CR-01. Renal involvement in Nail Patella syndrome

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Background.

Nail–patella syndrome (NPS) (also known as (hereditary onychoosteadysplasia HOOD syndrome) is a pleiotropic autosomal dominant disorder due to mutations in the gene LMX1B which mainly results in small, poorly developed nails and kneecaps, but can also affect many other areas of the body, such as the elbows, chest, and hips. Renal involvement in nail patella syndrome may be present in 40% of affected individuals. Renal involvement in this condition may present as proteinuria and about 5% of these patients develop renal failure.

Case:

12 year old boy presented with history of puffiness of the face associated with abdominal distension for the past 1 1/2 months. He was treated elsewhere as nephrotic syndrome with full dose steroids for 6 weeks, however his symptoms persisted and hence referred to CMC Vellore for further management. There was no history of hematuria, hypertension, jaundice or decreased urine output. There was a significant history of bilateral elbow deformities since birth.

O/E: Facial puffiness and pedal edema was present.

Acneiform rash seen over the entire body.

Features of dysmorphic facies present: high forehead and hairline, depressed nasal bridge and epicanthal fold

Musculoskeletal system: Bilateral fixed flexion deformity of the elbows. Swan neck deformity of both hands with inability to flex DIP joints of hands present.

Blood urea-49, creatinine-0.79 mg%.

Up/Uc ratio was markedly increased.

H&E showed features of mesangial proliferative glomerulonephritis. IF was non contributory.

EM showed deposition of dark fibrillar material in the electrolucent areas of GBM and mesangial matrix.

Conclusion: Renal involvement in nail patella syndrome is rare but can occur and can clinically manifest as proteinuria. As light microscopy and IF features are non specific, electron microscopy can confirm its diagnosis, if suitable clinical features are present.

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Background: Congenital Nephrotic Syndrome of the Finnish type (CNSF) is a rare and severe autosomal recessive disease caused by genetic defects in the components of the glomerular filtration barrier i.e. nephrin and podocin. The incidence is 1.2 per 10000 live births in Finland. Typical cases of CNSF have been reported all over the world but are very rare in India.

Case Report: A 2 year female child presented with complaints of generalized pitting edema, periorbital edema, pallor and repeated respiratory infections. One of the sibling had succumbed to nephrotic syndrome at 9 months of age. Investigations: Hb- 7.4gm% TLC -14700/cumm Urea-10mg% Creatinine- 0.39mg%. Urine – Albumin ++++. A diagnosis of Steroid resistant nephrotic syndrome was given when patient did not respond to initial treatment of cyclosporin and oral steroids and hence renal biopsy advised. Child showed no improvement and later developed renal failure. On histopathology- sections studied showed 3 cores of renal tissue with 25 glomeruli. About 5 glomeruli appeared immature with widened Bowmans space. Podocyte hyperplasia was present in four other glomeruli. Clusters of distal convoluted tubules showed dilatation with proteinacious material. Arteries and arterioles showed medial hyperplasia. About 10 % of the cortical area shows interstitial fibrosis. On Direct Immunoflorescence, one core with 13 glomeruli showed mesangial positivity for Ig A (1+), Ig M (3+), C3c(1+) and negative for C1q and IgG. Hence, diagnosis of CNSF was given based on histopathology and immunofluoroscence.

Conclusion: Here is a case of Congenital Nephrotic Syndrome of Finnish type being reported for its rarity in India.
CR-03. Glomerulocystic Kidney Disease - A rare case report

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Background: Glomerulocystic kidney disease is a rare kidney disease in which Bowman space dilatation is 2 – 3 times the normal size. The criterion for definition is that more than 5% of the glomeruli show cystic change. Very few case reports have been published in the literature. We herein present a case report of this rare entity.

Case Report: A nine year old male child presented with complaints of global developmental delay, progressive pallor and easy fatigability. Ultrasound revealed bilateral hyperechoic kidneys with no other urinary tract abnormality. There was no family history of similar problems. On further evaluation his serum creatinine was raised (6.2 mg/dl) with normal urinary protein levels. A renal biopsy was done to evaluate the cause. The kidney biopsy showed large glomerular cortical cysts in 20% of the glomeruli with juvenile tufts filled with proteinaceous coagulum.

