcome.

AIMS AND OBJECTIVES
This study was undertaken to investigate the clinical features and histopathological features of IgA nephropathy and its association with end stage renal disease (ESRD).

MATERIAL AND METHODS
This study included 21 cases of IgA glomerulonephritis with crescents over a period of 7 years. The clinical, laboratory and histopathological findings were assessed and correlated with progression to end stage renal disease (ESRD).

RESULTS
There were 14 males and 7 females with a mean age of 30.14 ± 11.7 years. The mean duration of follow up was 10.90 ± 8.56 months. The most common clinical presentations were hypertension and nephritic syndrome. The mean values calculated for the following parameters serum creatinine, serum albumin, haemoglobin, eGFR, and 24 hr urine protein were 3.11 ± 1.71 mg/dl, 3.06 ± 0.87 gm/dl, 10.51 ± 2.6 gm%, 32.66 ± 17.13 ml/min/1.73m2, 1795.42 ± 1193.61 mg/day respectively. 8/21 (38.09 %) cases progressed to ESRD, out of which 50% cases had at least 50% mesangial expansion, 37.5% had at least 50% glomerular sclerosis. Tubular atrophy was present in 87.5%, endocapillary hypercellularity was seen in 37.5%, and interstitial fibrosis in 37.5% cases. 14.3% cases showed >50% glomerular crescents, with 2 cases progressing to ESRD.

Conclusion
Hypertension and Nephritic syndrome were the more common clinical presentation in IgA nephropathy. Elevated serum creatinine levels and decreased eGFR were associated with higher percentage of crescents and ESRD. The percentage of glomerular crescents, extent of glomerular sclerosis and tubular atrophy had significant association with progression to end stage renal disease.
Concurrent anti-GBM and ANCA disease: clinical features and outcomes
Mary Mathew1, Nishika Reddy2, Ravindra Prabhu

Background
Anti-GBM disease is a lethal autoimmune disorder associated with autoantibodies against the $\alpha_3$ chain of the type IV collagen of the basement membrane against kidney, choroid plexus, lung, retina and cochlea. It is an aggressive disease with glomerular crescents, rapid progression to end stage renal failure and dialysis dependency. In 20-25% of patients, association with ANCA antibody portends a high rate of recurrence.

Aims & Objective
The aim of this study was to determine the clinical features and outcomes of patients with concurrent anti-GBM and ANCA disease.

Material & Methods
A 5 year retrospective study from 2011 to 2015 revealed 7 cases of anti-GBM disease of which 4 were positive of ANCA antibodies.

Results
There were eight cases of anti-GBM disease, of which 3 cases were positive for p-ANCA and 1 was positive for c-ANCA. The ages ranged between 17 and 73 years and 3 were females and 1 was a male. All patients presented with anemia, hematuria, proteinuria and high creatinine levels. 2 patients presented with fever and a 17 year old girl presented with polyarthralgia with lower limb rashes. All patients received dialysis and immunosuppression and 1 patient received plasmapheresis. All patients are alive and are on follow up.

Conclusions
Concurrent anti-GBM and ANCA disease is rare and associated with high mortality rate. Unusual presentation may results in delay in diagnosis and treatment. Aggressive treatment with immunosuppression and plasma exchange is warranted to induce remission and long term follow-up and maintenance immunosuppression is necessary to prevent relapses.
OS 10

Renal amyloidosis-Any change over the decades?

Abin Koshy¹, Ashwani Kumar¹, Mani Luthra², Vinay Sahuja³, Kusum Joshi¹, K.L. Gupta³, Ritambhra Nada¹
¹Departments of Histopathology, ²Immunopathology, ³Nephrology, Postgraduate Institute of Medical Education and Research, Chandigarh, India.

Background:
The spectrum of renal amyloidosis in India is different from the West. With the improvement in diagnostic strategies, better treatment options for myeloma and chronic inflammatory diseases the spectrum is likely to have changed. This study from North India was planned to analyse any change in pattern and compare with the west.

Aims & Objective
Compare spectrum of renal amyloidosis with the patterns prevalent in the past and with western countries.

Material & method
All cases of renal amyloidosis (2009-2014) were analyzed and classified as primary immunoglobulin (Ig) related, secondary inflammation related with no Ig light chain restriction and SAA negativity were analysed for other primary amyloidosis.

Results
Most of renal amyloid were serum amyloid-A (SAA) positive (60%), and only quarter were Ig amyloidosis (27%), unlike Western literature where most were Ig amyloidosis succeeded by SAA amyloidosis due to rheumatic diseases. In our study SAA amyloidosis was associated with tuberculosis (60%) and chronic rheumatic conditions (21%). 16% cases didnot reveal any significant chronic inflammatory disease and were likely to be SAA positive primary amyloid. Seven cases, which were Ig and SAA negative, were stained with transthyretin (TTR) and alpha-fibrinogen (aFib) which showed TTR positive in 2 cases and fibrinogen in 1 case. Proteomics of 2 cases confirmed SAA amyloid, of which one was HLA linked. One case was autoinflammation associated SAA positive due to MLP3 mutation. Percentage of Ig and SAA amyloid were similar as reported three decades ago though cases are better characterised in present study. In similar cohort 87% secondary amyloid and 10% Ig primary amyloid with TB as commonest association (59%) was documented. There were no cases suggestive of Lect amyloid.

Conclusion
Spectrum of amyloid in India in terms of SAA positivity and Ig related remains same over the decades, though with availability of expanded immunohistochemistry, molecular technique/proteomics, some cases are better characterised. Compared to west, SAA amyloid outnumbered Ig primary amyloid. Newly described amyloid like Lect, Apolipoprotein I/IV amyloid were not seen in our population. TTR and aFib amyloid were rare.
Telepathology in native renal biopsy: Heralding a new era in long distance collaboration.
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BACKGROUND
New and innovative technologies including whole slide imaging (WSI) are now available for digital imaging and telecommunication. These tools can be appropriately leveraged to foster rapid and accurate diagnosis and utilize international expertise as needed for challenging cases. Previous studies have examined WSI usage in the setting of organ transplant and indicated that WSI technology is a reliable method for accurate and rapid diagnostic evaluation of biopsy materials. The value of these tools in assessment of native renal biopsies is still under investigation.

AIMS AND OBJECTIVES
We conducted a pilot study to assess feasibility of accurate interpretation and diagnostic concordance using imaging in 20 challenging medical renal biopsy cases. Experience with this set of cases will be used to eventually develop and streamline use of whole slide imaging technology to share cases and build a good learning environment.

MATERIALS AND METHODS
Renal biopsy images were shared between collaborators of cases presenting at UIHC over last 10 months. Before establishing a dedicated WSI system we exchanged images of native renal biopsies from 20 cases. These included hematoxylin and eosin, special stains including Jones methenamine silver, periodic acid Schiff and trichrome, immuno-fluorescence and electron micrographs. Broad diagnostic categories included IgA nephropathy, lupus nephritis, membranous nephritis and pauci-immune necrotizing and crescentic glomerulonephritis.

RESULTS
There was 100% concordance in the broad diagnostic category. Mild differences were noted in categorization of chronicity including interstitial fibrosis and tubular atrophy. Turnaround time was less than 24 hours despite the time zone differences.

CONCLUSIONS
Native renal biopsy slides may also be amenable to WSI and long distance collaboration for diagnosis. While biopsy processing (excluding immunofluorescence and electron microscopy) may be feasible at smaller medical centers, expertise to read the same is not readily available. Imaging technologies offer an efficient means to provide rapid diagnosis and enhance the knowledge base. Since we did not use WSI there was interobserver variability in assessment of disease chronicity. As the next step forward, we propose multicenter collaboration in pooling resources to establish WSI programs which will have value-added clinical benefit to patient care.

Key words: WSI, Telepathology, renal biopsy, native.
Nephron sparing surgery: Pathological aspects with emphasis on margins and value of frozen section

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Background:
Nephron sparing surgery (NSS) is being performed increasingly for treatment of small renal tumors, including those incidentally detected as well as in selected patients having normal contralateral kidney. This often necessitates assessment of renal parenchymal margins by frozen section analysis.

Aim:
To analyze the pathologic characteristics of NSS seen at our institute including frozen section margin assessment.

Material and Methods:
A retrospective clinicopathological analysis of NSS seen at our institute from 2004 to 2014 was done. Histopathology slides were retrieved from our archives; clinical details and follow-up of patients were obtained from electronic medical records.

Results:
One hundred fifteen cases of NSS patients were identified. Male to female ratio was 2.2:1 (79 males and 36 females). The mean age was 51.8 years (range 28-80 years). One hundred five cases had a malignant diagnosis of which the commonest was conventional renal cell carcinoma (81 cases). Majority of the cases (81 cases) had small tumors (pT1a, 56 cases and pT1b, 25 cases; pT = pathologic stage) with median tumor size of 3.2 cm. Intraoperative frozen consultation margin was positive in 18/74 cases of which 10 were revised. The margin at frozen section was 1mm - 5mm in 49/74 cases. The mean margin for all cases was 2.1mm. Four cases were upgraded to pT3 on final histopathology and 2 of these patients underwent radical nephrectomy. Follow-up was available in 74 patients. The median follow-up was of 27 months (range 4-105 months). One patient of papillary renal cell carcinoma type 2 and another patient of chromophobe renal cell carcinoma had recurrence during follow-up.

Conclusions:
Conventional RCC is the commonest histology seen in NSS. Frozen section consultation has definite role in evaluation of margins of NSS and is not associated with an increased risk of local recurrence in small renal tumors even when margins are close/involved. The biology of small tumors allows conservative approach following NSS.
Comparative Assessment of Creatinine-based Estimates of Renal function in Healthy Indian Adults

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Background:
Precise measurement of renal function is imperative for the diagnosis and management of kidney disease. The estimation of glomerular filtration rate (GFR) are based upon prediction equations derived from chronic kidney disease (CKD) cases.

Aims and Objective:
The present study was carried out to investigate the status of renal function in 196 healthy adults without kidney disease using creatinine-based prediction equations.

Materials and Methods:
Biochemical estimation of serum creatinine (SCr) concentration was performed by Jaffe’s alkaline picrate method. Participants were divided into two groups with GFR above and below 90 ml/min/1.73 m². The CKD stages were divided into five stages on the basis of estimated GFR. The GFR were calculated according to the prediction equations of Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) and Modification of Diet in Renal Disease 2 (MDRD2).

Results:
The mean ± SD GFR ≥90 and 60 ml/min per 1.73 m² in all the age groups by both the CKD-EPI and MDRD2 estimates, while MDRD2 predicted stage 3A CKD with GFR within 45-59 ml/min per 1.73 m² in subjects above 40 years.

Conclusions:
The normal serum creatinine reference interval does not necessarily reflect a normal renal function. The MDRD2 prediction equation underestimated GFR compared to CKD-EPI at GFR Key words: creatinine, GFR, CKD-EPI, MDRD2, Kidney Disease
Clinicopathological Profile of Paraprotein Associated Kidney Disease in Monoclonal Gammopathies: An Analytical Observational Study

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BACKGROUND:
Renal involvement in monoclonal gammopathies presents with different clinico-morphological patterns, which can occur either at onset or late phase of haematological disease or after chemotherapy. The spectrum of renal involvement in monoclonal gammopathies is also expanding with newer renal lesions being described with advancement in diagnostic methods, renal biopsy is mandatory for accurate diagnosis, as the different morphological patterns carry therapeutic and prognostic implications.

AIMS and OBJECTIVES:
a. To analyze the kidney biopsy findings in patients with monoclonal gammopathies.
b. To assess the clinico-pathological and haematological profile in patients with kidney disease in monoclonal gammopathies.

MATERIALS & METHODS:
14 diagnosed cases of monoclonal gammopathies with renal derangement during January 2015 & October 2016. Clinical, renal and haematological details of these patients are collected and analysed.

RESULTS:
Clinical profile: 4 cases presented with acute kidney injury, 3 each with nephrotic syndrome and RPRF, 2 cases with subnephrotic proteinuria, 1 with nephritic syndrome and 1 a case of plasmacytoma with isolated elevated serum creatinine. Spectrum of renal lesions identified: Kidney biopsy findings are summarised as: 4 each with cast nephropathy, and renal amyloidosis, 2 Proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID), 1 case each of MPGN with monoclonal restriction, LCDD, LHCDD, and light and heavy chain amyloidosis. Haematological profile: 6 - multiple myeloma, 5 - marrow plasmacytosis, 1 - Normal active marrow and for 2 case marrow is not available.

CONCLUSION:
Among all, cases with cast nephropathy in kidney biopsy has showed increased bone marrow plasma cell burden and positive electrophoresis as well as immunofixation in comparison to others which show less plasma cell burden, which are now a days labelled as monoclonal gammopathy of renal significance (MGRS). These cases may not meet the criteria for overt multiple myeloma, however knowledge on these lesions can help us identify the disease early and improves the renal outcome.
Clinicopathological characteristics of non diabetic renal disease in 390 patients with type 2 diabetes mellitus

Aims & Objective:
To investigate the clinical and pathological features of non-diabetic renal disease (NDRD) in the patients with type 2 diabetes mellitus (T2DM), and evaluate the diagnostic importance of the renal biopsy in T2DM patients.

Methods:
A retrospective analysis based on medical records of 390 patients with T2DM who underwent renal biopsy from January 1986 to October 2015 in our hospital.

Results:
Out of the 390 T2DM patients, 258 cases were males and the rest were females. The mean age was 51.1 ± 12.4 years and the duration of T2DM is 8.65Â± 5.36 years. Hypertension, edema, nephrotic syndrome, diabetic retinopathy were respectively 320(82.05%), 285(73.08%), 162(41.54%), 228(58.46%), 289(74.1%). 195 (50%) had DN, 170 (43.59%) had NDRD, and 25 (6.41%) had NDRD superimposed on DN. Of 170 cases of NDRD, 28.82% was idiopathic membranous nephropathy(IMN), 22.35% was IgA nephropathy (IgAN), 20.59% was mesangial proliferative glomerulonephritis(MsPGN),7.06% was focal segmental glomerulosclerosis ( FSGS), 5.29% was renal tubular interstitial inflammation, and 4.12% was hypertensive renal injury. The others were ANCA associated with vascular inflammatory, amyloidosis, membrane proliferative glomerulonephritis, allergic purpura nephritis. Patients with NDRD had a shorter duration of diabetes and lower frequencies of diabetic retinopathy (DR, 8.46%).

Conclusion:
There were all kinds of different pathological pattern in the patients with NDRD of T2DM. IMN was the most common in the NDRD, followed by IgAN. NDRD was not completely exclusive by a longer duration of diabetes and diabetic retinopathy. Renal biopsy can make a definitive diagnosis to distinguish DN, NDRD and NDRD superimposed on DN in the T2DM patients and be helpful to ameliorate renal outcomes.
Clinicopathologic profile of BK Polyoma Virus Nephropathy: 5-year experience from 41 case

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Background:
Allograft dysfunction due to BK polyoma virus is one of the dreaded complication in current Immunosuppression era. Graft biopsy remains the gold standard in diagnosis and management.

Aims & Objective:
To study the clinicopathologic profile of BK polyoma virus nephropathy (BKPVN) in allograft patients.

Materials and Methods:
Allograft biopsies during the period of Jan 2012 to Nov 2016 (59 months) were screened for biopsy proven BKPVN. Clinical, laboratory parameters & management were noted from medical records. Light microscopy, Immunohistochemistry (SV40, C4d) were performed and pathologic lesions studied. Immunofluorescence (IgG, IgA, IgM, C3, C1q, kappa & lambda) were done in 16 cases.

Results:
A total of 211 cases were morphologically suspected to be BK Polyoma requiring SV40 Immunoperoxidase stain, out of 2290 allograft biopsies referred to our center. Out of these, 34 patients with 41 episodes were SV40-IHC/biopsy proven accounting for 1.5% incidence, and formed the study group. Males were 20 and females were 14 in number, ranging from 13-68 yrs (mean 42 yrs). Imunosuppressive medication details included Tacrolimus based triple drug maintenance regime. Induction was given in 20 cases. Clinically, slow progressive graft dysfunction was the noted in all, except for one (Sr creatinine range 0.8-5.9 mg/dL). One of these was diagnosed in protocol biopsy done at 3 month. Post-transplant duration for diagnosis ranged from 3-34 months. On histology, typical tubular epithelial inclusions were seen in 34 cases (82%), and rest were depicting nuclear enlargement with hyperchromasia. All of them were accompanied with tubulointerstitial inflammations (t1-t2 & i1-i2 Banff score). Interstitial fibrosis & tubular atrophy (IFTA) was nil in 16, mild in 18 & moderate in 7. None of them had severe IFTA. BKPVN was associated with acute vascular rejection in two cases (5%) and mutli-infection with bacteria in two cases (5%). Immunofluorescence had fine granular deposits with IgG (3+ intensity on a scale of 0-4) along the tubular basement membranes in 4/16 cases (25%). Patchy granular deposits were noted with C4d Immunoperoxidase stain in 3/41 cases (7.3%). Immunosuppression was reduced in all of them, with addition of Quinolones / Leflunomide. Cidofovir was added in 5 cases. Details on graft survival are available in only 4 patients (1 complete recovery, 1 partial recovery, 2 graft loss).

Conclusions:
Slow progressive graft dysfunction is the most common presentation of BKPVN, requiring low threshold for allograft biopsy. Granular deposits along tubular basement membrane (IF-IgG; & IHC-C4d) is a helpful surrogate clue to suspect & confirm BKPVN by Immunohistochemistry. Strong suspicion for BKPVN is crucial and benefits the long term survival of allograft.
Oxford classification of IgA nephropathy: Histological subclassification and prognostic significance: First Indian Study

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BACKGROUND
IgA nephropathy is the commonest form of glomerulonephritis worldwide & is characterised by presence of prominent IgA deposits in the mesangial regions which are detected by direct immunofluorescence. It has a wide spectrum of presentation, both clinically as well as histologically. Hence worldwide classification of IgAN is necessary which will help in following ways: 1) Prediction of clinical course 2) Guiding selection of therapy & predict response 3) Guiding and stratifying inclusion in clinical trials. Providing insights into pathophysiology that may help with design of therapy in future. We studied total 71 cases of IgA nephropathy, applied Oxford scores on all cases and tried to find out prognostic significance of each of M, S, E & T score.

OBJECTIVES:
1. To classify the cases of IgA nephropathy at our institute according to Oxford Classification and estimate demographic distribution of Oxford scores (i.e. M, S, E & T) and of each of the subtypes of IgA Nephropathy as per the Oxford Classification (e.g. M1S1E0T0 etc)
2. To find out correlation between Oxford scores (M, S, E & T) & prognosis of the patient with respect to biochemical investigations

METHODS:
The H/E, PAS & Silver Jones stained slides of cases of IgA nephropathy of six years from 2007 to 2013 were given Oxford retrospectively.
The biochemical parameters were recorded at the time of biopsy, 6 months and 1 year after the initial biopsy.
Chi square test & student test were used to find out significant correlation between biochemical parameters & Oxford score.

RESULTS:
Significant correlation was found between S score & S.creatinine, GFR, BUN to creatinine ratio. Same was true with T score & E score. But M score did not show any significant correlation with any of the above mentioned parameters.

CONCLUSION:
1. Mesangial cellularity is the most commonly present pathology in IgA nephropathy in the present population but it did not show any clinical as well as prognostic correlation.
2. M1S0E0T0 is the commonest subtype found in IgA nephropathy patients in this population
3. S, E & T scores are the significant clinical & prognostic indicators whereas M score is not.
LUPUS NEPHRITIS: A CORRELATION OF LIGHT MICROSCOPY, IMMUNOFLUOROSCENCE, SEROLOGY AND CLINICAL FEATURES – A FIVE YEAR STUDY AT A TERTIARY CARE HOSPITAL

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INTRODUCTION:
Systemic Lupus Nephritis is a chronic inflammatory autoimmune disease with renal involvement in up to 50%-60% of patients with a predominance in women of reproductive age. The classification of lupus nephritis has evolved over the past 40 yrs as more lesions were identified and defined. Interpretation of renal biopsies in lupus nephritis has proven to be a diagnostic challenge with the need to correlate pathological findings and serological results with clinical features.

AIMS AND OBJECTIVES:
To study the different glomerular patterns on light microscopy and immunofluorescence and correlate the serological results and clinical features of lupus nephritis in renal biopsies examined in last five years at our institute. The injury patterns have been classified according to the latest ISN/RPS 2004 classification.

MATERIALS AND METHODS:
Total 116 biopsies of lupus nephritis were recorded in the past five years from 2011-2015 in our institute. Light microscopy findings were compared with the immunofluorescence patterns, integrated with serological results and the clinical details were correlated.

RESULTS:
Total 116 cases of Lupus Nephritis were evaluated in the last 5 years from 2011-2015. The age range was 6 years to 63 years with mean age of 34.5 years, of which 102 were females (87.9%) and 15 males (12.1%). The clinical presentation varied with oedema (52.58%), hematuria (35.34%), joint pain (28.44%), oral ulcers (11.20%), skin rash (11.20%), fever (9.48%), photosensitivity (8.6%) and hypertension (20.68%) and no clinical details were available in 18.96%. Serological tests for Antinuclear antibodies and dsDNA antibodies were correlated. According to the ISN/RPS 2004 classification patterns of class I were noted in 2 cases (1.72%), class II in 7 cases (6.03%), class III in 26 cases (22.41%), class IV in 32 cases (27.58%), class V in 24 cases (20.68%) and class VI in 3 cases (2.58%) and mixed patterns were noted in 20 cases (17.24%). Immunofluorescence studies for Ig G, Ig M, Ig A and complements C3 and c1q, were done in 105 biopsies and correlated with the light microscopy details.

CONCLUSION:
It is important to realize that renal biopsy findings alone cannot establish a diagnosis of lupus nephritis. Hence, to make a definitive diagnosis of Lupus nephritis, a correlation of histology, immunofluorescence patterns, serology and clinical history is necessary.
Evaluation of anti PLA2R antibodies in the sera and glomerular deposits in membranous nephropathy.


Background:
Idiopathic membranous nephropathy is an antibody mediated autoimmune glomerular disease. Several target antigens have been explored till now, phospholipase A2 receptor being the major. Antibodies against PLA2R can be studied in the sera and their nephritogenic potential can be confirmed by their presence in the glomerular deposits in cases of primary membranous nephropathy.

Aims and Objectives
1. To evaluate the anti PLA2R antibodies in the sera of the patients in membranous nephropathy and correlate their presence with the glomerular deposits by immunohistochemistry (IHC). 2. Assessing the concordance between the two methods; indirect immunofluorescence (IDIF) and ELISA used for detecting the circulating anti PLA2R antibody. 3. To analyse the efficacy of detecting anti PLA2R antibodies in the sera and glomerular deposits in differentiating primary from secondary membranous nephropathy.

Material and methods:
27 cases of membranous nephropathy were chosen and were divided into primary and secondary based on presence or absence of clinical or microscopic criteria. Group 1 comprised of primary membranous nephropathy (n=22) and group 2 comprised of secondary membranous nephropathy (n=5). The sera from all the patients was analysed using IDIF and ELISA techniques. A titre of more than 20RU/ml was considered positive. We analysed anti PLA2R antibodies in the glomeruli on immunohistochemically (IHC) stained sections. The sensitivity, specificity, positive predictive value and negative predictive value were calculated for all three methods and were compared statistically using Mc Nemar test.

Results:
1. All the patients with circulating anti PLA2R antibodies showed presence of glomerular deposits.
2. Detection of anti PLA2R on IHC differed significantly in sensitivity (81%) and specificity (60%) from IDIF/ELISA (sensitivity 50%, specificity 100%) with a p value of 0.017. 3. Both IDIF and ELISA showed a high level of concordance in their results.

Conclusion:
The circulating anti PLA2R antibodies were found to be nephritogenic in all the positive cases. Detecting Anti PLA2R antibodies in the glomerular deposits by IHC appears to be more sensitive but less specific for detecting primary membranous nephropathy.
Background.
Diabetic nephropathy (DN) presents histologically as Nodular and Diffuse Glomerulosclerosis (DGS). Kidney biopsy in diabetics is generally undertaken when there is persistent proteinuria, nephritic and nephrotic syndrome especially in absence of diabetic retinopathy to rule out DN. Renal diseases other than DN termed as 'non diabetic renal disease' (NDRD) can be found and various studies have come across supporting this fact.

AIM & OBJECTIVES:
1) To study the histomorphological features in kidney biopsies in patients with type 2 DM. 2) To establish frequency of DGS and NDRD in patients with type 2 DM. 3) To evaluate morphological characteristics of NDRD in these renal biopsies. 4) To correlate histopathology findings with clinical and laboratory parameters.

MATERIALS AND METHODS:
This is a retrospective study of renal biopsies in Type 2 diabetics with atypical clinical presentations. Diabetic patients who underwent kidney biopsy at Kasturba Hospital, Manipal between January 2009 to August 2014 were included in the study. Type 1 DM renal biopsies and post transplant diabetic renal biopsies were excluded. Demographic and Clinical data were retrieved from the patient files. Biopsy slides were collected and reviewed and categorized into 3 groups: Group 1 - diffuse and nodular diabetic nephropathy (DGS) Group 2 - Both DGS and NDRD. Group 3 - isolated NDRD cases Statistical analysis was carried out.

RESULTS:
91 renal biopsies were performed in Type 2 Diabetics between Jan 2009 till Aug 2014. Group 1 - 41(45.1 %) Group 2 - 19(20.9%) Group 3 - 31(34.1%) M:F = 5.5 :1 Age - 28 - 80 years, majority in 5th decade. RPRF- Commonest clinical presentation. Diabetic Retinopathy had a high PPV (97.2%) for DGS Hematuria > NDRD, Proteinuria > DGS, GFR > NDRD Most common NDRD was FSGS, followed by PIGN, AIN and ATN.

CONCLUSIONS:
1) High prevalence of NDRD i.e. 55% suggests renal biopsy to be performed in type 2 diabetics with an atypical clinical course as the treatment and prognosis differs widely DN and NDRD. 2) Clinical features like hematuria, absence of diabetic retinopathy and lesser duration of DM are good clinical predictors for the presence of NDRD 3) GFR is higher in NDRD than DN. Hence, NDRD have a better renal prognosis. 4) A wide spectrum of NDRD can be present in diabetic patients ranging from primary and secondary glomerulonephritis to tubulointerstitial and vascular lesions.
A Survey of C4d (IHC) expression patterns in Renal Allograft biopsies contextualized to clinical outcome- Over a period of one year

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Background: C4d staining in peritubular capillaries in renal allograft biopsies during an acute rejection episode is the footprint of AMR (antibody mediated rejection) associated tissue damage. C4d is graded on a scale of 0-3+ and even minimal staining is considered positive on IHC.

Aims and Objectives:
To study C4d IHC grades / patterns in Renal allograft biopsies with graft dysfunction and correlate it with histomorphology (Banff’s score). The histologic diagnosis given based on these parameters is further correlated with the clinical management of the patient, taking it as the gold standard. Material and Methods:
Renal allograft biopsies received in Max labs from Jan – Dec 2016 were retrospectively analysed and correlated with the treatment given to the patient on basis of biopsy diagnosis. All allograft biopsies were scored according to the Banff’s criteria and C4d was done by IHC on paraffin sections. C4d was scored in peritubular capillaries (ptc’s) from 0-3+ depending on the percentage of PTC’s with positive staining. Retrospective analysis of the treatment given to the patient was done with the histologic diagnosis and C4d score. Antibody mediated rejection was treated with plasmapheresis/IVIG/Bortezomib/rituximab. Pulse solumedrol was given in Acute cellular rejection and in resistant cases- ATG was given to the patient. Mixed Rejection was treated either as the dominant type of rejection or for both. ATN was treated conservatively. Chronic Rejections were usually not treated aggressively.

Results:
A total of 70 Allograft biopsies were received from Jan – Dec-2016. Renal allograft biopsies were reported as ATN (18 cases), ACR(10 cases), AMR(6), mixed ACR + AMR(11 cases), Chronic AMR (6), Recurrent / denovo GN (8), Resolving AMR (4) and chronic TMA/ CNI toxicity(5). Two cases were ABO incompatible allograft biopsies with ATN and managed conservatively. The most prevalent C4d score was 1+ with minimal staining in < 10 % of peritubular capillaries (ptc’s) - seen in ATN, ACR and resolving AMR amounting to 40 % of allograft biopsies. Next was score 0, when no ptc showed complete membranous staining. Plasma entrapment was taken as Negative. 28.5 % of allograft biopsies were Negative with diagnosis of Chronic TMA, CNI toxicity, Recurrent/ denovo glomerulonephritis and ATN. Score 2 (Focal, with staining in 10-50 % of ptc’s) was seen in 21.5 % of allograft biopsies comprising of AMR and Mixed rejection. Score 3 (diffuse, marking > 50 % of ptc’s) was seen in 10 % of biopsies with diagnosis of AMR and Chronic active AMR. Two ABO incompatible biopsies showed 3+ staining. We did not some across C4d Negative AMR.

Conclusions:
In our cases of morphologic AMR, which were treated accordingly- usual C4d score was either 2+ or 3+ in all the cases. According to literature, any peritubular capillary marking with C4d by IHC technique should be considered positive. We conclude that 1+ C4d score should be interpreted with caution along with other morphologic features like glomerulitis and peritubular capillaritis to make a diagnosis of AMR as 1+ score was predominantly seen in ATN and few cases of ACR as well. We came across 2 cases of C4d positive Acute AMR where DSA was Negative.
Clinical and morphological spectrum of primary non-proliferative glomerular diseases in western Indian population

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Background:
Glomerulopathies present as varied spectrum of clinical diseases. However in India, we lack substantial morphological database of biopsy proven Glomerulopathies from all the regions.

Aim & Objectives:
We are reporting a retrospective analysis of clinical and morphological spectrum of primary non-proliferative glomerular diseases in western Indian population.

Materials & Methods:
A retrospective analysis of native renal biopsy reported between January 2011 to December 2015 at our centre were reviewed along-with clinical and laboratory investigation data. All renal biopsy were processed for light & immunofluorescence study (IgA, IgG, IgM, C3, C1q, Kappa & Lambda). Electron microscopy (EM) study was carried out in the selected cases. Clinical findings evaluated were presence of oedema (generalized/ localized), haematuria, hypertension, diabetes, or any other significant diseases. Laboratory data collected were serum creatinine, urinary protein/creatinine ratio, 24 hours urinary protein and relevant serological examination.

Result:
Total 3740 renal biopsies reported at our centre from January 2011 to December 2016. Out of 3740, 2814 were native renal biopsies of which 851(538/313, M: F) cases were of non-proliferative morphology. In present study most common disease was Focal & Segmental Glomerulosclerosis - FSGS (36.7%), followed by Minimal Change Disease - MCD (35.7%) and Membranous Nephropathy – MN (27.6%). In paediatric age group (< 16 years of age), MCD (53.5%) was most common whereas in adult age-group (16-65 years), FSGS (36.8%) was predominant and in geriatric age-group (> 65 years) MN (49.0%). Generalised oedema is more common in FSGS followed by MCD. Haematuria detected in 6.6, 5.8 & 3.8% cases of MCD, FSGS & MN respectively. Hypertension was more common in FSGS followed by MN. Preceding Nephrotic syndrome was significant in FSGS while Hypothyroidism was more common in MN. Mean S. Creatinine was highest in FSGS, followed by MN, and MCD respectively, while highest UPCR was in MCD followed by MN and FSGS respectively.

Conclusion:
In paediatric age-group, MCD was more common at our centre whereas overall FSGS was the most predominant finding in our patient population which correlates with the few studies available from Indian subcontinent. However considering the ethnic heterogeneity in Indian population, centralized renal biopsy database is need of the time.
The GRACE IgAN Study: Epidemiology and Longitudinal Follow up
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Background:
In India about 30-40% of IgA Nephropathy (IgAN) patients (pts) have nephrotic syndrome and renal dysfunction at presentation.

Aims & Objective:
To study the epidemiology of IgAN. To study the associations among low and high risk groups scored by Absolute Renal Risk Score (ARR) criteria.

Material & methods:
This is a single center prospective longitudinal cohort study in South India started from March 2015. 111 out of 154 adults (≥18 yrs) pts diagnosed to have IgAN among 1400 native kidney biopsies till March 2016, were included in the study. Low Risk (LR): IgAN patients with an ARR score of ≥23 points. High Risk (HR): IgAN patients with an ARR score of <23 points. Rapid Progresser (RP): IgAN patients with ≥5ml/min/1.73m2/year fall in CKD EPI eGFR. Slow/Non Progresser (S/NP): IgAN patients with <5ml/min/1.73m2/year fall in CKD EPI eGFR. End of study outcome (EOS): Composite end-point of 50% decline in eGFR with eGFR <10ml/min/1.73m2, RRT or death whichever occurs earlier. Immunosuppression was given in pts with ≥1g/day of proteinuria and/or renal dysfunction.

Results:
60 (54.1%) & 51 (45.9%) were in the LR and HR groups respectively. Mean 24 hr urine protein was 2.8 +/- 2.1g/day and mean CKD EPI eGFR was 45.4 +/- 31.5 ml/min/1.73m2. MEST scores at baseline: M1 11%, E1 45%, S1 84%, T1 34% and T2 57%. The median duration of follow-up was 10 months.

Conclusions:
Our IgAN pts have significantly more systemic and tissue inflammation as evidenced by levels of serum hsCRP, serum Ig G and proportion of Endocapillary Proliferation in MEST score. One third (33%) of IgAN pts were Rapid Progressers - distributed similarly in both low & high risk groups. On multivariable analysis, predictors of EOS outcome were female gender, low hemoglobin, nephrotic proteinuria and MEST T2 score. Higher hs CRP levels had a protective effect. ARR risk score is a good predictor of EOS outcome but a poor predictor for rate of progression. Steroid treated Rapid Progressors had good decline in proteinuria that is sustained over time. Significant number of non-serious infections & drug toxicities were present in those receiving IS.
Clinicopathological Spectrum Of Thrombotic Microangiopathy in renal biopsies: A Retrospective Study of 5 Years

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Background:
Thrombotic microangiopathy (TMA) is a systemic disorder with predominant renal involvement. Apart from the common etiologies including hemolytic uremic syndrome (HUS) and Thrombotic thrombocytopenic purpura, there are various secondary causes.

Aims & Objectives:
To analyze the clinicopathological spectrum and etiological factors of TMA in renal biopsies.

Material & Methods:
A retrospective evaluation of renal biopsies received from 2012-2016 was performed. All histology reports were evaluated for the presence of features suggestive of TMA. All such biopsies were reviewed for the presence of TMA. Those cases with fibrin thrombi in glomeruli and/ fibrinoid necrosis of vessel wall were included for the study. Clinical and laboratory data was collected from the patient records.

Results:
A total of 35 biopsies showed features of TMA comprising of 31 native and four transplant biopsies. The age ranged from 9 to 65 yrs. TMA in transplant was due to CNI toxicity (n=2), antibody mediated rejection (n=1) and T-cell mediated rejection (n=1). Malignant hypertension (n=12) was the most common etiology in native biopsies followed by postpartum HUS (n=7), lupus nephritis (n=6) and atypical Hemolytic uremic syndrome (n=6). Females were more commonly affected. Most (93%) patients presented with proteinuria and active sediments (63%). The mean S. Creatinine was 5.7 mg/dl. The average platelet count was markedly low in atypical HUS (86.7x103/cmm) followed by postpartum HUS (127.4x103/cmm) and lupus nephritis (137.2x103/cmm). It was normal in malignant hypertension. The average S. LDH was markedly high in atypical HUS (Mean 3293u/l) followed by postpartum HUS. Fragmented RBCs were seen in peripheral blood of only three patients.

Conclusion:
Renal TMA has a wide clinicopathological spectrum and etiological factors. However, the typical clinical features of TMA including low platelet count, raised LDH and fragmented red blood cells are seen only in a few patients. Therefore, careful evaluation of renal biopsies and high degree of suspicion is essential for prompt diagnosis and management.
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Coincidental Non-neoplastic Lesions in Neoplastic Nephrectomy Specimens: a retrospective analysis

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Background:
Non-neoplastic changes are often present in the renal parenchyma of nephrectomies for renal neoplasms. Pre-existing non-neoplastic renal diseases or lesions may influence patient renal function after tumor removal. However, these are often missed and not mentioned in routine pathologic reports.

Aims and Objective:
To determine the incidence and spectrum of the non-neoplastic lesions in renal tissues obtained during radical nephrectomies for renal cell carcinoma.

Material and Methods:
A retrospective study of 50 radical nephrectomy specimens received between November 2015 and October 2016 and diagnosed with renal cell carcinoma was performed. Non-neoplastic renal parenchyma was evaluated for lesions in glomeruli, tubules, interstitium and blood vessels, and recorded semi-quantitatively. The parenchyma adjacent to the tumor was excluded for evaluation. Histochemical stains were performed when indicated. Clinical and laboratory data was recorded from patient records.

Results:
Out of the total 50 cases (36 males, 14 females), the most common tumor type was clear cell carcinoma (n=28, 56%) and the most common presenting clinical complaint was hematuria (n=30, 60%). 21 nephrectomies (42%) showed glomerular alterations, including mesangial proliferation (n=6), focal segmental glomerulosclerosis (n=5), diabetic lesions (n=5), hypertensive changes (n=2), crescents (n=3) and filaria (n=1). Tubulo-interstitial changes were found in 32 (64%) and vascular changes in 24 (48%) nephrectomies. Among the various histologic lesions found, tubulo-interstitial lesions were the most common (n=32, 64%) followed by vascular lesions (in the form of arterial intimal widening, fibrosis and hyalinosis) (n=24, 48%). 18 cases (36%) had unremarkable renal parenchyma.

Conclusions:
While non-neoplastic changes are frequent in nephrectomy specimens, they are often unrecognized. The adequate examination of non-neoplastic renal parenchyma is an important tool in recognizing patients at risk for progressive renal disease after nephrectomy and could be an essential step in providing preventive or treatment measures to patients undergoing nephrectomy for neoplastic processes.
Clinicopathological characteristics of focal segmental glomerulosclerosis (FSGS) combined with thin glomerular basement membranes

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Background:
Recent research shows that collagen IV mutations (COL4A3, COL4A4 and COL4A5 gene mutations) cause whole spectrum of disease, ranging from benign familiar haematuria to early onset Alport’s syndrome (AS). Somewhat new members in this spectrum are late onset FSGS that develops on the top of thin glomerular basement membrane nephropathy (TBMN) and familial FSGS.

Aims & Objective: Aim of this study was to investigate clinicopathological characteristics of focal segmental glomerulosclerosis (FSGS) combined with thin glomerular basement membranes. Material & methods We performed retrospective analysis based on medical records and found 30 patients (15 males and 15 females, median age 46 years) who underwent renal biopsy between 2003 and 2016 and were diagnosed with FSGS combined with thin glomerular basement membranes found on electron microscopy.

Results: At the time of renal biopsy most of patients presented with asymptomatic proteinuria and/or haematuria or with nephrotic syndrome. Median 24 hour proteinuria rate was 3.22 g (0.31-19.8 g) and median creatinine level was 117 µmol/L (56-430 µmol/L). There was no family history of AS or TBMN. However, one patient had positive family history for haematuria and 4 for end stage renal disease. There were 15 cases of primary and 15 cases of secondary FSGS and histological types were: perihilar (36.7 %), classical (36.7 %), tip-lesion (13.2 %), cellular (6.7 %) and collapsing type (6.7 %). Median portion of globally sclerosed glomeruli was 17.3 %, segmentally sclerosed glomeruli 15.7 % and interstitial fibrosis and tubular atrophy 16 %. Nodular hyalinosis in more than one arteriole or hyalinosis in full circumference was present in 57.1 % of cases and moderate or severe arterial fibrointimal thickening was present in 22.2 % of specimens. Median of average glomerular basement membrane thickness was 219.5 nm (133-254 nm). There were no lamelation or conspicuous variations in GBM thickness.