Conclusion: Glomerulocystic kidney disease is a rare kidney disorder which presents in early childhood. It is frequently associated with other urinary tract abnormalities. Many familial forms and associated mutations have been identified. Hence presence of glomerulocystic kidney disease in a patient requires complete clinical examination and suspected mutational analysis of the patient.
CR-04. Oxalate Nephropathy Associated with Superimposed Diffuse Proliferative Glomerulonephritis

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Introduction: Primary hyperoxaluria (PH) is a rare autosomal recessive disease caused by deficiency of an oxalate metabolizing liver enzyme. The disorder typically makes its appearance in early childhood, in either sex, resulting in recurrent calcium oxalate nephrolithiasis and renal failure. The diagnosis is based on clinical and sonographic findings, urine oxalate assessment, enzymology and / or DNA analysis.

Case report: A 6 yr old boy presented to our hospital with periorbital puffiness and high grade fever since one week. There was history of preceding pyodermal skin lesions 3 weeks back. X-Ray KUB showed left sided stag horn calculus. USG Abdomen showed bilateral grade 2-3 renal parenchymal changes with left mild hydronephrosis. Laboratory investigations showed low C3, normal C4, proteinuria with increased serum urea and creatinine. A clinical diagnosis of chronic kidney disease stage 4 with left stag horn renal calculi was made. Renal biopsy done showed diffuse proliferative glomerulonephritis and tubules with refractile crystals, focal epithelial necrosis and damaged basement membrane, prompting a diagnosis of DPGN ( ? Infective) superimposed on crystalline tubulopathy ? primary hyperoxaluria. Subsequently, elevated urinary oxalate level was noted. Left pyelolithotomy, post D-J stent was performed on the patient and he was discharged on oral pyridoxine. Stone analysis showed high calcium and oxalate levels.

Conclusion: Hyperoxaluria should be considered in any child with nephrocalcinosis and all kidney stones require appropriate analysis. Infections are common and renal failure is a dreaded complication. It is thus important to include assessment for this condition in the investigation for childhood renal failure.
CR-05. ‘Atypical’ Atypical Hemolytic Uremic Syndrome

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Institutions: Department of Nephrology¹, Kasturba Medical College, Manipal University, Manipal. Department of Pathology², Manipal Hospital, Bangalore.

Background: Atypical hemolytic uremic syndrome (aHUS) is due to genetic defects in alternate complement pathway (AP). Development of autoantibodies to factor H (FH) as an etiology is rare and constitutes only 6-10% of all cases of AHUS.

Case Report: We report a 12 year old boy with aHUS with unusual features who presented with a rapidly progressive renal failure preceded by a brief febrile illness. He presented with subnephrotic range proteinuria, active urinary sediments and anemia (Hb<9g %) Platelets and LDH levels were normal and no schistocytes at admission. There was no evidence of sepsis, complements were normal and immune work up was negative. His kidney biopsy showed diffuse arterial and glomerular microangiopathy with mesangiolysis and fibrin thrombi, no crescents and acute tubular injury. Genetic work up for aHUS showed high titers of complement factor H antibodies (8300UI/L) with a deletion in the CFHR1-R3 gene. Schictocytes and LDH rise presented later on in the course. His family members on screening were negative. He was treated initially with pulse steroids and plasma exchanges (PE) with fresh frozen plasma and intermittent hemodialysis. There was complete Renal and hematological recovery after 10 PE with decline in CFH-Ab levels. He was put on steroids and azathioprine as maintenance therapy and continues to be in remission.

Conclusion: This case is unique as the typical features of atypical HUS like thrombocytopenia and schistocytes were absent at presentation and etiology being Anti factor H Antibodies. Prompt diagnosis with factor H antibody assays and treatment with PE and immunosuppression can lead to complete remission.
CR-06. Mesangial IgA Immune Deposits in Dengue Shock Syndrome - A Case Report

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Institution: Departments of pathology and nephrology*, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow (U.P), India

Background: Dengue can be associated with a variety of renal disorders including acute renal failure, hematuria, and transient proteinuria. However, histological features are less known as renal biopsy is rarely performed. We report renal histology in a patient of dengue shock syndrome.

Case report: A 50-year old male patient presented with high grade fever and joint pain for 5 days followed by reduced urine output, and altered sensorium. Investigations revealed anemia, leucocytosis and thrombocytopenia. Urine examination showed 3+ proteinuria, 2-3 RBCs and 8 WBCs/hpf. Serum creatinine was 5.8 mg/dL. Dengue serology showed elevated titres of dengue specific IgM and was negative for IgG. Patient had persistent anuria of >3 weeks duration, for which a kidney biopsy was performed.

Kidney biopsy showed glomeruli with increased mesangial matrix and tubules with changes of acute tubular necrosis (ATN). Some of the tubules showed isometric vacuolation suggestive of osmotic injury. Mild lymphocytic inflammatory infiltrate was seen in the interstitium. On immunofluorescence glomeruli displayed mesangial deposits of IgA (3+) and C3 (2+). A histological diagnosis of ATN with mesangial IgA deposits was offered. After multiple sessions of hemodialysis, urine output improved to 1.5 liters/day. Patient was discharged with S. Creatinine of 3mg/dl and 2+ proteinuria.

Conclusion: We report a patient of dengue shock syndrome with acute kidney injury and proteinuria associated with glomerular IgA deposits. The immune deposits suggest an immune-related phenomenon. Spectrums of lesions encountered in dengue are largely under-recognised. Knowledge of these lesions is important to understand the pathogenesis of renal injury in Dengue fever.

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Background: In 20–25% of patients with antiglomerular basement membrane GBM nephritis, p-ANCA is also present. Cases with dual antibodies are considered to be a vasculitis-variant of anti-GBM antibody nephritis. Such patients may not have a typical presentation of pulmonary-renal syndrome, resulting in delay of the correct diagnosis and initiation of treatment.

Case Report: We report the case of a 48 year-old woman, old treated case of pulmonary Koch’s who was referred to our hospital for unexplained renal failure. She denied having abdominal pain, fever, or hemoptysis and was not using any other nephrotoxic drugs including over the counter medications. Urine routine examination revealed a proteinuria of 3+ and hematuria of 80-90 RBCs/hpf. Serologically p-ANCA was positive. Clinically a diagnosis of rapidly progressive glomerulonephritis was made. Crescentic Glomerulonephritis was diagnosed on renal biopsy. Immunoflouresence showed a linear positivity of IgG along the glomerular capillary basement membrane.

Conclusion: The prognosis of double positive disease compared to isolated antiglomerular basement membrane disease is controversial. However, the risk of recurrence is higher in patients with persistently elevated ANCA levels following resolution of the acute episode. Such patients require frequent follow up, long-term maintenance immunosuppressive treatment, and re-initiation of induction therapy including plasmapheresis if the disease recurs with rapidly progressive glomerulonephritis.
CR-08. Renal involvement in a case of Churg – Strauss syndrome

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Background: Renal involvement in Churg – Strauss syndrome is uncommon involving 16 to 27\% of cases with crescentic glomerulo-nephritis being a rare manifestation.

Case report: A 32 year old male presented with cough and dyspnoea, 2 months prior to the biopsy when he was empirically started on anti-tuberculosis therapy. At that point s.creatinine was 1.4 mg/dl and peripheral eosinophilia was recorded. There was a steady decline of renal function with s.creatinine rising to 4.5 mg/dl in 2 months. He was also found to be hypertensive. Total WBC count at the time of biopsy was 28,000/cu mm with 59\% eosinophils. p-ANCA was positive. The renal biopsy showed a crescentic glomerulo-nephritis with 19 out of 22 glomeruli having cellular and fibrocellular crescents. The interstitium showed a diffuse dense infiltrate of eosinophils. IF study showed no deposits. Correlating with the lung involvement, the peripheral eosinophilia and the p-ANCA positivity, a diagnosis of crescentic glomerulo-nephritis in Churg - Strauss syndrome was made. With a course of pulse IV steroids, followed by oral cyclophosphamide, there was a dramatic improvement with serum creatinine dropping to 1.5 mg/dl.

Conclusion: This case documents a rare case of crescentic glomerulo-nephritis in Churg – Strauss syndrome mistaken at the initial presentation as pulmonary tuberculosis.
CR-09. Immune mediated Crescentic MPGN secondary to HBV infection: A rare presentation of a common infection.

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Background: Infection with hepatitis B virus is associated with membranous glomerulonephritis, membranoproliferative glomerulonephritis, polyarteritis nodosa, IgA nephropathy and PIGN. Though small focal crescents may be seen, the presence of > 50% crescents is rare in the absence of cryoglobulinemia. We report a case of hepatitis B virus related crescentic MPGN.

Case report: 44-year-old male presented to the nephrology OPD with history of fever for 2 weeks and swelling in the lower limbs for 1 week. On initial evaluation, he had hypertension, nephrotic range proteinuria, hypoalbuminemia, microscopic hematuria with active urinary sediment, moderate renal dysfunction, normal Sr. Bilirubin and liver enzymes. His autoimmune workup was negative with low c3. USG abdomen showed normal sized kidneys and normal liver echotexture. Viral serology was positive for HBsAg and HBeAg with high HBV DNA titres. On renal biopsy, 10 out of 11 glomeruli showed circumferential cellular crescents with endocapillary proliferation and capillary wall showing double contoured basement membranes. IF showed diffuse granular deposits of c3(3+) and IgG(2+) along the capillaries and mesangium. He was treated with 3 doses of pulse methylprednisolone with 5 sessions of plasmapheresis followed by oral steroids at 1 mg/kg under the cover of renal modified dose of Entecavir. He was discharged on maintenance dose of Steroids and Entecavir. His serum creatinine normalized to 1.2 mg/dL on follow up with partial remission in proteinuria.