Conclusions: We have presented clinicopathological characteristics of focal segmental glomerulosclerosis (FSGS) combined with thin glomerular basement membranes. Our further plan is to test these patients for collagen IV mutations within our project ‘Genotype-Phenotype correlation in AS and TBMN’ to confirm possible underlying genetic background.
Spectrum of renal pathology findings in patients with ABO incompatible renal transplantation: A single centre study

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Background:
Due to limited number of donors, patients with ESRD have a long waiting time for transplantation throughout the world. To expand the donor pool ABO incompatible renal transplantation is being done. The literature has shown fairly good and comparable outcome in these patients as compared to ABO compatible renal transplantation.

Methods:
In our centre 56 ABO incompatible renal transplants were conducted between January 2013 to April 2016. Eighteen of these patients underwent indication renal graft biopsy following transplantation.

Results:
There were 12 female recipients and 44 male recipients. The age ranged from 14-65 years. All the patients received ATG for induction except 4 patients who received Basiliximab. The donor age ranged from 19 years to 65 years. There were 12 male donors and remaining were female donors. The indication for biopsy was renal graft dysfunction. Five cases were diagnosed as acute antibody mediated rejection (ABMR), two cases as acute cellular rejection (ACR), 1 case as combined AMR with ACR. Three cases were diagnosed as thrombotic microangiopathy (TMA) and three cases had acute tubular injury without evidence of rejection. Four cases had evidence of acute calcineurin inhibitor toxicity. All the cases of ABMR and 2 cases of TMA had diffuse C4d positivity in peritubular capillaries. Of the remaining patients, C4d was negative in 3 patients. C4d positivity in other patients ranged from 10-20%. On follow-up 3 patients died and two patients became dialysis dependent. Remaining patients have stable graft function.

Conclusions:
ABO incompatible renal transplantation is important to expand donor pool with good post transplant outcome. Acute antibody mediated rejection was more common than acute cellular rejection with majority occurring within 1st two months after transplantation in our subset of patients. Diffuse C4d positivity was seen in patients with acute antibody mediated rejection as compared to focal c4d positivity in patients without evidence of rejection (due to accommodation).
Morphology of Resolving Polyomavirus Nephropathy in Renal Transplant Patients

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Institute Of Pathology

**Aim & Objective:**

Reduction of immunosuppression is a common therapeutic strategy of polyomavirus nephropathy (PVN) but may be associated with rejection. The study aimed to evaluate the morphology of PVN in renal biopsies after reduction of immunosuppression.

**Methods:**

Eight of 241 patients who received kidney transplant between January 2012 and December 2015 presented with polyomavirus (PV) viremia and biopsy proven PVN. Morphological evaluation according to the Banff criteria and correlation with viremia after immunosuppression reduction was performed.

**Results:**

PVN grades A and B were diagnosed on average 4.9 months post-grafting in 1 and 7 patients, respectively. All 8 patients presented with post-transplant complications: delayed graft function (two), prior acute rejection (four), obstruction (one) and prolonged ureteral stent placement (one patient). Rebiopsies after immunosuppression reduction revealed a significant increase of tubulitis and interstitial inflammation score in all 7 re-biopsied patients. Follow-up biopsies revealed minimal interstitial inflammation and clearance of PV after 6-12 months in 5 patients (63%). In those patients, renal function returned to baseline. One patient with persisting PV in serum and kidney showed a decrease of interstitial inflammation but up to 50% scarring was seen. Rejection occurred in three patients (37%): In the first, T-cell vascular rejection was seen; after treatment with pulse corticosteroid therapy, there was regression of rejection and clearance of PV from kidney and serum 12 months after rejection. Renal function returned to baseline. The second patient presented with subclinical antibody-mediated rejection (detection of donor specific antibodies (DSA), unaltered baseline creatinine). Following plasmapheresis, DSA decreased but scarce PV-positive cells remained on follow-up biopsy 3 months later. The third patient required haemodialysis due to florid PVN, then immunosuppression was stopped. He achieved clearance of PV from serum and kidney but signs of rejection occurred, confirmed by tubulointerstitial and vascular rejection on the explanted kidney.

**Conclusion:**

PVN associated interstitial inflammation and tubulitis cannot be differentiated morphologically from T-cell tubulointerstitial rejection. Significant interstitial inflammation and tubulitis in PVN under low dose immunosuppression might represent immune reconstitution injury, which is spontaneously reduced after successful PV clearance from the serum and kidney. Concomitant rejection in PVN patients on low immunosuppression might be efficiently treated with transient pulse immunosuppressive therapy.
End organ damage due to Lupus vasculopathy/vasculitis independent of lupus nephritis status: An autopsy series from nephropathologist's perspective

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BACKGROUND:
In SLE, antibodies formed against various self-antigens circulate as immune complexes and their deposition in tissues cause injury. Although immune complexes circulate in blood, Lupus vasculopathy/vasculitis is not present in all cases. In this series, we demonstrate an association between Lupus vasculopathy/vasculitis and end organ damage independent of Lupus nephritis activity status.

AIMS AND OBJECTIVES:
To describe Lupus vasculopathy/vasculitis and end organ damage in kidneys and other organs with clinical correlation.

MATERIALS AND METHODS:
In this retrospective study, medical autopsies of SLE patients between 2006-2016 were evaluated for detailed gross and microscopic vascular lesions with resultant end organ damage. Clinical data was analysed. RESULTS Autopsy series of 20 cases was studied. Four cases had vasculitis, two had vasculopathy and three had luminal thrombosis. Two cases with vasculopathy had active renal disease (class IV A/C), renal dysfunction (creatinine 6.7 and 7.1mg/dl) and renal infarcts despite treatment. Extra-renal infarcts affecting brain, pancreas and heart were present. Four cases with vasculitis had active renal disease(class III and IV)in two, creatinine (2.3 and 4.6 mg/dl), and inactive in two(class I and II), creatinine (0.8 and 1.2 mg/dl). Vasculitis also affected brain, GIT, pancreas and adrenals. One case with class II Lupus had renal infarcts and normal creatinine. Three cases with APLA had active and inactive renal disease with increased creatinine in active cases. Two cases had renal thrombosis. NBTE alongwith embolic infarcts in spleen present in two and brain infarct in one case.

CONCLUSION
Cases with vasculopathy/vasculitis with active renal disease despite treatment indicate resistance and significant end organ damage suggesting vasculopathy is not innocuous lesion. Cases with APLA with end organ damage had NBTE, stressing need for cardiac work up in SLE. Vasculitis in inactive renal disease highlights its importance in SLE. This autopsy series emphasizes to include vascular lesions in assessing lupus activity and in renal histology.
Clinicopathological Study of C1q nephropathy: is it a defined diagnostic entity?

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Background:
C1q nephropathy is a heterogeneous entity with dominant/co-dominant deposits of C1q in renal biopsies, a diagnosis made after exclusion of other diseases including Lupus Nephritis (LN) and IgA nephropathy (IgAN). It usually manifests as steroid resistant Nephrotic syndrome. Glomeruli may show MCD-like pattern, FSGS or mesangial proliferation. It remains to be seen if C1q nephropathy is a single entity or diverse conditions with unifying feature of C1q deposition.

Aim:
We performed a retrospective study to analyze clinical presentation, histological spectrum, type of immune deposits on immunofluorescence in patients with C1q nephropathy.

Materials and methods:
Renal biopsies from January 2011-June 2016 were retrospectively identified for presence of C1q deposits of ≥2+ intensity on immunofluorescence (scale: 0-4) after exclusion of LN, IgAN. Histological patterns of glomerular involvement, tubulointerstitial and blood vessel changes were studied. Data for patterns, intensity of IgG, IgM, IgA, C3 and C1q deposits on immunofluorescence was available in all biopsies. Clinical features, laboratory parameters, complement levels, autoantibodies, viral serology and urine analysis were recorded.

Results:
Of 282 renal biopsies with ≥2+ mesangial deposition of C1q, a diagnosis of C1q nephropathy was made in 31 patients after excluding LN (n=216), IgAN (n=22) and other diagnosis (n=13 [membranous nephropathy, diabetic nephropathy and amyloidosis]). The age range was 3-70 years (M:F – 1:0.8); 2/3rd patients were < 30 years. Most patients (71%; n=22) presented with nephrotic syndrome (steroid dependent: 2, steroid resistant: 4). The other presentations were nephritic syndrome (n=4), nephrito-nephrotic (n=1) or advanced renal failure (n=4). The most common morphological pattern was MCD-like normal appearing glomeruli (n=13, 39.4%), mesangiotrophic GN (n=7, 21.2%), FSGS (n=6, 18.1%), MPGN (n=2, 6%) and nodular glomerulosclerosis (n=2, 6%). Concomitant capillary wall deposits of C1q were seen in two biopsies. IgM (1-2+), C3 (1-2+) were seen in 20 (65.4%), 11 (41.9%) biopsies respectively. Electron microscopy available in 4 cases showed foot process effacement and microvillous transformation. The mean 24-hour proteinuria was 5.1 ± 0.8 g/day, mean serum creatinine was 1.2 mg/dl and active urinary sediments were found in 1/3rd (n=10) patients. All cases had normal serum complement (C3: 140 ± 41 mg/dl, C4: 39 ± 16 mg/dl).

Conclusion:
C1q nephropathy usually presents as nephrotic syndrome with MCD-like pattern followed by mesangiotrophic GN, FSGS. Whether it is a distinct entity or varied manifestation of MCD or FSGS-like disease is still unclear, however, our findings suggests towards the later.
Significance of Peritubular Capillary Density in Early Renal Allograft Biopsies

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Background and aims:
Chronic renal allograft injury is known to result in decrease in peritubular capillary density. However, information about the changes in peritubular capillary density in acute allograft rejection is not clear. This study was performed to analyze the diagnostic utility of peritubular capillary density in early (<1 year) renal transplant biopsies by morphometry.

Material and methods:
Graft renal biopsies of consecutive 50 patients with duration of transplant ≤ 1 year of transplant, from January 2016 to October 2016 were included. PTC morphometry was performed after immunohistochemistry by endothelial marker CD31. The number of PTCs and tubules were assessed in at least five successive images of each biopsy acquired at 20X magnification using an Image Pro Plus 6.1 software and an average was calculated. Area of PTCs per unit area of the biopsy was also measured.

Results:
Of the 50 early transplant biopsies, rejection was diagnosed in 13 biopsies. The duration after transplant was (95.8 ± 93.4). In rest 37 biopsies with no evidence of rejection, the duration post-transplant was (93.52 ± 90.2). The average PTCs per field (0.32 ± 0.59) and the PTCs per tubule (1.98 ± 0.48) in rejection were higher but not statistically significant as compared to no evidence of rejection (ptc per tubule (1.97 ± 0.45) ptc's per field (0.27 ± 0.55). The PTC numbers did not show significant association with age of recipient, time to transplant, C4d positivity and presence of DSA. The mean PTC area was also not significantly different between the two groups.

Conclusion:
This study shows that PTC density in early renal allograft biopsies does not appear to have diagnostic utility in determining the etiology of acute renal allograft dysfunction.
A prospective study of renal involvement in patients with Primary Sjögren's syndrome. A study of 70 patients

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Background:

Primary Sjögren’s syndrome (pSS) is known to be associated with renal disease. However, few studies have prospectively evaluated renal involvement in pSS. The present study evaluated renal involvement in patients with pSS followed up prospectively for a period of one year.

Aims and Objectives:

To assess the spectrum of renal involvement, identify its potential association with demographic, clinical and immunological profile as well as to evaluate the impact of renal involvement on short term disease prognosis.

Methods:

Patients satisfying 2002 AECG criteria for pSS were included in the study and evaluated for renal involvement including renal function tests, urine examination, Arterial blood gases, Urine pH and urine acidification test (UAT) if indicated. Patients were followed up prospectively for one year for disease activity, development of renal involvement and dysfunction (eGFR). Patients were divided into two groups: those with renal involvement (renal group) and those without (non-renal group). Demographic, Clinical and laboratory parameters were compared between the two groups.

Results:

One hundred and seventy eight patients were screened of which 70 fulfilling the inclusion criteria were enrolled. Thirty five (50%) had renal involvement. They were significantly younger (37.6 ± 1.1 vs. 40.6 ± 10.8) and had fewer articular symptoms (40% vs 80%, p = 0.001) than those without renal involvement. Of the 35 patients, 29 had renal tubular acidosis (RTA), 1 had chronic kidney disease (CKD), 1 had nephritis, 1 had proteinuria. Renal biopsy was performed in 17 patients. The most common histological finding was tubulo-interstitial nephritis seen in 9 patients, characterized by invasion of mainly mononuclear lymphocytes. We found that these cells are predominantly CD4+ T-cells with lesser population of CD8+ T-cells. Three patients had features of IgA nephropathy, one showed focal segmental sclerosis, two of each patients showed acute tubular necrosis, interstitial fibrosis and tubular atrophy (IFTA). There was mild but significant increase in eGFR in renal group (p = 0.0373) compared to the non renal group (p = 0.24).

Conclusion:

Renal involvement in pSS is an under-recognized entity. These patients are younger and have less articular symptoms. Most common presentation is RTA with tubulointerstitial nephritis on biopsy. Without specific treatment, there is a gradual progressive decline of renal function. Renal biopsy is recommended in all patients with renal involvement.
CLINICOPATHOLOGIC SPECTRUM OF GLOMERULAR DISEASES IN A TERTIARY CARE HOSPITAL

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BACKGROUND:

Glomerular diseases are a leading cause of end stage renal disease globally. The prevalence of this disease varies according to geographical, etiological and socioeconomic differences. Biopsy proven renal diseases provide an accurate tool for clinical practice and investigation.

OBJECTIVE:

To evaluate the histopathological spectrum reported in native kidney biopsy and analyze the clinicopathological correlation in these biopsy proven renal disease (BPRD)

MATERIAL AND METHODS:

A retrospective review of consecutive native renal biopsies performed on patients at a tertiary care hospital in Lucknow was undertaken. All renal biopsies were studied by light microscopy and findings were correlated with immunofluorescence staining.

RESULTS:

A total of 106 consecutive kidney biopsy cases were included in the study. Male female ratio was 1.6:1 and age of presentation varied from 6 – 70 years with maximum number of cases belonging to third and fourth decade. The most common clinical syndrome as an indication for renal biopsy was Nephrotic syndrome. Secondary glomerulonephritis accounted for only 11% of the biopsy proven renal cases and the rest were primary glomerulonephritis. Minimal change disease (23.4%) was the commonest Primary glomerulonephritis followed by focal segmental glomerulosclerosis (13.8%) and membranous nephropathy (12.6%). Diabetic nephropathy was the commonest secondary glomerulonephritis followed by lupus nephritis.

CONCLUSION:

Histopathological examination with light microscopy and immunofluorescence techniques and correlation with clinical, biochemical and serological markers as done in this study, have proved useful for the accurate diagnosis of glomerular diseases. In our study, Minimal Change Disease was the most common biopsy proven glomerular disease with presentation not only in the childhood and adult nephrotic syndrome patients but also in the elderly group. Key words: kidney biopsy, spectrum, glomerular lesions.
Alternate Complement Pathway and Genetic Analysis of CFH and CFHR5 Genes in C3 Glomerulopathy

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BACKGROUND:
C3 glomerulopathy (C3GP i.e. dense deposit disease (DDD) and C3 glomerulonephritis (C3GN)) results from dysregulation of alternate complement pathway (ACP) due to genetic aberrations and/or generation of auto-antibodies to various regulatory factors. There is a lack of Indian data related to C3GP.

AIMS & OBJECTIVE:
To analyse ACP and CFH/CFHR5 gene abnormalities in the patients of C3GP.

MATERIAL & METHODS:
There were 132 cases of C3GP (2007-2015), of which 61 (DDD-32, C3GN-29) cases were enrolled for this study. Serological workup includes estimation of C3, C4 by nephelometry and complement factor H (CFH) levels, complement factor B (CFB) levels, alternate pathway functional assay (APFA), autoantibodies to factor H & B (FH,FB) and C3 convertase (C3NeF) was done by ELISA. CFH and CFHR5 genes (all exons) were screened for genetic mutations (PCR followed by Sanger sequencing). Patients were followed for 1-4 years.

RESULTS:
ACP dysregulation was confirmed by low APFA in all the cases. Low C3 and normal C4 was observed in 97% and 94% cases of DDD which was almost similar to 86% and 90% in C3GN. FH and FB levels were low in 58% and 20% of DDD which was also almost similar to 44% and 13% respectively in C3GN. Autoantibodies to FH were less common in DDD (13%) than C3GN (24%) and those to FB were equally present (17%) in both the groups. C3Nef was almost twice as common in DDD (44%) than in C3GN (20%). In DDD and C3GN, we found 5 already known SNPs (rs800292, rs1061147, rs1061170, rs2274700, and rs35292876) in exon 2, 7, 9, 10 and 13 of FH and 2 SNPs (rs9427662 and rs140691305) of CFHR5 at exon 1 and 3 respectively. Two patients died within one year, and most of the patients progressed to ESRD within 1.5 to 2 years.

CONCLUSION:
Autoantibodies to various regulatory factors were present in 74% of DDD and 59% of C3GN. C3Nef was twice as common in DDD whereas those to FH were more frequent in C3GN. FB autoantibodies were equally common in both groups. Genetic analysis confirmed the active involvement of CFH and CFHR5 genes in C3GP.
Atypical Hemolytic Uremic Syndrome: A 5 year experience from a tertiary care centre in North India

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Introduction:
Atypical haemolytic uremic syndrome (aHUS) presents as rapidly progressive renal failure caused by diverse etiologies, frequent need for dialysis/plasmapheresis, and progression to end-stage renal disease. aHUS in the West is commonly due to genetic mutations of the complement proteins. However, literature from India suggests that anti complement antibodies are more frequent.

Aims & objective:
To characterize clinical presentation, etiology and outcome of aHUS in children.

Methods:
Twenty-six aHUS patients were enrolled in this single-center retrospective study (2012-2016). Along with baseline demographic profile and laboratory profile, complement factors (serum C3, C4, CFH, anti-CFH antibodies, CFI and CFB) were estimated. Results Median age at onset of aHUS was 90 (60, 111) months with male (65.4%) predominance.

Results:
Diarrhoea was present in 6 (23%) and neurological involvement was present in 13 (50%). Hypertension was seen in 20 (76.9%) patients. Mean Haemoglobin was 4.86 ± 1.21 mg/dL, median platelets (IQR) 25000 (10000, 67250) and median LDH level (IQR) 2944 (2067, 4535). Blood urea and serum creatinine were median (IQR) 230 (169, 284) mg/dl and 4.8 (3.5, 6.5) mg/dl respectively. Hematuria and proteinuria were present in 21 (81%) and in 20 (77%) patients respectively. C3 & C4 were 84.6 ± 28 mg/dl, 24.3 ± 12.3 mg/dl respectively. Out of 26, 11 (42%) patients underwent renal biopsy, which was reported as thrombotic microangiopathy on histopathological examination. Antifactor H antibody was elevated in 12 (57.2%) out of 21 patients. Antifactor H level median (IQR) was 296.5 (4.1, 369.3) AU/mL (reference range < 27 AU/ml). Median (IQR) CFI, CFB and CFH were 38.0 (34.0, 41.5), 774 (545, 860) and 552 (145, 758) respectively. Functional assessment of alternate pathway was performed in 7 patients and was found to be depressed in 6/7 patients. Delay between first symptom and treatment was median (IQR) 10 (8, 27) days. Plasmapheresis was done in 17 (65.4%) patients with median plasma exchange cycles required for remission were 7 (5.8, 11.3). Immunosuppressant was given to 13 (50%) patients. Out of 26 patients 22(84.6%) achieved remission, 3(11.3%) died and 1 never achieved remission.

Conclusions:
In our cohort of aHUS, diarrhoea was presenting symptom in few patients. Neurological involvement was also common. Anti-factor H antibody mediated aHUS was the most common cause of aHUS in our cohort.
A clinicopathological profile of renal disease in adults at a tertiary care hospital

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Background:
Renal biopsy is an invaluable method used in evaluation of patient with renal disease. Light microscopy and immunofluorescence findings make it possible to establish accurate diagnosis, get information on evaluation and prognosis of disease process and develop approach in treatment of renal disorders.

Aims and objectives:
We in this study tried to evaluate the profile of renal biopsies received in a year and correlated the findings of routine microscopy to immunofluorescence (IF) staining.