Conclusion: In the absence of cryoglobulinemia, crescentic MPGN secondary to HBV infection is an extremely rare phenomenon. Aggressive immunosuppression with plasmapheresis under antiviral coverage improves the outcome.
CR-10. A Rare Case of ? Sero Negative Lupus

Authors: Dr. P. Sasanka (DM), Dr. K. Praveen Kumar DM, Dr. Desai Madhav DM.

Institution: Narayana Medical College, Nellore.

Background: The incidence of Seronegative lupus is very rare. Only 2% of lupus patients have seronegativity. Cases of SLE with a negative ANA, negative anti-DNA antibody and/or normal serum complement have been reported, indicating that lack of positive serologies does not exclude SLE.

Case History: A 20 year old female Non Diabetic & Non Hypertensive presented with facial puffiness & pedal edema, breathlessness, anuria, headache, cola colored urine since 4-5 days duration. No h/o suggestive of Vasculitis and Connective tissue disorders. Initially diagnosed as RPRF: Rapidly Progressive Renal failure. (RPGN/HUS/AIN.). During the course of hospital stay, after Renal biopsy she was diagnosed as Rapidly Progressive glomerular Nephritis with Membranoproliferative Pattern of Glomerular Injury. Immunological assays and complements are normal. Biopsy: LM: Wire loops, Endocapillary, Hypercellularity, Sub endothelial Deposits are seen. IF: IgG (+3), IgM (+1), C3 (+3) and C1q (+2) are positive over the capillary loops and mesangium.

Conclusion: In this case there are no features of ACR Criteria, ANA and dsDNA negative, Normal complement levels inspite of IF positivity, these findings suggested seronegative lupus. With written consent NIH protocol was initiated. Patient responded clinically and laboratory wise her creatinine was normalised and developed complete remission of proteinuria.
CR-11. Acute renal failure with cast nephropathy: A rare presentation of Multiple Myeloma

Authors: Pavneet Kaur Selhi, Sumit Grover, Pallavi Garg, Harpreet Kaur, Neena Sood

Institution: Dayanand Medical College & Hospital, Ludhiana, Punjab, India

**Background:** Multiple myeloma can involve kidneys in the form of cast nephropathy. ARF due to cast nephropathy can rarely be the first presentation of multiple myeloma. In this case a primary diagnosis of multiple myeloma was established on renal biopsy due to its characteristic histomorphology, which was later confirmed by bone marrow findings.

**Case Report:** A renal biopsy was performed for rapidly progressive renal failure of unknown cause with sudden rise in creatinine in a 52-year-old female patient. She had a long standing history of multiple joint pains with Raynaud’s phenomenon and gangrene of right and left index finger 12 years back with partial shedding of digits. The serum creatinine was 9.08 mg/dl; urine total protein excretion was 1.9 g/24h with 10-12 pus cells per hpf, however, no RBC was seen. A clinical diagnosis of Scleroderma Renal crisis was made. Renal biopsy showed pink eosinophilic fractured tubular casts surrounded by multinucleated giant cells focally with moderate mixed interstitial infiltrates. Henceforth, skeletal survey, serum protein electrophoresis, aspiration and biopsy of bone marrow were performed. Urine for Bence-Jones protein was positive, however, no M spike was seen on serum electrophoresis. Prominence of bone marrow plasma cells to the tune of 35% of ANC were seen making a diagnosis of Plasma cell dyscrasia- Multiple myeloma.

**Conclusion:** The renal biopsy is seldom the first test indicative of myeloma in a patient of ARF of seemingly unknown origin. Thus, the microscopic appearance of the tubules in this biopsy was easy to appreciate and was diagnostic of light chain cast nephropathy.

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Background: Multiple myeloma is a malignancy of plasma cells; most often leading to overproduction of monoclonal Immunoglobulin; so called M-protein. Myeloma Cast Nephropathy (myeloma kidney), refers to intrinsic Acute Kidney Injury (AKI) that results as the filtered light chain component of M-protein (Bence-Jones protein) exerts toxic & obstructive injury to tubules.

Aim: To evaluate clinicopathological features of 4 cases of cast nephropathy.