Material and method:
We did a prospective study of 110 kidney biopsies received from June 2015 to July 2016 at Indira Gandhi Medical College, Shimla. H&E and special stained sections were examined and findings were correlated with immunofluorescence staining.

Results:
The study included 110 cases. The mean patient age was 41 + 14.9 ranging from age group 18-70 years. The male:female ratio was 1.68:1. The most common indications of renal biopsy were nephrotic syndrome (48.2%), followed by rapidly progressive renal failure (18.2%), chronic renal failure (11.8%). Primary glomerulonephritis (PGN) comprised 65 (59.1%) of the total patients. Among the PGN cases, the most common one was IgA Nephropathy (26.1%), followed by membranous glomerulonephritis (24.6%), focal segmental glomerulosclerosis (13.8%). Secondary glomerular disease (SGN) accounted for 22 (20%) of the total cases. The most common SGN was lupus nephritis (63.6%), followed by equal distribution of amyloidosis (18.2%) and diabetic nephropathy (18.2%). Tubulointerstitial disease and vascular disease comprised 19 (17.3%) and 04 (3.6%) of total patients respectively.

Conclusion:
Renal biopsy and IF appears to be an important tool for diagnosing glomerular diseases. This study showed Prevalence of IgA nephropathy is increasing in Himachal Pradesh as compared to other published Indian studies. Immunofluorescence helped in making diagnosis where light microscopy findings were equivocal and it also helps in understanding immunological mechanisms involved in various renal lesions. Hence it is less time consuming and effective method in diagnosing glomerular diseases on renal biopsies. Key words: kidney biopsy, immunoflorescence, glomerulonephritis, IgA nephropathy.
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Title: MONOCLONAL GAMMOPATHY OF RENAL SIGNIFICANCE: HOW WIDE IS THE MORPHOLOGICAL SPECTRUM?

Abstract

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INTRODUCTION:

Renal lesions are frequently seen with monoclonal gammopathies. First clue to myeloma often results from the workup of unexplained renal diseases. Many of these cases with renal involvement fulfil the criteria for MGUS but not overt myeloma, are classified as MGRS.

OBJECTIVE:

To document spectrum and prevalence of MGRS out of all the monoclonal renal diseases.

MATERIAL AND METHODS:

The biopsy and clinical records from Department of Histopathology, Nephrology and Medicine, PGIMER were searched from January 2006-October 2016.

RESULT:

All monoclonal diseases diagnosed on renal biopsies and fulfilled the criteria for MGRS were analysed. There were 178 monoclonal diseases diagnosed on renal biopsies and 70(39%) had renal symptoms as first manifestation. Male to female ratio was 2.3:1 and average age at presentation was 53.5 years. Lesions were tubulopathic in 98 cases (94 cast nephropathy and 4 cases of tubulopathy) and glomerulopathic in 80 cases (AL amyloidosis (n=41), MIDD (n=19), PGNMID (n=7), HCDD (n=2), monoclonal MGN (n=2), cryoglobulinemia (n=4), HLCDD (n=1), monoclonal C3GP (n=2), immunotactoid glomerulopathy (n=2). On evaluation, most of the tubulopathic cases and 2/3 of amyloidosis had multiple myeloma, whereas most of the glomerulopathic cases eg AL amyloidosis (n=7), MIDD (n=4), HCDD (n=2), PGNMID (n=7), cryoglobulinemia (n=2), monoclonal C3GP (n=2), immunotactoid (n=2), monoclonal IgA (n=1) qualified for MGRS. Although the clinical presentation of MIDD and PGNMID were almost similar, the outcome of PGNMID were better as compared to MIDD.

CONCLUSION:

Almost one third (28/80 = 35%) of glomerulopathic cases of monoclonal renal deposition disease do not fulfil the criteria of myeloma, hence categorized as MGRS. Renal manifestation is their first presentation, hence vigilant immunofluorescence interpretation is the key factor. The pathologic spectrum of renal diseases in this setting is wide; a comprehensive kidney biopsy analysis including EM is required to determine the exact nature of the disease. These renal diseases are invariably associated with high morbidity and poor renal outcome.
Morphological patterns of renal injury in tropical diseases- an autopsy series. Aravind S1, Muralidaran C1, Shilpi Thakur1, Raja Ramachandran2, KL Gupta2, R K vashista1, Ritambhra Nada1
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Introduction:
Malaria, filaria, dengue, scrub typhus, leptospirosis, acute gastroenteritis are important tropical diseases particurlary in India. These tropical diseases are considered as most important cause of acute kidney injury, particularly in younger individuals who do not have any comorbidities. Histopathological findings of these tropical diseases in kidney varies and less studied.

Aim and objectives:
To study the different morphologic forms of renal injury in patients with tropical infections.

Materials and methods:
20 Autopsy cases in patients with tropical infections were retrieved. Clinical details were taken from medical records. Pattern of histopathological injury in kidneys were recorded.

Results:
Of 20 cases, number of cases with clinical diagnosis of scrub typhus (4), malaria(4), dengue(4),leptospirosis(4),filarial(2),diphtheria(1),chickenpox(1) were included. Age varies from 16 to 65 years. 12 were male and 8 were females. Creatinine was increased in 9 cases (1.5 to 8.5mg/dl ). Mesangiolysis and endotheliosis were most common form of glomerular injury seen in 6and 4 cases respectively, whereas glomerulitis and fibrin thrombi were seen only in two cases. Endotheliosis was seen only in scrub typhus and dengue. Although ATN seen in all cases, severity varies from cases to cases. Severe ATN was noted in malarial cases. Pigment cast was seen in 4 different cases whereas bile cast seen in single case of leptospirosis. Vacuolar nephropathy was seen in 4 cases. Acute interstitial nephritis (AIN) was seen in 5 cases. Interstitial oedema and hemorrhage were seen in 3 and 7 different cases respectively. Peritubular capillary dilatation and margination were seen 8 cases. Thrombotic microangiopathy was seen in 2 cases. Amyloid and medullary angitis were seen in single case.

Conclusion:
There are no specific features in any of these clinical setting, however, constellation of features could be of diagnostic utility in appropriate setting eg morphological findings like endotheliosis, AIN were seen specifically in scrub typhus, dengue and leptospirosis.
EVALUATION OF ROLE OF COMPLEMENT PATHWAY AND PODOCYTE NUMBER IN PROLIFERATIVE GLOMERULONEPHRITIS

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BACKGROUND:

Proliferative glomerulonephritis encompasses a spectrum of disorders which can be either immune complex mediated or complement mediated. Immune complex mediated GN is caused by glomerular deposition of immune complex and Complement-mediated GN is caused by glomerular deposition of complement factors resulting from dysregulation of the alternative pathway (AP) of complement. Studies have evaluated the role of C4d, a by-product of classical and leptin pathway activation. Podocyte depletion is associated with proteinuria, which is adversely associated with progression of proliferative glomerulonephritis.

AIMS AND OBJECTIVES:

To correlate the expression of complement factors C4d, C3 and podocyte number per glomerular volume with clinicopathological spectrum of patients with proliferative glomerulonephritis.

METHODS:

Renal biopsies of 30 patients diagnosed with proliferative glomerulonephritis were reviewed and immunostaining for C4d and WT1 was done. The staining pattern and intensity of glomerular C4d in various proliferative glomerulonephritis was observed. The staining was graded from negative staining to highly intense positive glomerular staining. Immunofluorescence expression of C3 was semi-quantitatively graded from 0-4+. The number of podocytes per unit glomerular volume was calculated by morphometric analysis using Weibel Gomez formula on a single histologic section, which was described in previous studies in literature. Morphometric analysis was done using Image J software. The podocyte number was compared among the different subtypes of proliferative glomerulonephritis.

RESULTS:

In membranoproliferative glomerulonephritis (MPGN) C4d staining was of mild to moderate intensity, while in immune complex mediated proliferative glomerulonephritis and lupus nephritis C4d staining was moderate to highly intense. But in IgA nephropathy(IgAN) it was either absent or very minimally present. Podocyte number was significantly reduced in many cases of IgAN compared to other proliferative glomerulonephritis. In Post infectious glomerulonephritis podocyte number was higher than other proliferative glomerulonephritis. Membranoproliferative glomerulonephritis and lupus nephritis had intermittent range of podocyte depletion.

CONCLUSION:

Classical complement pathway activation highlighted by C4d immunostaining was demonstrated in all proliferative glomerulonephritis, with weak expression in MPGN and total absence or very minimal expression in cases of IgAN. Podocyte depletion is significantly seen in IgAN and to a lesser extent in other subtypes of proliferative glomerulonephritis.
Distribution of Natural Killer Cells in Renal Allograft Biopsies and its correlation with Banff score

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BACKGROUND:
Transplantation, though life-saving for patients with end-stage renal disease, is complicated by allograft loss/failure due to rejection. Our understanding of the etiological mechanisms, clinical and morphological features of rejection is still evolving. The role of innate immunity, in particular, natural killer cells (NK cells) in transplant biopsies is of interest.

AIMS AND OBJECTIVES:
To study the pattern of CD16 and CD56 positive NK cell infiltrates in allograft biopsies and correlate with clinical parameters and microcirculation injury scores.

MATERIAL AND METHODS:
Clinicodemographic data was collected for 50 consecutive allograft recipients whose renal biopsies were performed between January 2015 and December 2016. We analyzed the expression of immune markers CD4, CD16 and CD56 in the infiltrates within glomerular, peritubular capillary and interstitial compartments. These findings were correlated with clinical parameters and microcirculation injury scores.

RESULTS:
The study population included 9 cases of antibody mediated rejection (AMR) (4 cases of early AMR and 5 cases of chronic AMR), 16 cases of IFTA-NOS, 5 case of borderline cellular rejection, 4 cases of BK nephropathy and 4 cases showing acute cellular rejection. 12 patients showed allograft biopsies with no evidence of rejection and three biopsy specimens from healthy donors were included for comparison. Biopsies with no evidence of rejection (1-19 months post transplant; mean 5 months) showed interstitial CD4 and CD16 infiltrates with no evidence of glomerulitis. AMR biopsies, in addition showed significant CD4, CD16 infiltrates in the glomerular compartment which correlated with Banff score > g1. Compared to healthy donor, biopsies with no evidence of rejection and acute cellular rejection, AMR had significant number of CD56+ interstitial infiltrates. BK nephropathy biopsy showed significant CD16 + interstitial infiltrates.

CONCLUSION:
Post transplant renal biopsies show CD16 + interstitial infiltrates irrespective of evidence of rejection (Healthy donor kidney biopsies are devoid of CD16+ infiltrates). g≥1 correlated with presence of CD56+ interstitial infiltrate, suggesting association with AMR thus highlighting the role of NK cells as an early indication of transplant rejection.
Clinicopathological Profile of Pediatric Renal biopsies: A single center experience from North-India

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Abstract

Background: Renal biopsy is an important diagnostic modality for the assessment of kidney diseases in children. Available literature suggests great variation in epidemiology of renal histopathology all over world. However, there is a paucity of data on pediatric renal biopsies from low-income countries.

Aims and Objective:

To describe indication and clinico-histopathological correlation of pediatric renal biopsies from a tertiary care teaching institute from India Materials and methods: We analyzed all pediatric (age<18 years) renal biopsies performed in Sawai Man Singh Medical College, Jaipur, Rajasthan between Jan-2015 to Dec 2016. All renal biopsies were subjected to light and immunoflorescence microscopy. Clinical data were collected by retrospective review of medical records of the subjects. Allograft renal biopsies were excluded.

Results:

Out of 124 renal biopsies, 4 (3.2%) were inadequate and were not included in the study. The mean age was 11.08 ± 5.5 years with a male predominance (n=70, 58%). The commonest indication for renal biopsy was nephrotic syndrome (n=72, 60%) followed by acute nephritic syndrome (n=31, 25.8%), unexplained renal failure (n=7, 5.8%), hematuria (n=4, 3.3%) and rapidly progressive renal failure (n=3, 2.5%). Majority had primary glomerular disease (n=103, 85.8%) while 12 (10.0%) had secondary glomerular disease, three (2.5%) had acute tubular necrosis and two (1.7%) had acute tubulointerstitial nephritis. Among primary glomerular disease, minimal change disease (n=49, 40.8%) was commonest followed by post infectious GN (n=13, 10.8%), C3 dominant glomerulonephritis (n=12, 10.0%), focal segmental glomerulosclerosis (n=12, 10.0%), IgA nephropathy (n=7, 5.8%) and others (n=11, 9.2%). The commonest cause of secondary glomerular disease was lupus nephritis (n=9, 7.5%). The commonest cause of nephrotic syndrome was minimal change disease (68%), followed by focal segmental glomerulosclerosis (16.67%) and C3 dominant GN (4.17%). The commonest cause of acute nephritic presentation was post-infectious GN (32.2%), C3 glomerulopathy (25.8%), lupus nephritis (19.3%) and IgA nephropathy (16.1%).

Conclusion:

Our study provides insight on spectrum of renal diseases prevalent in this part of world. The commonest finding in pediatric renal biopsies being minimal change disease followed by post-infectious GN, C3 dominant GN and focal segmental glomerulosclerosis
Endothelial Nitric Oxide Synthase and Inducible Nitric Oxide Synthase Expression in Acute Antibody-Mediated Rejection

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BACKGROUND:
Advances in immunosuppression therapy modalities have significantly reduced acute cellular rejection rates. However, acute antibody-mediated rejection (AMR) is among the most important complications affecting long-term outcomes. Endothelial cells are targets for antibodies and endothelial cell injury leads to eventual tissue injury in AMR. Nitric oxide (NO) is an important cellular signaling molecule involved in many physiological and pathological processes. Nitric oxide synthase (NOS) has three isoforms that synthesize NO. Endothelial NOS produces NO in the vascular endothelium and plays crucial roles in regulating vascular tone, cellular proliferation, leukocyte adhesion, and platelet aggregation. Inducible nitric oxide synthase (iNOS) expression is induced in inflammatory diseases. Neuronal nitric oxide (nNOS) is constitutively expressed in specific neurons of the brain.

AIMS & OBJECTIVE
The aim of this study was to investigate eNOS and iNOS expression in acute AMR and compare interstitial fibrosis and tubular atrophy (IF/TA).

MATERIALS AND METHODS:
A retrospective study examined kidney biopsies obtained from 43 patients undergoing kidney transplantation between 2010 and 2015 who were diagnosed with C4d positive acute AMR. These biopsies were stained immunohistochemically for eNOS and iNOS antibodies. Staining of antibodies was scored on a scale ranging from 0 to 3 and were compared with IF/TA.

RESULTS:
There were 30 male (70%) and 13 (30%) female recipients. Median age at transplantation was 35 years (range, 9-66 y). Thirty-five patients received organs from living donors and from 8 deceased donors. The mean time from transplant to acute AMR diagnosis was 198 days (range, 3-1738 d). Immunohistochemical evaluation revealed expressions of eNOS and iNOS in glomeruli, peritubular capillaries and vessels. However, there was not a statistically significant correlation between IF/TA and the expressions of eNOS in glomeruli, peritubular capillaries and vessels (p = .592, p = .857, p = .644 respectively). The expressions of iNOS in glomeruli, peritubular capillaries and vessels were not correlated with IF/TA (p = .824, p = .371, p = .947 respectively).

CONCLUSIONS:
In the present study, the expression of eNOS and iNOS in glomeruli, peritubular capillaries and vessels was revealed. Nevertheless, protein expression of eNOS and iNOS may not be related to IF/TA in acute AMR.
Significance of microvascular inflammation in rejection episodes using Banff 2013 criteria

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Background:
Banff 2013 update fine tuned diagnostic criteria of both acute antibody mediated (ABMR) and cellular rejection (ACR). Main morphologic changes are definitions of glomerulitis, C4d negative ABMR, V lesions' in ABMR and transplant glomerulopathy based on electron microscopy features. We attempted validating these criteria in a retrospective cohort of ABMR to see how different the diagnosis would have been, obviously having impact on therapeutic options.

Aims and objective:
To evaluate indicated allograft biopsies using Banff 2013 criteria in DSA positive and negative patients and compare with previous diagnostic reports. Materials and methods: Indicated renal allograft biopsies (1 year) were re-evaluated according to 2013 Banff criteria.

Results:
Out of 24 cases, 9 cases were DSA positive; 7 C4d positive and 2 C4d negative. These C4d negative cases, with ptc 2 were not considered ABMR previously, thus denied benefit of treatment. There was no change in diagnosis of C4d positive ABMR. In DSA negative cases, 1 out of 12 C4d positive cases and 2 out of 5 C4d negative cases were labeled as suggestive of ABMR based on MVI ≥2 and these should have been searched for antibodies for other minor antigens. Total 3 cases of C4d negative ABMR would have been diagnosed. Electron microscopy of biopsies with 15 biopsies with ACR alone added evidence of early transplant glomerulopathy in 12, with 3 also showing ptc multilayering suggesting chronic ABMR. Only V lesions in 2 cases with ABMR were designated as having additional ACR, which were now could be categorized as ABMR only. Total inflammatory score was added in 10 cases.

Conclusion:
Using 2013 Banff criteria there was change in diagnostic categories; 4 cases of C4d negative ABMR, 2 with chronic ABMR, 12 with TG and 2 as V lesions of ABMR were diagnosed. Role of EM stands evident as additional information in 12/40 (30%). In MVI positive ABMR with HLA class I and class II negative DSA, search DSA for other minor antigens should be done.
Expression of miR-21 and miR-148b and its correlation with histopathological and biochemical variables in patients with IgA nephropathy

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Background:
IgA nephropathy is the most common form of glomerulonephritis worldwide. Around 20 to 40 % of IgAN patients develop ESRD. The pathophysiology of the disease is still partially known. miRNAs are short sequences of 20 to 22 nucleotides length which regulate gene expression by targeting mRNAs. In recent studies, different miRNAs have shown correlation with the severity of IgAN. miR-148b found regulating the C1GLT1 which explains the glycosylation process in IgAN. miR-21 was found associated with the renal fibrosis. We have chosen miR-21 and miR-148b for our study to see further scope towards establishment of a novel biomarker for IgA nephropathy. Objectives: A. Correlation of mir-21 and miR-148b levels with the clinicopathological data for IgAN. B. Correlation of miR-21 and miR-148b levels in IgAN patients with the healthy controls.

Material and methods:
Biopsy proven IgAN patients fulfilling inclusion and exclusion criteria were recruited after the written and informed consent taken from the patients. Plasma was immediately stored at -80 degree C after blood collection. miRNA-21 and miRNA-148b levels were quantified on probe based method through real time qPCR method. Spike in control, no template control, and known concentration of miRNA were used to check the amplification and making standard curves. Statistical analysis was performed through R language and graph pad. The miRNA results were compared with age matched controls who were apparently normal.

Result:
We found miR-21 and miR-148b levels significantly higher in disease group then the healthy control. We found the blood urea level inversely correlated with the miR-148b level and directly with the miR-21. The increased level of blood urea was also significantly correlated with the S and T values of Oxford (MEST) scoring system.

Conclusion:
miRNA levels in few cases were seen many fold increased and correlated with HAAS classification stages. MEST classification also showed some positive result with the urea level in the blood. There was significant difference of miRNA level in IgAN patients in comparison with healthy controls. The findings need to be validated in a larger subset of patients to draw meaningful conclusion.
Array of pathological diagnoses in adult patients presenting with Nephrotic Syndrome with bland sediment; single centre experience in 2016

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Background

Varied pathological processes lead to the common clinical presentation of nephrotic syndrome. Management ranging from high end immunosuppressive agents to conservative management with antiproteinuric measures depend on the underlying pathology. Nephrotic syndrome is the leading indication for native kidney biopsy in our centre.

AIM

Our aim was to identify the pattern of pathologies operating in the current time in our population leading to nephrotic syndrome as glomerular diseases are more commonly encountered in Asia compared to western world. Having an objective assessment of common causes help in better management and quicker intervention where necessary.

Material & Method

We analysed the biopsies of patients presented with nephrotic syndrome with bland sediment in 2016, available up to the start of study. 100 biopsies were reviewed with clinical history via detailed reports and pathology slides were re reviewed when necessitated. Histology under light microscopy and immunofluorescence staining was available. Mean age of the sample was 36 years (SD-15.44) with 59 females and 41 males.

Result

The main pathological diagnoses were minimal change disease, idiopathic membranous nephropathy and lupus nephritis having percentages of 33%, 15% and 12% respectively. Interstitial involvement was seen in 69% with interstitial fibrosis noted in 16%. More than 25% glomeruli being sclerosed was noted in 17% of biopsies.

Conclusion

Commonest cause of nephrotic syndrome in adults in our study was minimal change disease. 16% and 17% showed chronic interstitial changes and significant chronic glomerular changes respectively, suggesting need for early approach to intervention.
Patterns Of Malignant Renal Tumors Presenting At A Tertiary Care Centre Over 5 Years; Our Experience

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Objective:
To study the histopathological spectrum of renal tumors in resected nephrectomy specimens at a tertiary care hospital and analyse the age distribution and various characteristics of renal tumors

Methods:
The study was carried out at Pathology Department, BPS Govt. Medical College, Khanpur Kalan and cases presented over a 5-year period from Jan 2012 to Dec 2016 were analysed retrospectively. All the patients with renal tumours managed surgically with tumour nephrectomies, were included. Patients' demographic and clinical data were obtained from clinical charts and the histopathological features of tumours were retrieved from biopsy reports.

Results:
Amongst the studied cases during the study period, majority of cases were attributed to renal cell carcinoma, while 3 cases of squamous cell carcinoma and 2 cases with Wilms tumour were also reported. The details of age and sex distribution, anatomical location of the tumor (upper or lower pole), presence of metastasis, subtypes of carcinoma, any associated risk factors such as history of smoking, family history of any malignancy, presence of staghorn calculus, etc. were also noted.

Conclusion:
The spectrum of adult renal tumours in this study was moderately in co-relation with that of previously reported literature. RCC was the commonest malignant tumor. However, variations were noted in relation of site and frequency of occurrence of some rare carcinomas as compared to published literature.
Utility of C4d in subclassification of Membranoproliferative Glomerulonephritis

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Background:

Subclassification of Membranoproliferative glomerulonephritis into immune complex mediated (ICMPGN) and complement mediated (CMPGN) provides etiological information to the nephrologist, directs investigation strategy as well as transplant outcomes. C4d has been reported to be useful in this regard, as it is a marker of Classical (CP) or Lectin (LP) pathway activation; and its absence is considered the hallmark of Alternative (AP) pathway activation.

Aims and Objectives:

To study the utility of C4d in subclassifying MPGN

Materials and methods:

Cases of MPGN diagnosed between 2011 and 2016 with complete panel of immunofluorescence and electron microscopy were included. They were classified into ICMPGN and CMPGN using standard criteria. Immunohistochemistry for C4d was performed and staining scored as diffuse/variable with intensity 0-3+. In cases where C4d staining was discordant with the immunoglobulin (Ig) deposition, paraffin IF using Proteinase K digestion was performed to unmask immunoglobulins.

Results:

Fifty one cases of MPGN were included. These were divided into ICMPGN -16 cases and CMPGN -35 cases including 22 Dense Deposit Disease and 13 C3 Glomerulonephritis. Only 7 of the 35 CMPGN had negative to trace staining for C4d (20%) consistent with AP activation. Five cases (14.28%) had 1+ C4d and 9 cases (25.71%) had 1-2+ staining, which was mostly concordant with similar amounts of deposits of Igs. In 14 cases (40%) staining of C4d ranged from 2 – 3+, with a variable staining pattern across glomeruli. In 5/14 cases there was concordant Ig deposition, suggesting overlying CP/LP pathway activation. Nine cases did not have concordant staining for Igs and paraffin IF performed on 3 of these cases did not unmask any immunoglobulins. One case of suspected C4 DDD was also identified. The C4d staining pattern in all cases of ICMPGN was diffuse, 2 – 3 + intensity associated with polyclonal deposition of IgG in 16 cases, IgM in 15 cases and IgA in 8 cases. In 3 cases C1q was negative to traces, suggesting involvement of the Lectin Pathway.

Conclusion:

Insignificant C4d deposition was noted in only 20% of CMPGN consistent with AP activation. In the rest variable staining intensities ranging from 1+ to 3+ was noted suggesting overlying CP/LP activation. A proportion of cases did not show concordant Ig deposition even after enzymatic digestion to unmask Igs, possibly representing remote CP/LP activation. Thus, the absence of C4d staining in cases of MPGN is useful in confirming the diagnosis of a CMPGN but the presence of C4d does not negate the diagnosis, as variable secondary CP/LP activation is common. Observing the ultrastructural features is imperative and testing for complement pathway abnormalities would aid in the correct diagnosis.
Prevalence of glomerular anti-PLA2R staining in Indian patients of Membranous Nephropathy


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Background:
M-type Phospholipase A2 receptor (PLA2R) has been identified as the primary target auto antigen in idiopathic membranous nephropathy (iMN). Previous studies have shown a higher sensitivity for detection of anti-PLA2R in tissue sections compared to serum. There is a paucity of data on prevalence of anti-PLA2R in Indian patients of MN

Aims and Objectives:
To study the prevalence of anti-PLA2R positive MN in our adult and pediatric (< 18 years) population and correlate with immunofluorescence (IF) profile and clinical features

Materials and methods:
All cases of MN diagnosed between 2014 and 2016 were included. Staining for glomerular anti-PLA2R antibody was performed by direct immunofluorescence on enzyme treated paraffin embedded sections. Correlation with the IF profile and clinical parameters (Viral markers, ANA/dsDNA, investigations to identify underlying malignancy including faecal occult blood, imaging, haematological evaluation) was performed. Cases were divided into idiopathic and secondary MN based on clinical criteria

Results:
The study population included a total of 91 patients ranging from 12 to 65 years (80 adults and 11 pediatric) with a histopathological diagnosis of MN. Of 62 adults with iMN, 31 were positive for anti-PLA2R (50%) and of 8 pediatric patients, 5 were positive (62.5%). Of 18 adult patients with identifiable secondary causes for membranous nephropathy, 4 (22%) showed staining for anti-PLA2R and all these patients were cases of membranous lupus nephritis. None of the patients with seropositive HBsAg infection showed positive staining. Three patients of pediatric secondary MN (membranous lupus nephritis) were negative. The immunofluorescence profile (presence of IgA and C1q staining) did not reliably differentiate idiopathic versus secondary MN

Conclusion:
The prevalence of anti-PLA2R staining in our adult cohort is lower than reported from other Indian centres and Western data; however we found a higher percentage of anti-PLA2R positive pediatric iMN. Similar to previous literature, we also report presence of anti-PLA2R antibodies in a significant number of cases of Membranous lupus nephritis
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CALCIUM OXALATE DEPOSITION IN RENAL TRANSPLANT BIOPSIES: A SERIES OF 11 CASES
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Introduction:
Calcium oxalate deposition in renal transplant is a potential risk factor for graft dysfunction and its significance is usually undermined. Patients with end stage renal disease are uncommonly biopsied before renal replacement hence native renal disease is not always known. Primary hyperoxaluria may remain undiagnosed and undergo renal transplant often presenting with early severe graft dysfunction and sometimes graft loss.

Aims and objectives:
To screen and analyse the transplant cases which have severe oxalate deposition as primary cause of renal dysfunction.

Material and Methods:
We retrospectively evaluated all transplant biopsies from 2011 to 2016 (n=1766) and 111 cases which showed massive oxalate deposition were included in analysis.

Results:
Mean age at the time of biopsy was 33.5 years, range being 14-53 years. Male to female ratio was 2.66:1. Time of biopsy post-transplant was as early as 4 days to 14 years. 9 cases (81.8%) presented within first 3 months. 2 patients (18.1%) had prior history of recurrent calculus disease, one patient had IgA nephropathy as primary renal disease. One had definite negative history of calculus disease and cause of native renal disease was unknown in 8 patients. All the patients presented with rise in creatinine, value ranging from 1.8 to 4.8 mg. Tacrolimus levels were available in 3 patients, were higher than normal in 2 and low in one patient. One patient had chronic diarrhoea and mycofenolate induced diabetes at the time of biopsy. All patients had moderate to severe acute tubular injury, and widespread colourless oxalate crystal deposition predominantly in cytoplasm of proximal tubular cells and in lumen. All the biopsies were polarised and showed multi-coloured birefringence. Tubulo-interstitial chronicity was mild in all the cases (Banff grade I). 2 patients (18.1%) also had evidence of focal thrombotic microangiopathy (TMA). On follow up, urinary oxalate levels were high in 3 patients post-transplant, one of these is maintained on dialysis.

Conclusion:
Significant oxalate crystal deposition in allograft biopsies is a serious condition. The etiologies are varied and early and accurate identification is of utmost importance for eventual graft outcome.
THROMBOTIC MICROANGIOPATHY (TMA) IN DIABETIC NEPHROPATHY: AN UNDERRECOGNIZED PHENOMENON?

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INTRODUCTION
Diabetic nephropathy is a leading cause of chronic renal disease worldwide and is characterized by microvascular injury and endothelial dysfunction. Thrombotic microangiopathy (TMA) is associated with a wide group of disorders and has been extensively studied in patients with hemolytic uremic syndrome and hypertension. Studies analyzing prevalence and significance of TMA in patients with diabetes mellitus/diabetic nephropathy.

AIMS & OBJECTIVES
The current study aims to evaluate the incidence and significance of TMA in patients with diabetic nephropathy and to study their clinical profile

MATERIALS & METHODS
In this retrospective study, renal biopsies of patients with diabetes mellitus received between the period of January 2010 and December 2016 were reviewed. Their clinical profile assessed and biopsies were morphologically assessed for the presence of TMA

RESULTS
A total of 681 cases of diabetic nephropathy were evaluated during this period. Of these, 50 patients (7.3%) were found to have TMA in addition to changes of diabetic nephropathy. The mean age of the patients was 58 years (age range 30-75 years) and the male: female ratio was 39:11. Forty seven (94%) of these patients presented clinically with a significantly raised or rapidly rising serum creatinine level. An associated significant proteinuria was found in 25 (50%) patients. Almost 50% (n = 24) patients had hypertension (HTN) in addition to diabetes, of which 2 had malignant HTN. Histologically, 47 (94%) patients showed active TMA lesions including arteriolar and/or arterial necrosis with luminal thrombotic occlusion and RBC fragmentation. The remaining three revealed chronic features of TMA i.e. thrombosis and recanalization. Associated ischemic glomerular changes including mesangiolysis, fluffy appearing mesangium and mesangial fragmented RBCs were note in 5 patients. Most of the patients had class IV (74%) or Class III (24%) diabetic nephropathy, only one patient had changes consistent with Class IIa. Tubulointerstitial chronicity was significant (>50%) in 60% patients, moderate in 28% and mild in 14%.

CONCLUSION
The above clinic-pathological profile suggests that patients with diabetes presenting with a raised or rapidly rising serum creatinine, renal biopsy should be performed and assessed for presence of TMA. This would help in appropriate management and better renal outcome.
The Spectrum Of Graft Nephrectomies: AllMS Experience
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Introduction:
Graft failure remains an important clinical problem with varying etiologies. Depending on institutional protocols the percentage of transplants ending in nephrectomies varies from 0.5 to 43.5%. Most institute follows protocol of removing all early and late failure as needed.

Aims and objective:
To evaluate the histopathological spectrum of graft nephrectomies at our centre.

Material Method:
Twenty seven (27) cases of graft nephrectomies over a period of six years (2010-16) were retrieved from archives. The clinical data from the histopathology report forms and case files of the patients were collected. Histopathology was reviewed and the causes were grouped into vascular compromise, rejection and infection.

Result:
The age of the patients ranged from 15 years to 60 years and male to female ratio was 4.4:1. The most common causes for graft nephrectomy was ischemia related (62.96%) graft vessel thrombosis is commonest of all, followed by rejection (25.93%), infections like mucormycosis(7.41%) and one case of Post-transplant lymphoproliferative disorder (PTLD)(3.70%).

Conclusion:
In the current series graft vessel thrombosis is the commonest cause of graft dysfunction and nephrectomies in early Post transplant period.
CASE REPORTS
TUBERCULOSIS AS A MICROBIOLOGICALLY PROVEN ETIOLOGY OF MEMBRANOUS NEPHROPATHY

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Background:
Secondary causes of membranous glomerulonephritis (GN) include SLE, other autoimmune diseases, neoplasms and infections like Hepatitis B and C viruses, Syphilis and parasites. The association of Tuberculosis with membranous GN is rare. We report the first case of microbiologically proven tubercular interstitial nephritis and membranous nephropathy occurring concurrently in the same patient.

Case presentation:
A thirty-two-year-old Indian male presented with generalized edema for six months. Urine revealed proteinuria of 9.0 grams/day and 5-8 red blood cells per high power field. A renal biopsy revealed ten glomeruli with diffusely thickened capillary walls. The interstitium showed necrotizing granulomas. Immunofluorescence showed coarse granular deposits of IgG and C3 along the glomerular basement membrane. Ziehl Neelson staining revealed numerous acid fast tubercle bacilli in the interstitium. Computed tomogram of the abdomen, performed post-biopsy showed a hypodense mass lesion in the middle third of the left kidney.

Results:
A diagnosis of membranous nephropathy and interstitial nephritis with renal tuberculosis was made. Chest X-Ray did not reveal any lesions suggestive of active pulmonary tuberculosis. The patient was treated with anti-tubercular therapy. On follow up visit one month later, significant improvement in proteinuria and resolution of hematuria was noted.

Conclusions:
Granulomatous interstitial nephritis in association with Tuberculosis is a rare entity. It was identified in only 6 of 2798 renal biopsies in a series from India, despite Tuberculosis being endemic in the Indian subcontinent. The occurrence of GN in association with Tuberculosis is rarer still. Yalcin Solak et al in a review of the English literature identified 15 cases of GN associated with Tuberculosis, which included only a single case of membranous GN. In the present case, the identification of Acid fast bacilli in the biopsy lends credence to the tubercular etiology of both the interstitial nephritis and membranous lesions. The etiologic link is further strengthened by the amelioration of symptoms and renal status with treatment by anti-tubercular drugs alone. Recognition of tubercular etiology and institution of anti-tubercular therapy will greatly influence the prognosis of such cases.
Light chain deposition Nephropathy with Renal Amyloidosis
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Background.
Light chain deposition disease (LCDD) and light chain amyloidosis are systemic diseases caused by abnormal production of monoclonal immunoglobulin light chain and deposition of systemic tissue. Both diseases are usually secondary manifestations of lymphocytic proliferative disease, such as multiple myeloma, lymphoma. Kidney is the most common involved organ, often leading to progressive renal failure. A case of light chain deposition nephropathy with renal amyloidosis is discussed in this paper.

Case presentation.
A 57-year-old Chinese male, intermittent edema of double lower limbs associated with proteinuria for more than 3 years. Urine volume is about 400ml/day. Diuretic therapy is not effective. He denied rash and joints pain, without history of hypertension and diabetes.

Results.
Blood test showed WBC count 7.79×10^9/L, platelets count 215×10^9/L and hemoglobin 85g/L. Urinary protein was 3.82g/24h. Other indices of biochemistry showed BUN 19.24mmol/L (3.1-8.0mmol/L), creatinine 103umol/L (57-97umol/L), albumin 17.7g/L (40-55g/L). Serum protein electrophoresis showed as globulin 6.4%(11.1-18.8%), M protein negative, light chain 0.66g/L (1.7-3.7g/L) and light chain 1.47g/L (0.9-2.1g/L). Detection of urine Bence Jones protein showed free light chain was positive. ANCA, autoantibodies including PLA2R antibody and hepatitis virus were negative. Renal biopsy: The specimen for light microscopy contained 7 glomeruli, 2 of which were globally sclerotic. There were pale pink and unstructured material deposits in glomerular mesangial area and capillary wall by PAS staining. Renal tubular epithelial cells had vacuoles and granular degeneration. Tubular lumina contained protein casts, and the interstitium was infiltrated with lymphocytes and monocytes. The walls of small arteries were thickened, on which pale pink and unstructured material deposits. Immuno fluorescence staining showed IgA and IgG were strongly positive, while IgM was weakly positive. Immunohistochemical staining showed and light chain were also positive in local area. Besides, Congo red and oxidized Congo red staining were positive. Electron microscopy demonstrated that disordered amyloid fibrils were labeled in GBM and mesangium. The fiber diameter was from 7.7nm to 13.61nm. There were electron-dense granules deposits, ribbon like, on the inner side of the basement membrane.

Conclusions.
By light microscopy, immunofluorescence and electron microscopy, this case was conformed light chain deposition nephropathy with renal amyloidosis. It was detected in a specimen of kidney tissue that electron-dense granules deposited on the inner side of glomerular basement membrane associated with amyloid deposits. These two diseases occurred in the same individual was rare. The specific pathogenesis was unclear, yet to be further explored in the future.

Keywords:
Light chain deposition, renal amyloidosis, monoclonal immunoglobulin
Introduction:
Japan Cryoglobulinemic vasculitis (CV) and anti-neutrophil cytoplasmic antibody (ANCA) associated vasculitis (AAV) both belong to systemic vasculitis affecting capillaries, small-sized and medium-sized vessels, but rarely complicated each other. We report a case of systemic vasculitis caused by combined CV and AAV.

Case Report:
A 83 year old Japanese woman admitted to our hospital due to rapidly progressive renal failure and gastrointestinal hemorrhage. She presented an elevated serum creatinine (7.53 mg/dL), a high titer of MPO-ANCA (208IU/ml), cryoglobulinemia (type III) and a decreased serum complement level (CH50<10U/ml). During hospitalization, purpura developed on her lumbar skin and its biopsy showed necrotizing vasculitis with IgM and MPO deposition. In spite of intensive treatment including double filtration plasmapheresis and high-dose of steroid, she did not improve and died with aspergillus pneumonia. Autopsy revealed necrotizing crescentic glomerulitis in majority of glomeruli with partially membranoproliferative glomerular changes. The interlobular arteries showed necrotizing and sclerosing arteritis. Immunofluorescence revealed glomerular IgG, C3 and MPO staining and electron microscopy disclosed fibrillary deposits in the glomerular sclerotic lesion. The gastroduodenal ulcers were also associated with necrotizing vasculitis with IgM and MPO deposition. AAV is usually characterized by pauci immune vasculitis but sometimes accompanied by MPO and immunoglobulin deposition. On the other hand, CV is characterized by IgM and/or IgG deposition with organized deposits in electron microscopy.

Conclusions:
The present case had both characteristics in different levels of vasculature in various organs and we diagnosed this case with systemic vasculitis caused by combination of CV and AAV. A possible relationship between CV and AAV in the pathogenesis of vasculitis is discussed with a review of the previous literatures.
Acute pyelonephritis as an uncommon cause of acute kidney injury. A report of 7 cases in a 6 month period.

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Background:
Acute kidney injury is a rare complication of acute pyelonephritis in patients who do not have urinary obstruction or diabetes. Although urinary tract infection is common in adults, pyelonephritis is rarely considered in the differential diagnosis of acute renal failure.

Materials & Methods:
We reviewed renal biopsy records from the period of May 2016 to October 2016, comprising of 1171 native kidney biopsies. Out of these, 7 cases were found with a diagnosis of acute pyelonephritis. The laboratory and the pathological findings in all of the above 7 cases were reviewed and the findings on follow up were collected. We report on the details of the above biopsies of acute kidney injury were the pathologic finding were that of acute pyelonephritis.

Results:
Of the 7 cases analysed 4 were males and 3 females. All the patients were adults (age group of 26-70 years). Patients presented with advanced renal failure (s. creatinine from 4mg/dl -7.7 mg/dl) and 2 required hemodialytic support. Proteinuria was variable, ranging from subnephrotic to nephrotic range. Some had active urinary sediment.

One of the patients presented in postpartum period. None of the patients was diabetic. Only 2 had fever. Urine culture grew E. coli in 2 cases. Histopathology in all the cases showed a dense infiltrate of neutrophils, lymphocytes, a few plasma cells and occasional eosinophils within the interstitium along with neutrophilic casts. There was background chronicity in 2 cases.

3 patients had complete recovery, 2 with chronicity had a partial recovery and 2 were lost to follow up.

Conclusion:
Acute pyelonephritis is an uncommon but important cause of AKI and can occur in an otherwise normal individual without comorbidities as mentioned in the literature. This diagnosis may be missed if there is no biopsy, as the typical clinical findings are absent in these patients.
G6PD Deficiency Is Not An Uncommon Cause Of Pigment Nephropathy

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Introduction - Combination of acute kidney injury (AKI), jaundice and hemolysis is a common presentation of thrombotic microangiopathy (TMA), however tropical infections like malaria, dengue, leptospira and drugs like antimalarial can have similar presentation. However hemolysis leading to pigment nephropathy needs to be worked up in such situations. The usual practice of using antimalarials in above situation without proper investigation leads to diagnostic dilemma. Patients with relatively uncommon genetic causes of hemolytic anemia like glucose 6-phosphate deficiency (G6PD) may present with AKI, jaundice and hemolysis after receiving antimalarials. We report three patients of G6PD deficiency who presented with TMA like clinical picture but histologically as pigment nephropathy.

Patients and methods: There were three patients, all males, aged 20 years, 40 years and 41 years who presented with fever, jaundice, decreased urine output, cola colored urine and advanced renal failure giving a clinical impression of TMA. All three patients received antimalarial (quinine) therapy in outside hospitals for fever. First patient had another episode of similar illness 3 years back. Hemogram showed low hemoglobin in all 3 patients with a microangiopathic hemolytic blood picture and increased reticulocyte count. Acute events of all three patients were managed by multiple sessions of hemodialysis and conservative support. After stabilization of hemogram kidney biopsy was performed.

Histopathology - Renal biopsies showed normal glomerular morphology. There was no evidence of intra-glomerular or extra-capillary hypercellularity. Tubules showed features of acute tubular necrosis with red blood cell casts and pigments in all the cases. Interstitium was edematous with mild mononuclear cell infiltrate and the blood vessels were largely unremarkable. A diagnosis of acute tubular injury with pigment nephropathy was suggested. Detailed hemolytic work-up was done. All three patients came out to be positive for G6PD deficiency.

Conclusion - Glucose 6-phosphate dehydrogenase deficiency is a relatively uncommon disease presenting as pigment nephropathy and TMA like picture. In tropical countries G6PD deficiency although not common but is not a very rare disease and in these patients while prescribing antimalarial drugs, it is important to do detailed hemolytic work-up which is an important cause of preventable recurrent AKI in tropical countries.
Acute crescent glomerulonephritis in a patient with IgG4-related kidney disease

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Introduction:
IgG4-related disease (IgG4-RD) is a fibroinflammatory disorder that may involve almost each organ or system. IgG4-related kidney disease (IgG4-RKD) is referred to renal lesions associated with IgG4-RD. The most frequent morphological type of renal lesions is IgG4-related tubulointerstitial nephritis (IgG4-TIN) in relation to increased IgG4 positive plasma cells infiltration and interstitial fibrosis.

Case report: Herein, we present a rare case with coexisting of IgG4-RKD and acute crescent glomerulonephritis with concomitant severe tubular interstitial lesions instead of classic IgG4-TIN. Conclusion IgG4-RKD and acute crescent glomerulonephritis can occur in the same patient. This case may give us a more clear viewpoint of it.
Clinicopathological features of idiopathic membranous nephropathy combined with IgA nephropathy: a retrospective analysis of 9 cases
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Background:
The concomitant presence of idiopathic membranous nephropathy (MN) and IgA nephropathy (IgAN) is rare. The few previous reports available that is was rely heavily on immunofluorescence and electron microscopic examination to identify patients with combined idiopathic MN and IgAN. However, it may bring some confusion in diagnosing these complications by immunofluorescence and electron microscopic examination, because some secondary MN also showed mesangial EDD in additional to subepithelial deposition, while the IgAN sometime have minimal subepithelial EDD with mesangial massive deposition. Recently, it has been demonstrated that M-type PLA2R is a specific marker for idiopathic MN and is hardly detected in secondary MN and other glomerular diseases. Here, we report 9 cases of phospholipase-A2-receptor (PLA2R) positive idiopathic membranous nephritis combined with IgAN, while reviewing publications regarding the pathological characteristics of this glomerolonephritis complication.

Case presentation:
9 cases of renal biopsy tissues were retrospectively reviewed, including the clinicopathological features, the results of the immunofluorescence assays, and the electron microscopic examination. The patients mainly presented proteinuria and microscopic hematuria, and the serum anti-PLA2R was detected as positive in all of the patients. Histologically, a wide thickening of the glomerular basement membrane was observed in each of the 9 cases. Additionally, there existed mild hyperplasia in the mesangial cell and the matrix of the mesangial area. Immunofluorescence assays showed prominent glomerular granular staining on the glomerular capillary loops for IgG (++/+++), IgG4 (++/+ + + +), and PLA2R (+/+ + ). In addition, moderate IgA positive stains were focally or sparsely limited to the mesangial areas. Electron microscopy revealed subepithelial and mesangial electron-dense deposits.

Conclusions:
The results from the case analyses indicated that idiopathic MN combined with IgAN possess the clinicopathological features found in both components. It is suggested that serum anti-PLA2R and tissue PLA2R are important biomarkers that can assist in the diagnosis of idiopathic MN associated with IgAN.
COEXISTENCE OF MULTIPLE MYELOMA WITH RENAL CELL CARCINOMA.

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Background
We present a rare case of a patient with renal cell carcinoma and coexisting multiple myeloma.

Case presentation
55yr old female, a known case of hypertension presented on May 2016 with azotemia and proteinuria. Patient had a past history of radical nephrectomy done elsewhere which was diagnosed as renal cell carcinoma in July 2016. Due to persistent azotemia even after treatment with hemodialysis, renal biopsy was done.

Results
The renal biopsy revealed findings of cast nephropathy with amyloid positive casts. The immunofluorescence findings were negative. Bone marrow aspirate revealed Plasma cell myeloma. Kappa lambda ratio was 0.008. Cytogenetic evaluation of the bone marrow aspirate revealed an abnormal female karyotype with deletion 6q21. Multiple myeloma panel by FISH revealed Monosomy13, loss of 14q32, negative for IGH@rearrangements and negative for deletion of 17p. On reevaluation of the nonneoplastic renal parenchyma of nephrectomy specimen findings suggestive of cast nephropathy were noted.

Conclusion
Our case shows the rare coexistence of renal cell carcinoma with multiple myeloma. It also shows the importance of evaluation of non neoplastic renal parenchyma in tumor nephrectomy specimens.
Light Heavy Chain Deposition Disease - A rare entity with an even more rare combination.

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Background:
MIDD are systemic disorders characterized by deposition of monoclonal immunoglobulins in many organs, but the kidneys are the most commonly involved. It is of three types depending on the composition of the deposits: Light chain deposition disease (LCDD), heavy chain deposition disease (HCDD) and light and heavy chain deposition disease (LHCDD). Of these three types, LCDD is the most common, comprising about 80% of the cases.

Case presentation: 59 yr old lady, k/c/o diabetes and hypertension since last 10 years presented with complaints of loss of appetite since last 6 months, cough with expectation since last 3 months and oliguria since last 1 month. Investigations: Baseline serum creatinine was 1.2mg/dl which increased to 9.3mg/dl in 5 months. Urinalysis: 3+ proteinuria, WBCs: 2-4/HPF, RBC: 5-6/hpf. Urine spot protein creatinine ratio:3.93, Serum albumin 3.3mg/dl. USG: Normal kidney with increased cortical echotexture.

Results:
Renal biopsy : 2/9 glomeruli were sclerosed and obsolescent. The remaining viable glomeruli were enlarged and showed diffuse thickening of glomerular basement membranes along with prominent PAS positive mesangial widening. Diffuse tubular atrophy was evident in 70 -80% of the cortex. Immunofluorescence showed 5 glomeruli with significant peripheral and mesangial, linear continuous to coarse granular deposits of IgA with Kappa light chain restriction. Deposits of IgA and Kappa light chain were also seen in tubular basement membranes. Bone marrow examination revealed plasma cells comprising 60% of marrow nucleated cells. Serum immunofixation done showed M band, elevated serum kappa light chain levels with kappa lambda ratio of 265.025.

Conclusion:
We present a case of LHCDD with a rare combination of IgA heavy chain and Kappa light chain. The diagnosis of LHCDD requires careful pathologic evaluation of the renal biopsies and a high index of suspicion. The diagnosis cannot be reached by light microscopy alone and immunofluorescence with the use of specific antisera is required. Ultrastructural findings may provide confirmatory evidence of light/heavy chain deposition in various renal compartments.
Cryocrystalglobulinemia manifested as fulminant purpura and acute kidney injury in a patient with multiple myeloma

Suxia Wang

Background:
Cryoglobulinemia secondary to multiple myeloma usually manifested as type I, which contained monoclonal immunoglobulins with a tendency to accumulate in the form of crystals or substructures, and occluded the microvascular lumens, resulting in hemolysis and thrombosis in multiple organs with a very high morbidity. The kidneys and skin were the most affected systems.

Case presentation:
A 57-year-old women was admitted to our hospital because of fever, fulminant purpura and acute kidney injury. She suffered from repeated but progressive ankle pain and frequently purpuric rash in the extremities in two years. Four days before admission, she froze in a trip, presented with chill and fever to 38.5°C, and the outbreak of purpura involving skin from feet, ankle to thigh, buttocks, face and the whole body. She had leukocytosis (WBC count: 14.68 x 10⁹/L), mild anemia (Hb 115-107 g/L) and thrombocytopenia (PLT count: 79-95 x 10⁹/L), but no schistocyte was detected. The urinary analysis showed microscopic hematuria (RBC: 10-17/HP) and urinary microalbumin( 328mg/L). Serum creatinine elevated rapidly from 37.1 to 155 μmol/L. Immunofixation electrophoresis showed a monoclonal IgG λ in both of serum and urine. The serum cryoglobulin was positive and contained IgG lambda components. Bone marrow biopsy showed a 20% of immature and mature plasma cells, FISH showed 2.0% restrictively expressed clonal lambda light chain, which consisted with a diagnosis of multiple myeloma. The skin biopsy revealed diffuse hyaline thrombi with crystals plugging the microvascular lumens of dermis.

Renal biopsy findings:
There were 23 glomeruli included in the specimen for light microscopy, diffusely swelling of endothelial cells with substantial intracapillary thrombi were identified in the majority of glomeruli. Some of them showed wrinkled GBM with segmental double contours. The arterioles showed endothelial swelling and detachment, with thrombi occluded the vascular lumens, surrounded by granulomatous infiltration of neutrophils and mononuclear cells. By electron microscopy, diffuse widening of subendothelial area of glomeruli and duplication of segmental GBM were seen, and the dense linear arrays of crystals were identified in some of intracapillary deposits and a few crystals sparsely in tubular lumens. Immunofluorescence microscopy revealed intravascular deposits of IgM(++), IgG(++) , C3(++), C1q(+), k(++) and λ(++) light chains in both of glomeruli and arterioles.

Final diagnosis:
cryocrystalglobulinemia associated kidney and skin injury, with a pattern of intravascular crystalline deposits and thrombosis consisted of type I cryoglobulins (IgG λ), and secondary acute TMA lesions, which was mostly caused by multiple myeloma.
Concurrent anti-glomerular basement membrane nephritis and IgA nephropathy: a case report and literature review

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Anti-glomerular basement membrane (GBM) disease is characterized by circulating anti-GBM antibodies and crescentic glomerulonephritis (GN) with deposition of immunoglobulin (Ig) G along the GBM. In a limited number of cases, glomerular immune complexes have been identified with anti-GBM disease. A 38-year-old female presented azotemia, hematuria, and proteinuria without pulmonary symptom. A renal biopsy showed crescentic GN with linear IgG deposition along the GBM with mesangial IgA deposition. The patient was diagnosed concurrent anti-GBM nephritis and IgA nephropathy. Therapies with pulse methylprednisolone and cyclophosphamide administration were effective. Concurrent anti-GBM nephritis and IgA nephropathy is rare among cases with anti-GBM disease with immune complexes. This rare case with concurrent anti-GBM nephritis and IgA nephropathy with review of literature is noteworthy.

Key words:
Anti-glomerular basement membrane, Immunoglobulin A, crescentic glomerulonephritis
Systemic lupus erythematosus-myositis overlap syndrome with lupus nephritis: An unusual presentation
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Background
The term overlap syndrome includes co-existence of signs, symptoms and immunological features of two or more connective diseases occurring simultaneously. Systemic lupus erythematosus-myositis overlap syndrome is rare and its frequency varies from 4-16% in different series.

Case report
We describe a case of lupus nephritis with myositis in a 28-years-old female. She presented with facial puffiness, haematuria and proteinuria. Her serum creatinine was raised with positive anti-nuclear antibodies. Kidney biopsy and immunofluorescence findings were those class V lupus nephritis. She also had proximal myopathy with a swelling in the right forearm, which on cytology was suggestive of myositis. Her serum creatine kinase was elevated. She was treated with immunosuppresants with resolution of her muscle weakness.

Conclusion
Renal involvement is seen in one-third of systemic lupus erythematosus patients and majority of them are diagnosed at the time of presentation. Systemic lupus erythematosus-myositis overlap syndrome is rare with prognostic implications. Reports suggest that systemic lupus erythematosus-myositis overlap syndrome with lupus nephritis has a poorer outcome.
Possible causes of Pauci-immune crescentic Glomerulonephritis in a Tuberculosis patient— A Case Report
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BACKGROUND
Pauci-immune crescentic Glomerulonephritis (PICGN) is a potentially life threatening disease, leading to renal failure within few weeks. Majority of these cases are associated with systemic vasculitis and test positive for ANCA. We present a case report of a male patient with c ANCA positive PICGN and history of Tuberculosis and Anti-tubercular treatment (ATT) and discuss the possible etiology of PICGN.

CASE REPORT
A 63 year old male presented with loss of appetite and weight, on and off fever, cough and expectoration for 3 months and drooping of right eyelid and sensorineural deafness for 4 days. There was history of Anti tubercular drugs intake for the past 3 months. Investigations showed anemia (Hb-8.8gm/dl), leucocytosis (TLC – 14800/dl) and his serum creatinine doubled within last two months (1.2 to 2.6). Urine examination showed 2 + albumin, full field of RBCs and 3-6 WBC/hpf. His serum was positive for c-ANCA. His sputum was AFB negative. Ultrasonography showed Normal kidneys. Chest X-Ray revealed old healed tubercular lesions. A renal Biopsy was done with a clinical diagnosis of rapidly progressive renal failure. Renal Biopsy showed Crescentic Glomerulonephritis on light microscopy, which was pauciimmune on Immunoflourescence and normal on Electron Microscopy. Crescentic Glomerulonephritis in this patient could be a result of

1. C-ANCA vasculitis
2. Tuberculosis with a renal LIMITED vasculitis – due to autoimmune reaction.
3. Anti tubercular drugs resulting in Crescentic Glomerulonephritis – due to autoimmune reaction.

The patient was started on immunosuppressive agents with close follow up for any sign of reactivation of tuberculosis.

CONCLUSION
Delineating the exact cause of PICGN can affect the patient follow up and/or treatment. Thorough history and all relevant investigations are indispensable.

KEYWORDS
Pauci-immune crescentic Glomerulonephritis, c ANCA, Tuberculosis, Anti tubercular treatment, vasculitits
Background:
Systemic sclerosis (SSc) is a chronic autoimmune disease characterized by vascular alterations and autoimmune activation leading to widespread organ fibrosis. SSc microangiopathy includes loss of small vessels and proliferative obliterative vasculopathy; however, vascular inflammation indicating association with systemic vasculitis is rare. It has been suggested that unusual vascular manifestations in SSc patients might be associated with systemic vasculitis, including cryoglobulinemic vasculitis.

Case presentation:
A 67-year-old man with diabetes mellitus type I and SSc presented with fatigue, abdominal pain, occasional diarrhea, anemia, purpura of arms and legs, and worsening renal function (serum creatinine from 170 to 270 umol/l in the last 3 months). Urine examinations showed only mild hematuria with scarce erythrocytic casts and low proteinuria, which tended to indicate tubulointerstitial disease. ANA was positive, ENA was negative and ACMA antibodies were present. ANCA was negative but mixed cryoglobulinemia 1857 mg/l (IgG and IgM) was noticed. C4 component of complement was lowered. Serology tests for hepatitis B and C were negative. Skin biopsy suggested immune complex vasculitis.

Results:
Renal biopsy revealed diffuse proliferative and exudative immune complex glomerulonephritis with double contour formation and diffuse necrotizing vasculitis of small interlobular arteries and arterioles, and acute tubular injury. Ultrastructurally, there were not only subendothelial and mesangial deposits consistent with cryoglobulinemic glomerulonephritis but also glomerular chronic thrombotic microangiopathy related to SSc. After treatment with plasmapheresis, rituximab and methylprednisolone pulses, renal function improved but proteinuria transiently increased into the nephrotic range and laboratory signs of thrombotic microangiopathy occurred, so the dosage of methylprednisolone was quickly diminished.

Seven months later, when renal function worsened again (serum creatinine 330 umol/l, cryoglobulins 250 mg/l), kidney re-biopsy revealed residual immune complex glomerulonephritis and chronic thrombotic microangiopathy without active vascular lesions, but marked interstitial fibrosis and tubular atrophy developed. Biopsy of small salivary glands confirmed secondary Sjögren’s syndrome.

Conclusions:
SSc patients with suspicious small vessel vasculitis should undergo appropriate clinico-serological investigation, including cryoglobulins detection.
In SSc patients, worsening of renal function might be associated with cryoglobulin-related vasculitis and glomerulonephritis, even in the absence of marked hematuria and proteinuria, which could be confirmed only by renal biopsy.
**Monoclonal Gammopathy of Renal Significance – guess the trigger for manifestation?**

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**Background**

The spectrum of plasma cell dyscrasias ranges from Monoclonal Gammopathy of Undetermined Significance (MGUS) to smoldering myeloma and frank multiple myeloma. Although MGUS cases do not have any end organ damage, but a proportion of cases can manifest with renal injury when it is called Monoclonal Gammopathy of Renal Significance (MGRS). Herein we describe a case of Acute hepatitis E infection which precipitated the development of MGRS.

**Case report**

A 39 year old lady had acute cholestatic hepatitis (positive IgM anti-HepatitisE-antibody) in January 2016 with normal RFT initially. Subsequently jaundice had gradually improved, but she started developing nausea and vomiting. On evaluation bilirubin and liver enzymes were normal, but serum creatinine was 5.5 mg/dl. Renal biopsy was done with provisional diagnosis of RPRF? Drug induced acute interstitial nephritis. On light microscopy, the needle biopsy included three normal glomeruli. There were PAS negative fractured casts in the tubules with giant cell reaction around them. The tubular epithelial cells showed cytoplasmic vacuolization and bile pigment. On DIF, the casts showed kappa restriction. A diagnosis of bilirubin proximal tubulopathy and light chain cast nephropathy was made and a possibility of myeloma was suggested.

**Results**

On SFLC assay, the $\kappa:\lambda$ ratio was 27 and $\beta_2$ microglobin was 8036 ng/ml. Bone marrow examination showed 5% plasma cells. On skeletal survey there were no bony lesions and serum calcium was 8.6mg/dl. Hematology consultation was taken and patient was started on cyclophosphamide / bortezomib / dexamethasone regimen for MGRS.

**Conclusion**

The present case is unique in two aspects. Firstly, the patient developed MGRS triggered by acute hepatitis E in less than a month. Secondly, the MGRS lesion manifested in the form of Light chain cast nephropathy. MGUS cases with renal injury must be treated with chemotherapy to reduce nephrotoxic light chain burden and preserve renal function.
Rare Dual Fungal Infection in Renal Transplant Recipient

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Case report –
A 41 year old male, renal transplant recipient was put on triple immunosuppression. He developed an episode of acute humoral rejection in immediate post-transplant period, which was managed with anti-thymocyte globulin. He presented at 11 months post-transplant with a lesion on extensor surface of right forearm bearing multiple discharging sinuses. It measured 5x5 cm, which gradually increased in size over last 5 months. It was excised with extensive debridement. Grossly skin was intact. Cut surface showed greyish white nodule with central blackish areas. Microscopically epidermis was unremarkable. Dermis showed a necrotic lesion containing multiple colonies of pigmented budding yeasts and hyphae, foreign body giant cells and mild mixed inflammatory infiltrates. Tissue culture showed pigmented, black mycelial fungi conforming to the morphology of pheohyphomycetes. Patient was managed with oral anti-fungals and close monitoring of cyclosporine levels. Ten days later, patient developed another lesion on right tibial region measuring 1.5x1 cm having whitish nodule on cut surface. Microscopically budding yeasts and hyphae of candida albicans were seen. Antifungals were continued for 3 months. Patient is stable with normal renal functions.

Discussion – Deep tissue fungal infections occur in 3.8% to 9% of renal transplant recipients. Candida infections are most common fungal infections in patients with heightened immunosuppression. A combination of rare dematiaceous fungi and candida presenting simultaneously as localized infection in post-transplant recipients is rarely reported. Pheohyphomycosis is a dematiaceous fungus characterized by mycelial and / or conidia form with brown-black pigment which presents as localized infection in late (after 2 years) post-transplant or as systemic infection in early post-transplant period. The unique finding in index case was that pheohyphomycosis presented as localized infection in early post-transplant period (within a year) despite heightened immunosuppression. Though it is regarded as non-pathogenic in clinical specimens, the clinical setting in which they are isolated becomes important before making decisions regarding therapy. Conclusion – The localized presentation of a combination of pheohyphomycosis and candida albicans infection in early post-transplant period despite heightened immunosuppression was unique finding. Early diagnosis in index case resulted in curative resection and prolonged patient survival.
CR 18
Antibody-mediated Rejection superimposed with the recurrent C3 Glomerulonephritis
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Abstract

C3 glomerulonephritis(C3GN) is a newly established glomerulonephritis(GN) and had been thought as a variation of membranoproliferative GN(MPGN). Recently, cases of recurrent C3GN in transplant kidney have been reported and revised for cases thought as recurrent MPGN. We experienced a case of superimposed active antibody mediated rejection on recurrent C3GN, which was reminiscent of postinfectious GN morphologically, i.e. mesangial proliferation with inflammatory cell infiltration, C3 deposition and the presence of humps in a glomerulus. A 38-year-old man with allograft dysfunction after kidney transplantation(KT) was admitted to our hospital. He was initially diagnosed with MPGN 20 yrs ago. He received kidney from his father 11 years ago and was diagnosed as recurrence of primary disease one year after KT. Allograft failure developed 2 years after KT. Ten years after, the second KT, B-cell crossmatch-positive KT(DQ7 DSA) was done. During follow-up, the first allograft biopsy(4th month) reveald microvascular inflammation(g3, ptc1, C4d0) with recurrence of MPGN pattern GN which was reclassified as C3 GN. He recieved steroid pulse, rituximab, plasmapheresis and immunoglobulin therapy. The second biopsy(10th month) showed glomerular lobular accentuation with cellular proliferation and microvascular inflammation. Same treatment was done. The third biopsy(11th month, demonstrated that BKVAN was complicated with the previous diagnoses. Glomerlar changes were far progressed and cellular crescents were developed. As azotemia did not improve after repeated therapy, one cycle of bortezomib (1.3 mg/m2 x 4 doses) was injected. Thereafter, allograft function stabilized and BK viremia turned to be undetectable after 6 months. The present case suggests that bortezomib therapy could be applicable to patients with refractory AMR even combined with BKVAN. The progression of recurrent C3 GN may contribute to this allograft dysfunction through the dysregulation of alternative pathway of complement system.
Collagenofibrotic glomerulopathy- A report of 2 cases
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Background:
Collagenofibrotic glomerulopathy (CG), is characterized by abnormal accumulation of type III collagen fibrils within the mesangial matrix and subendothelial space and a marked increase in serum type III procollagen peptide levels. The clinical manifestations include proteinuria, peripheral edema, hypertension and occasional progression to end stage renal disease.

Case 1:
51 yrs male, hypertensive and recently diagnosed diabetic presented with pedal edema which progressed to anasarca over last 3 months. BP is 180/90 mmHg. CBC was normal. ESR was 78. Urine showed 3+ albumin and occasional RBC’s. Urine protein creatinine ratio was 9.4 g/g. Total cholesterol was 173 mg/dL. Serum albumin and creatinine were within normal limits.

Case 2:
36 yrs female hypertensive (x 2 yrs), hypothyroid & nondiabetic - presented with nephrotic range proteinuria (4.7 gm/day). Urine showed 4+ albumin. Serum creatinine 0.76 mg%. HB -9.0 gm%. Complements are normal. ANCA is negative.

Results:
Light microscopy revealed 15 glomeruli (case1) and 16 glomeruli(case 2). All the viable glomeruli were enlarged with deposition of pale eosinophilic acellular material in the mesangium, which was weak PAS-positive and silver negative. Congo red stain was negative for birefringence. Trichrome stain reveals bright red band of deposits. Silver stain showed double contoured capillary basement membrane. Mild diffuse acute tubular injury was also seen. Hyalinosis in the small arterioles and tunica media hyperplasia was noted. Immunofluorescence: Case 1-Focal and segmental trapping of IgG (2+ to 3+), IgM (1+), C3 (3+), C1q (2+), Kappa (trace to 1+) & lambda (1+ to 2+) was seen in the area of sub-endothelial region. Case 2- Focal and segmental trapping of C3 (2+ to 3+) in the area of sub-endothelial region. EM revealed variably sized curved organoid structures with frequent periodicity in the subendothelial area and mesangium.

Conclusion:
CG may present as an isolated, sporadic form usually seen in adults, or as a familial form in children. The disease produces a slowly progressive decline in renal function often leading to end-stage disease. Electron microscopy and supporting light microscopic clinch the diagnosis. Immunohistochemical assays specific for type III collagen can be used to support the diagnosis.
CR 20
Multifocal Sarcomatoid Urothelial Carcinoma of the renal pelvicalyceal system and ureter – A diagnostic dilemma

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BACKGROUND:
Sarcomatoid urothelial carcinomas (SUC) are characterized by intimately mixed malignant epithelial and malignant mesenchymal components. The epithelial components may be squamous, glandular or high-grade transitional type, while the mesenchymal components may be chondrosarcoma, malignant fibrous histiocytoma, osteosarcoma, leiomyosarcoma, fibrosarcoma or rhabdomyosarcoma. SUC in the urinary tract occurs predominantly in the urinary bladder with the renal pelvis being an extremely rare site for SUC.

CASE PRESENTATION:
A 75 year-old male presented with swelling on the left side of the abdomen for 2 months. Clinical examination and Ultrasonography showed an enlarged left kidney with thinned out parenchyma with internal echoes. Two calculi were seen, one in pelvis (2.3 mm) and the second at the Vesico-Ureteric junction (6mm). The upper ureter was dilated and tortuous (Moderate Hydroureter). Contrast enhanced-CT confirmed the Ultrasound findings. Urine cytology done on 3 consecutive days was scantily cellular with no malignant cells identified in any of the samples. Per-operatively, the left kidney with left hydroureter with mass in the pelvi-ureteric junction along with the spleen and vascular stump was resected. Grossly, the left kidney was hydronephrotic, 18x14x13 cm in size, with dilated renal pelvis and calyces; peripheral atrophic renal parenchyma and obliterated renal pyramids. A single stone was identified in the renal pelvis. The entire pelvicalyceal system showed presence of multiple, grey-white, irregular, friable growths varying in size from 0.5x0.5x0.5cm to 4x4x3.5cm. The pelvi-ureteric junction and the entire length of the ureter (22 cm) also revealed similar growths filling the lumen with resected end of the ureter being grossly involved. The spleen was grossly unremarkable. No adrenal gland or lymph nodes were identified. The post-operative course was uneventful and the patient was discharged.

RESULT:
Histopathological examination was consistent with the diagnosis of a multifocal Sarcomatoid Urothelial carcinoma involving the renal pelvicalyceal system, the pelviureteric junction and the full length of the ureter. The epithelial component consisted of a high grade Transitional Cell carcinoma with squamous differentiation, positive for Pancytokeratin, CK5, CK7 (focal) and epithelial membrane antigen. The Sarcomatoid component showed smooth muscle differentiation and was positive for vimentin, smooth muscle actin and CD 10. Mitotic activity was 2-3/HPF. Foci of necrosis were present. Lymphovascular invasion was identified. The tumor was invading the wall of the pelvis, involving the renal sinus, the perinephric fat and the surgically resected end of the ureter. The rest of the kidney showed features of chronic pyelonephritis and benign nephrosclerosis.

CONCLUSIONS:
A multifocal SUC with involvement of Pelvicalyceal system is a rare event. IHC is essential to confirm the diagnosis and rule out other differentials including pure sarcoma, Sarcomatoid variant of Renal cell carcinoma and Pseudosarcomatousmesenchymal proliferation.
Background -
Calcium phosphate (CP) crystal deposition is frequently observed in kidney allografts. Hyperphosphatemia with or without hyperparathyroidism has been identified as risk factors especially in kidney allografts with delayed function. We describe a patient presenting with graft dysfunction with intra-tubular CP crystal deposition without any other abnormalities on renal biopsy.

Case presentation:-
A 49-year-old male underwent live donor kidney transplantation following end stage renal failure secondary to diabetes and hypertension. There were no immediate postoperative complications and serum creatinine improved from 434 μmol/L to 97 μmol/L over three days. His immunosuppressive regimen consisted of ATG as the induction agent and prednisolone, mycophenolate mofetil and tracrolimus as the maintenance agents. Six weeks after surgery, the patient presented with reduction in urine output and progressive lower limb swelling. Results The patient’s serum creatinine was 543 μmol/L and doppler studies showed a patent renal artery. Pulse methylprednisolone, plasma exchange and intravenous immunoglobulin were administered while pending renal biopsy. Tacrolimus dose adjusted according to the tacrolimus trough level. However the response was suboptimal and the patient was dialysis dependent. Allograft biopsy revealed 15 foci of intra-tubular non-polarizable CP deposits, which stained positive with von Kossa stain, associated with acute tubular injury. There was no evidence of rejection, tacrolimus toxicity or viral cytopathic changes. Immunostains for C4d, IgG, IgA and IgM were negative. Serum phosphate levels were only marginally elevated (4.6 mg/dL), and there was no biochemical evidence of hyperparathyroidism pre or post renal transplantation. Urine calcium excretion of the recipient and donor were normal. Following renal biopsy, he was started on potassium citrate to increase the clearance of CP. Patients' urine output and serum creatinine gradually improved (132 μmol/L) and became dialysis independent after one month of treatment. Conclusion In view of the response to therapy and the absence of any other abnormality to explain graft dysfunction; this case suggests that isolated intra-tubular CP crystals may have contributed to the impairment in graft function. Although the clinical relevance of isolated acute CP crystallization causing allograft dysfunction remains controversial, the implication of CP crystals in graft biopsies needs further study. Early detection and treatment would be important to prevent further deterioration in graft function.
Renal Cortical Necrosis; Five consecutive cases within short span of time

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Background:
Renal cortical necrosis is characterized by patchy or diffuse destruction of all the elements of renal cortex resulting from significantly diminished renal arterial perfusion due to vascular spasm and microvascular injury. It is a rare cause of AKI in developed countries with frequency of 1.9-2% of all patients of AKI. In contrast incidence of RCN is higher in developing countries ranging from 6-7%. Obstetric complication is the main cause of RCN in developing countries, earlier it was about 20-30% which has been declining to 5% in Indian subcontinent during past two decades.

Aim:
The aim of this study is to review five consecutive cases of RCN diagnosed within very short span of time.

Cases:
Histopathologically diagnosed five cases of RCN during one month span in September 2016 at Armed Forces Institute of Pathology, Dhaka were included in this study. All the cases were referred cases from a tertiary level obstetric center of Dhaka city.

Result: Among histologically proved five cases of RCN the mean age was 24.2 + 3.4 year. All the cases were female, had the history of post partum haemorrhage followed by septicemia. All they presented with acute renal failure dependent on haemodialysis for more than 21 days. On histological examination 02 (40%) show coagulative necrosis of all the glomeruli, 02 (40%) show coagulative necrosis of > 50% of glomeruli and in one (20%) case necrosis of about 25% of glomeruli. One of the glomeruli shows global sclerotic change of most of the glomeruli. Diffuse coagulative necrosis of renal tubules is seen in 02 (40%) cases and 03 (60%) cases show focal coagulative necrosis. In all the cases interstitium shows moderate focal lymphocytic infiltration and mild oedema. Among all 01 (20%) was found with IgA nephropathy as an associated diagnosis.

Conclusion: Renal cortical necrosis is still commonly encountered as an obstetric complication in our setting which can be easily diagnosed by renal biopsy. This type of grave consequences should be prevented by better monitoring of pregnancies.
Membranous glomerulopathy with organised (tubular) subepithelial deposits in a hepatitis B positive patient
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Background:
Membranous glomerulopathy is characterized by the subepithelial immune complex deposits. These deposits are unorganized, granular or confluent (patternless) but rarely may be organised like spherules. Pathogenesis of these organised deposits is obscure. We have identified a case of hepatitis B with nephrotic syndrome having membranous glomerulopathy with subepithelial tubular deposits.

Case presentation:
A 45 years old female with hepatitis B positive, on tab enticavir 0.5 mg for 6 months, was admitted to our side for evaluation of anasarca and 4+ proteinuria. On admission her BP was 100/70 mm Hg. General examination was unremarkable except anasarca. On abdominal examination she had ascites with no palpable organomegaly. All other systems were normal. Her investigations revealed- Hb-10.1 gm/dl, CBC-WNL, S. Protein-3.9 gm/dl, S. Albumin-1.9 gm/dl, LFT-WNL, B. Urea-27mg/dl, S. Creatinine-1.1mg/dl, Urine- protein-4+, RBCs-nil, Pus cells-4-6/hpf, 24hrs urinary protein-4.9 gms, Triglyceride-241mg/dl, T. Cholesterol-215 mg/dl, HBsAg-positive, anti HCV-negative, HBV DNA- 2.6 lacks copies/ml, ANA negative, S. C3,C4- normal, USG abdomen- mild hepatomegaly, both kidneys are normal in size and echotexture with maintained CMD. Bone marrow biopsy- normal.

Results:
In renal biopsy, on light microscopy glomeruli reveal diffuse mild thickening of capillaries with patchy intramembranous mottling. There is no evidence of tuft necrosis, subendothelial deposits, endocapillary cellularity, intracapillary thrombi or crescent formation. DIF studies reveal confluent staining for IgG without significant staining for light chains. EM study reveals many subepithelial organized deposits showing nearly equispaced arrays of focally hollow appearing tubular structures measuring 59-80nm in cross sectional diameter. Mesangial interposition/splitting of GBM is not observed. Visceral epithelial cell foot processes show significant effacement. Mesangial areas do not show increased cellularity or electron dense or organized deposits.

Conclusions:
This case is rare but distinct histopathological variant of membranous nephropathy with organised subepithelial deposits, pathogenesis of which remains elusive.
Hypothyroidism with IgA Nephropathy: A Rare Association
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Background:
Varieties of renal dysfunction may coexist with thyroid disorders. Association of hypothyroidism with different glomerular diseases although well documented is rare. We hereby report a case of hypothyroid with biopsy proven IgA nephropathy that was treated successfully with medical management.

Case report:
A 22- year-lady presented with the complaints of generalised swelling for one month followed by easy fatigability, weakness and palpitation. On examination she had mild periorbital swelling with peripheral pitting oedema with pulse rate of 62/minute and blood pressure of 160/100 mmHg. Systemic examination was normal. Blood tests revealed Hb 11gm/dl, WBC 7400/mm3, ESR 38 mm at first hour. Her blood glucose , urea , creatinine , liver function tests and serum electrolytes were within normal limits. A urine analysis showed specific gravity of 1.012, pH 6.5, protein 4 +, Nil RBCs, WBCs 2-3/high power field and 24 hours urine protein was 3341.6 mg. Fasting lipid profile showed mixed lipidemia with total cholesterol 227 mg/dl, triglyceride 216 mg/dl, HDL 42 mg/dl and LDL 146 mg/dl. Her viral serology for Hepatitis B , C & HIV and anti-nuclear antibody (ANA) tests were negative and ASO titre and coagulation profile were normal. Thyroid function test showed free T3 of 2.69 (2.5-3.9 pg/ml), free T4 1.04 (0.61-1.12 ng/ml) and TSH 11.89 µIU/ml. Renal biopsy showed features of diffuse mesangioproliferative glomerulonephritis with 3/10 sclerosed glomeruli ,interstitial fibrosis and inflammation with atrophied tubules and medial hypertrophy of the arterioles. Immunofluorescence microscopy showed IgA (3 +), IgM (1 +), IgG (negative), C3 (negative), Kappa (1 +) and Lambda (2 +) suggestive of IgA nephropathy class V with advanced chronic glomerulonephritis. In view of the presence of associated hypothyroidism , the patient was started with prednisolone, thyroxine, diuretics, ACE-I and atorvastatin. On follow up visits she had clinical response with decrease in oedema and normalisation of blood pressure. Her 24 hours urinary protein excretion was reduced to 713.1 mg and TSH to 0.48 at the end of 6 months.

Conclusion:
In conclusion, hypothyroidism may be associated with different types of nephrotic syndrome. Coexistence of the two pathologic conditions could be explained by a common autoimmune pathogenesis.
Lupus Nephritis after Liver Transplantation

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Background:
Liver transplantation (LT) is definitive approach for the treatment of end-stage liver disease. Renal dysfunction in LT recipients is not uncommon. Calcineurin inhibitors (CNI) induced nephrotoxicity, diabetes and/or hypertensive nephropathy are major clinical conditions associated with kidney dysfunction after LT. Glomerulonephritis (GN) is also reported in LT recipients. Lupus nephritis is a rare complication after LT.

Case Presentation:
We present the clinical manifestations of LN in a 18-year-old girl who received a cadaveric liver transplant. The cause of end-stage liver disease was Alagille syndrome. Her drugs included tacrolimus and ursofalk. After 13 years of LT, she developed nephrotic syndrome without other symptoms of systemic lupus erythematosus. The amount of proteinuria was 20g/24 hours. Antinuclear antibody was positive, but anti-dsDNA and anti-Smith were negative. The serum complements were decreased. Pulse methylprednisolone was given and the dosages of all immunosuppressive drugs were set. Her edema and hypoalbuminemia worsened. Rituximab could not be administered due to allergic reactions. She then developed pneumonia and died owing to multi-organ failure.

Results:
Diffuse endocapillary and mesangial proliferative GN was detected in the renal biopsy. There was mild interstitial fibrosis and tubular atrophy. Mild interstitial inflammation was seen. Arteriolar hyalinosis was also seen considering CNI-induced nephrotoxicity. A full-house pattern was documented under immunofluorescent examination. Electron microscopy revealed subendothelial, subepithelial and mesangial electron-dense deposits. The tubular basement membrane showed irregular thickening, splitting and accumulation of electron-dense particles. These findings were evaluated as compatible with LN WHO class IV-G (A/C) and electron microscopic changes of Alagille Syndrome.

Conclusions:
The etiology of renal dysfunction in LT recipients is multifactorial and many individuals have more than one lesion on biopsy as presented in the current case. CNI-induced nephrotoxicity and glomerulonephritis should be considered in LT recipients have a decreased kidney function. Glomerulonephritis may have resulted from the progression of pre-existing GN or new development of after LT. It was thought that Alagille syndrome might have prepared the ground for immune complex glomerulonephritis. Heavy proteinuria, hypoalbuminaemia and severity of renal impairment in LT recipients are the most important clinical variables that prompted a decision to perform a kidney biopsy.
Membranoproliferative Glomerulonephritis associated with small vessel vasculitis: A diagnostic dilemma in a child

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Background:
Membranoproliferative glomerulonephritis (MPGN) is a common diagnosis in children having a nephritic-nephrotic presentation. However, arterial involvement in children is almost unknown. We report an unusual case of a child with small vessel vasculitis associated with MPGN on renal biopsy and discuss the differential diagnosis.

Case presentation & result:
A 11-year-old girl had been apparently well until 20 days before admission, when she developed dry cough and fever with chills. Five days later she developed facial puffiness and swelling of lower limbs. On admission, she was found to have hypertension (150/110 mm Hg), pallor, pitting pedal edema, hypoalbuminemia, and moderate renal failure with a serum creatinine of 3.8mg%. There was no joint swelling or rash. Complete hemogram showed normocytic normochromic anemia (Hb-8.7g%) and neutrophilic leukocytosis (TLC-16,000/cumm). Platelet count and coagulation profile were normal. Urinalysis showed 3+ proteinuria and 20-25RBC/HPF. 24-hour proteinuria was 1.8 g/day. During the hospital stay, her renal function worsened, with creatinine level increasing from 1.6 to 2.4 mg/dL over 3 days. Serum complement levels were not low [C3 = 160 mg/dL (normal: 60-120 mg/dL) and C4 = 37.4 mg/dL (normal: 15-25mg/dL)]. ANA, ANCA and anti–ds DNA were negative. Viral serological tests for hepatitis B and C and HIV were non-reactive. Renal ultrasound showed normal-sized kidneys. A renal biopsy was performed which showed glomeruli displaying diffuse global endocapillary proliferation with lobular accentuation. Splitting of GBM was appreciated on silver stain. Interlobular arteries included in the biopsy revealed transmural fibrinoid necrosis with neutrophilic infiltrate. On immunofluorescence, granular capillary wall deposits of IgG (2+), C3 (3+), and C1q (1+) were noted. IgM and IgA were negative. None of the blood vessels included in the biopsy submitted for immunofluorescence showed vasculitis and were negative for immune deposits. A diagnosis of immune-complex mediated MPGN with small vessel vasculitis was offered. She improved after administration of steroids.

Conclusion:
Small vessels vasculitis in renal biopsies of pediatric patients is rare. In our patient, the pathogenesis of small vessel vasculitis is not very clear. Cryoglobulinemia, hepatitis B and C infection as well as ANCA-associated vasculitis were excluded. A rare possibility of immune-complex mediated arterial involvement remains the diagnosis after exclusion of all other causes.
Dual positive ANCA and anti-GBM mediated crescentic glomerulonephritis

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Background:

Literature states that 5-14% of ANCA positive patients also have anti-GBM antibodies. While ANCA positive crescentic glomerulonephritis (CGN) is a relapsing disease, anti-GBM disease is a one shot disease. ANCA positive CGN has a better prognosis than anti-GBM disease. Dual positive patients generally behave like anti-GBM disease. Screening all ANCA positive patients for anti-GBM antibodies is necessary to judge prognosis and guide treatment.

Case presentation and Results:

We report three cases of ANCA positive patients who were diagnosed to have concurrent anti-GBM disease by immunofluorescence in renal biopsies. The mean age was 52 years. The male-female ratio was 1:2. The presenting symptom was fever with oliguria in the first patient with a history of pontine infarct and pulmonary tuberculosis; fever with hematuria in the second patient; and fever with dry cough in the third patient. Interstitial lung disease was suspected in the third patient whereas others did not show evidence of lung involvement. All were hypertensive and non-diabetic. The mean creatinine was 8.3 mg/dl. The third patient was p-ANCA positive whereas others were c-ANCA positive. In all patients, renal biopsy revealed necrotising crescentic glomerulonephritis with chronicity changes. Fibrinoid necrosis of vessels was noted in the third patient. On immunofluorescence, all the biopsies showed linear positivity for IgG. By ELISA, two patients except the first were positive for anti-GBM antibodies. The first patient was pulsed with methyl prednisolone whereas others were put on methyl prednisolone, cyclophosphamide and plasmapheresis. The second patient was dialysis independent whereas others two were dialysis dependent.

Conclusion:

As all our patients were elderly with associated chronicity changes in the biopsies, we speculate that the patients might who were ANCA positive to begin with would have developed anti-GBM antibodies. These cases would have diagnosed as ANCA associated CGN if immunofluorescence was not done. Hence anti-GBM antibodies have to be done in all cases due to its aggressive course.
Simultaneous Tubular and Glomerular involvement with Cryoglobulinemic vasculitis in Multiple Myeloma: report of a rare presentation.

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Background:
Renal manifestations in myeloma are varied and may involve any compartment of the kidney. Tubulopathic light chains cause cast nephropathy or proximal tubulopathy, usually associated with tubulointerstitial nephritis. Glomerular involvement includes amyloidosis and monoclonal immunoglobulin deposition diseases. We report an unusual renal manifestation of multiple myeloma in an elderly patient presenting with both tubular and glomerular involvement on renal biopsy with associated cryoglobulinemic nephropathy.

Case Presentation:
A 63-year old male, diabetic for 10 years, was referred to the Department of Nephrology, SGPGIMS, Lucknow with features of distal symmetrical lower limb polyneuropathy, decreased vision and renal involvement in form of proteinuria, and mild renal dysfunction. A diagnosis of Diabetic nephropathy was considered. However, within two weeks he developed extreme fatigue, maculopapular rash in lower limbs, small and large joint polyarthritis and advanced renal failure. Investigations showed nephrotic range proteinuria, active urinary sediments and normal serum complement level. Fundus examination showed no features of diabetic nephropathy, instead retinitis pigmentosa was present.

Results:
Renal biopsy showed light chain cast nephropathy and glomerular involvement. Glomeruli displayed membranoproliferative pattern with monoclonal immunoglobulin deposition disease and features of cryoglobulinemia. Immunofluorescence showed kappa restriction in the tubular casts and glomerular deposits. Serum light chain assay and immunoelectrophoresis revealed IgG kappa light chain restriction.

Conclusion:
The exact mechanism of the varied renal manifestations of multiple myeloma in different patients is not known. We report a rare patient of multiple myeloma presenting with both tubular and glomerular involvement along with clinical and morphological features of cryoglobulinemia. Also whether retinitis pigmentosa is an incidental finding or may be related to the disease is not clear.
Nephrotic syndrome presenting with pulmonary venous thromboembolism and renal artery infarct - IgA Nephropathy

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Background:
Thromboembolic complications are not uncommon in glomerular diseases with nephrotic range proteinuria. Several factors are involved in the pathogenesis- thrombocytosis, decreased levels of antithrombin III, plasminogen, protein S and increased levels of factor V and VIII. Venous thromboembolism varies from 2% in children to 42% in adults in different studies. A relative risk of 1 to 5.5 of having arterial thromboembolic events have also been described.

Case report:
A 28 year old male presented with worsening breathlessness for 2 months. Patient was diagnosed outside with hypertension and was on calcium channel blockers. Dyspnea evaluation showed evidence of pulmonary thromboembolism and deep vein thrombosis in the right leg. CT also showed a wedge shaped suspected infarct in the lower pole of the right kidney which was also seen in DMSA. Patient was evaluated for prothrombotic state. Protein C,S,Factor V leiden, APLA were negative. Evaluation showed mild renal failure, microhematuria with nephrotic range proteinuria.

Result:
Renal biopsy showed IgA nephropathy with mesangial hypercellularity and chronic tubulo interstitial nephritis. Immunofluoresence was positive for IgA, IgG and C3. Patient was started on prednisolone and warfarin.

Discussion:
Studies have shown that absolute risks for thromboembolic events in venous and arterial were 1.02% per year and 1.48% per year respectively. Most common venous thromboembolism is deep vein of the leg followed by pulmonary. Arterial events present as myocardial infarctions or stroke. There are few reports of nephrotic syndromes with membranous nephropathy or minimal change. As far as we know this is the first reported case of an IgA nephropathy with a renal artery infarct and also with a pulmonary thromboembolism.
IgA mediated anti-GBM disease – a case report

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Background:

Anti-GBM disease is one of the most severe forms of glomerulonephritis which can manifest as Goodpastuer's syndrome, isolated anti-GBM glomerulonephritis or isolated anti-GBM pulmonary haemorrhage. Usually, anti-GBM disease is mediated by IgG antibodies. IgA-mediated anti-GBM disease is very rare disease with only 15 cases described in the literature.

Case presentation:

We present a case of 65 year-old, obese man, with glucose intolerance, hypertension, hypercholesterolemia, duodenal ulcers, arthralgia, high blood pressure and pretibial oedemas who was admitted to the hospital because of gross haematuria and nephrotic range proteinuria. Chest X-ray showed pleural effusion with consolidation of adjacent parenchyma. His serum creatinine increased from 92 to 313 µmol/L over the past two months. His serum protein levels were low with proteinuria of 11.8 g/day and haematuria. All serology tests were negative including circulating IgG anti-GBM autoantibodies. C3, C4 and serum IgA levels were normal. Urine and serum electrophoresis showed no monoclonal IgA, kappa or lambda light chains. Results Kidney biopsy showed necrosis and cellular crescents in 12% glomeruli, segmental sclerosis and fibrous crescents in 48% glomeruli. Global sclerosis was present in 40% of glomeruli. Interstitial fibrosis and tubular atrophy was present in 45% of tissue and severe hyaline arteriolosclerosis was also present. On immunofluorescent microscopy glomeruli were strongly globally linear positive for IgA, and week for IgG, kappa and lambda light chains. C3 showed weak granular positivity. IgM and C1q were negative. IgA was also focally positive in tubular basement membranes. There were two glomeruli with normal ultrastructure without immune deposits on electron microscopy. As soon as IgA-mediated anti-GBM disease diagnosis was made, patient was treated with plasmapheresis, oral cyclophosphamide and steroids. After two months he was clinically stable on albumin substitution and oral prednisone but cyclophosphamide was discontinued after second cycle due to the sepsis. Chronic hemodialysis was started 3 months after the onset of the disease. Conclusions Most commercial assays for detection of anti-GBM antibodies are specific to detect IgG antibodies. Standard assays for anti-GBM antibodies cannot detect IgA antibodies binding to different epitopes of type IV collagen. Diagnosis of this rare entity relies on pathologist interpreting kidney biopsy.
Reno-pulmonary syndrome due to alternate complement pathway dysregulation- A new kid on the block

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Background:
Dense deposit disease (DDD) results from dysregulation of alternate complement pathway (ACP) and is characterised by C3 deposition in kidneys. Confirmed extrarenal associations are lipodystrophy and age-related macular degeneration (AMD). We document a histologically confirmed pulmonary manifestation in a patient with DDD.

Case presentation:
22-year male presented with a history of facial puffiness for 2 years, hypertension for 1 year, and renal dysfunction and shortness of breath for 1 month. Urine showed 3+ albumin, 5-6 RBCs and proteinuria (0.35g/day). Investigation revealed hemoglobin 8.4mg/dl, creatinine 12.1gm/dl, total protein/albumin of 6.1/3.0gm/dl. Serology for all viral markers and ANA/ANCA were negative. In view of the normal sized kidney, a renal biopsy was done with a provisional diagnosis of crescentic glomerulonephritis. Kidney biopsy showed mesangioproliferative pattern and crescents, 3+ granular positivity for C3 in glomeruli and tubular basement membranes were suggestive of C3 glomerulopathy. Electron microscopy showed dense osmophilic intramembranous deposits confirming the diagnosis of DDD. Serological workup confirmed the abnormalities in ACP (low C3, APFA, factor H and normal C4, factor B) due to presence of antibodies (C3Nef and anti-FB) and presence of four SNPs of CFH gene (rs1061147, rs1061170, rs2274700 and rs35292876) in exon 7, 9, 10 and 13 respectively. During hospital stay, he developed symptoms suggestive of pulmonary haemorrhage. Plasmapheresis was done 5 times to remove the autoantibodies, however, due to the progression of pulmonary symptoms (reticulonodular shadows) he succumbed to illness. Lung biopsy confirmed the pulmonary haemorrhage due to C3 mediated vasculitis in alveolar capillaries.

Conclusion:
This case documents the extra-renal manifestation as pulmonary hemorrhages resulting from C3 alveolar capillaritis in a case of DDD presenting as a reno-pulmonary syndrome due to ACP dysregulation, presence of C3Nef & FB autoantibodies with significant SNPs of CFH.
Postinfectious GN with IgA dominance with ANA and ANCA positivity: Diagnostic dilemma.

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52 years-old female with no previous comorbidities presented to hospital with low grade fever, sore throat followed by hematuria and a decrease in urine output. There was no history of cough with expectoration, cold, burning micturation, flank pain, rash or joint pains. Investigations showed serum creatinine of 8 mg% with proteinuria and an active urine sediment. The patient was initiated on hemodialysis and serological markers sent. Serology showed ASO+ with titre > 200, ANA+, p-ANCA+ (Anti MPO+) and normal complement levels. Started empirically with injection methylprednisolone and a kidney biopsy was done which on LM and IF showed IgA dominant post infectious glomerulonephritis. Under the influence of steroids, the patient improved and gradually came off dialysis. The steroids were gradually tapered off. Two months later her serum creatinine was normal and the urinalysis too was normal. Considering rapid and complete response to steroid, we also label it as IgA dominant post infectious glomerulonephritis. After 6 months, she presented with dry cough since 1 month. This time HRCT done showed nodules in the lung, biopsy done labeled as 'organizing pneumonia'. In view of previous Anti MPO+ and respiratory symptoms, repeated anti MPO which was positive but this time no renal involvement (no proteinuria and active urinary sediments) and diagnosed as lung limited vasculitis. This case represents the complex autoimmune interaction and a difficulty in labeling the patient based on the clinical scenario, serology, histopathological findings and a difficulty in initiating appropriate immunosuppressive medications.
Crystaglobulinemia: expanding monoclonal renal spectrum

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Background:
Crystalline nephropathy refers to renal parenchymal deposition of crystals leading to kidney damage eg nephrocalcinosis, oxalate nephropathy and crystalline podocytopathy. Monoclonal proteins can also deposit in the kidney as crystals and cause tissue damage. We present a case of cast nephropathy with crystaglobulin within the glomerular capillary loops in case of multiple myeloma.

Case presentation:
60 years old male was diagnosed multiple myeloma in 2014, and was treated with thalidomide, dexamethasone and bortezomib. After 2 years he presented with acute renal failure and reduced urine output. He had compression fracture of vertebra, osteolytic lesions in mandible, pelvis and left femur, and rapidly rising serum creatinine and proteinuria. He had bicytopenia(Hb 9gm/dl), TLC 3600/mm3, platelets 122x103, serum A/G ratio was 0.7(4.28/6.1gm/dl), urea/creatinine 173/9.2 mg/dl and Na/K+ 142/3.7meq/dl. SPEP/UPEP showed M band in the gamma region and was confirmed to be IgG kappa on immunofixation with k: ratio 23.28(138.8:5.79) on free light chain assay. Bone marrow examination showed 25% plasma cells. Kidney biopsy showed 8 glomeruli with PAS negative rounded secretions within the glomerular capillaries. The tubules showed PAS negative fractured casts with focal giant cell reaction and significant interstitial inflammation. Direct immunofluorescence showed kappa restriction in the glomerular luminal secretions and tubular casts. All other immunoglobulins and complements were negative. Glomerular crystalline contents were confirmed on electron microscopy and cryoglobulins were excluded. Closest differential diagnosis on morphology was cryocrystaglobulins which was excluded by negative serology for cryoglobulins and negative electron microscopy.

Conclusions:
A rare complication of multiple myeloma results from crystallisation of monoclonal proteins in the Glomerular or systemic vasculature leading to vascular injury, thrombosis and occlusion. These crystals within the glomeruli highlights a widened morphological spectrum of myeloma kidney with heavy tumour burden akin to myeloma cast nephropathy and are labelled as crystaglobulinemia. Only a carefull histomorphology is key to its diagnosis.
Gout- A rare cause of Secondary Amyloidosis

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Background:
AA amyloidosis may complicate a number of chronic inflammatory conditions, including rheumatoid arthritis, juvenile chronic polyarthritis, inflammatory bowel disease, familial periodic fever syndromes, chronic infections, and certain neoplasms. Gout is a very rare cause for AA amyloidosis. Here we present a case of gout ina 24 yr old man with nephropathy and secondary amyloidosis , both diagnoses revealed on renal biopsy.

Case presentation:
24 yr year old gentleman with h/o joint pains since the age of 15 yrs. He gradually developed additive, persistent involvement of small and large joints involving the small joints of the hands, wrist, elbows, knee, ankle and toes . 3 years after onset of joint symptoms, he started developing recurrent, non painful, nodular lesions in the feet and fingers . Noticed swelling in the feet 3 months back . This gradually progressed to involve his legs . Examination: Vital signs – stable. No pallor, icterus, clubbing or generalized lymphadenopathy. No oral ulcers, red eye, alopecia, photosensitive rash, psoriatic plaques. Febrile, B/l pitting pedal edema in the feet and legs . Investigations: Hb 13.2, TC 10,400, Platelets- 5,00,000, ESR 110 , URE – Albumin ++++, 10-15 granular casts, 6-8 pus cells . Urine C/S negative Uric acid 11.4, Ferritin 334, Blood urea 16.8, Serum creatinine 0.66, Liver function tests – ALP 242.3, T bil 0.5, D bil 0.2, SGOT 29.2, SGPT -17, Albumin 2.8 Globulin 3.6 USG :normal sized kidneys with suspected medical renal disease.24 hr urine protein – 8.6 gm24 hr, urine uric acid ANA-2 + speckled in 1:100 dilution cANCA & p ANCA – negative S.C3: normal . anti dsDNA : negative

Result:
Renal biopsy was done which revealed Uric acid nephropathy with microtophi in the renal medulla and secondary amyloidosis( SAA positive by IHC)

Conclusions: Gout is a very rare cause of secondary amyloidosis. There are only about 28 cases reported in literature.
POST RENAL TRANSPLANT LYMPHOPROLIFERATIVE DISORDER- A REPORT OF INITIAL CARDIAC MANIFESTATION

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BACKGROUND
Post-transplant lymphoproliferative disorders (PTLD) are heterogeneous lymphoid disorders that complicate solid organ or hematopoietic transplants. They range from indolent polyclonal proliferations to aggressive lymphomas, are most often extra-nodal and occur within a year of transplantation. The incidence of PTLD following renal transplants is 1%, and cardiac involvement is also uncommon. We present an autopsy case of PTLD that developed 7 years after renal transplant and had an initial predominant cardiac involvement.

CASE PRESENTATION
A 30-year-old male underwent live-related renal transplant 7 years ago for chronic kidney disease and was on regular immunosuppression. He presented with a recent onset dyspnea on exertion and palpitation. On clinical evaluation, a diagnosis of atrioventricular dissociation was made and a permanent pacemaker was implanted. A fortnight after the procedure, clinical and radiological investigations revealed right cervical lymphadenopathy, sub-cutaneous / intra-muscular nodules, penile swelling, ill-defined hypodense hepatic mass, left eye proptosis related to bony erosions of left supra-orbital ridge and wall of frontal sinus, osteolytic lesions involving left temporo-parietal bones and mild pericardial effusion. A biopsy from the liver mass showed features of non-Hodgkin’s Lymphoma; EBV-LMP was negative on Immunohistochemistry. Over the next 2 months, he had repeated episodes rapidly filling and finally a fatal pericardial effusion. A complete autopsy was performed.

RESULT
At autopsy, moderate haemorrhagic pericardial effusion was noted. The heart showed lymphomatous deposits over visceral pericardium with extension into interatrial septum, summit of interventricular septum, basal aspects of atrioventricular valves and ventricular myocardium. Other organs involved were liver, bilateral native kidneys, pancreas, intestine and stomach. The lymphoid cells were positive for LCA and CD 20; a diagnosis of high-grade B cell non-Hodgkin’s lymphoma was made.

CONCLUSION
The unusual feature of our case was that the heart was the first and extensively involved organ by PTLD, which occurred 7 years after renal transplant. Such PTLDs are often EBV negative, less responsive to altered immunosuppressive regimens and associated with less favorable prognosis.
JEM-ARM200F
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The JEM-ARM200F, incorporating a STEM Cs corrector and a microscope column with dramatically improved mechanical and electrical stability, achieves the world's highest STEM (HAADF) resolution of 0.08 nm as a commercial TEM. The Cs-corrected extremely small electron probe achieves a remarkably increased current density, one order magnitude larger than conventional TEMs. Thus, the JEM-ARM200F provides ultimate atomic-level analysis and also higher throughput with dramatically shortened measurement time.

Point resolution: 0.08 nm (STEM)
0.19 nm (TEM)
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Accelerating voltage: 80 kV, 200 kV
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