Methods: A total of 4 cases diagnosed with cast nephropathy between Jan’2010-Apr’2014 were analysed retrospectively as per histopathological and clinical criteria.

Results: All 4 cases were between age 44-62 yrs, 3 were males & 1 female. 1 case showed light chain cast nephropathy with early features of acute tubulopathy. Second case showed evidence of light chain deposition disease with inflammatory tubulointerstitial nephritis alongwith light chain cast nephropathy. Rest 2 of the cases showed presence of cast nephropathy with superadded hypertension induced changes.

Conclusion: Cast nephropathy is seen in 30 to 50% patients of multiple myeloma. The likelihood of underlying cast nephropathy is increased in cases of more profound AKI. The propensity of a particular myeloma light chain to produce cast depends on amount of Bence-Jones protein & its tendency to aggregate together with Tamm-Horsfall protein. Cast nephropathy can be misinterpreted as interstitial nephritis, amyloidosis, light chain deposition diseases, etc., hence vital for nephrologists to review the histology with pathologists to avoid misdiagnosis.
CR-13. Light Chain Cast Nephropathy With Acute Proximal Tubulopathy – A Case Report

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Institution: Department of Pathology¹ and Department of Nephrology², JSS Medical College, JSS University, Mysore

Background

The kidney is affected in a variety of dysproteinemias, the pathogenesis and the morphology of which varies depending on the etiology. The common morphological presentation of the affected kidney includes myeloma cast nephropathy, monoclonal immunoglobulin deposition disease, and amyloidosis. However, direct tubular damage by nephrotoxic light chains is an important pathologic mechanism for renal damage in some patients.

Case report

A 72-year-old female patient presented with extreme tiredness. On investigation, the patient was found to have anemia, proteinuria and severe renal failure requiring dialysis. Renal biopsy showed periodic acid Schiff negative casts filling some of the tubular lumens. Tubulopathic changes in the form of apical blebbing, flattening and simplification of brush border, tubular necrosis and loss of nuclei were also noted. Lambda light chain restriction was seen on immunofluorescent studies. On further evaluation, she was found to have plasma cell myeloma.

Conclusion

Proximal tubular damage is an important pathogenic mechanism in a subset of patients with myeloma and renal damage. Some of the light chains are capable of producing both distal nephron obstruction and proximal tubule damage. While some degree of clinically insignificant proximal tubular damage is present in most patients with plasma cell dyscrasias and nephrotoxicity, this lesion can be responsible for rapid renal deterioration.
CR-14. Cast Nephropathy – Incidental coexisting lesion in tumor nephrectomy specimen

Authors: Mohammed Musheb¹, Mahesha Vankalakunti¹, Deepak D², Shivashankar V², Somanna M², Rohan A³, Vishwanath S³, Ravishankar B³, Kishore Babu³, Prakash GK³, Ballal HS³.

Institution: Departments of Pathology¹, Urology², & Nephrology³. Manipal Hospital, Bangalore.

Background: Pathologic evaluation of tumor nephrectomy focuses mainly on diagnosis, grading, and staging of the neoplasm. The presence of coincidental non-neoplastic disease may have significant long term implications as these being non-localized can be expected in the contralateral kidney as well. Cast nephropathy is exceedingly rare in tumor nephrectomy and literature search did not reveal such cases.

Case Report: A 66-year-old diabetic male presented with vague abdominal pain / features of inguinal hernia and incidentally found to have mass lesion in left kidney on abdominal CT scan. Investigations revealed Hb-9.3 gm/L, serum creatinine-4.4 mg/dL, eGFR-24.3 ml/min/1.73m². Histopathologic evaluation showed a Clear cell renal cell carcinoma, pathologic stage: pT3a Nx. Non-neoplastic kidney revealed cast nephropathy. Immunohistochemistry for Kappa/lambda: 2+ and 3+ respectively in casts. Subsequent bone marrow examination revealed 50 % plasma cells. Intense "M" spike was seen in gamma region with Kappa light chain-9380 mg/L and Lambda-17.1 mg/L (K:L ratio 548). Serum IgG levels were elevated (2460 mg/dL). Skeletal survey showed lytic lesions in sacrum. He was treated with Bortezomib based regimen. At 2 months, serum creatinine is restored to 2.3 mg/dL.

Conclusion: Cast nephropathy in our patient could be suspected by careful examination of special stains highlighting importance of same in all nephrectomy specimens. Admixture of light chain monoclonal casts with Tamm-Horsfall cast decreases the accuracy of assessing monoclonality. We therefore conclude that adequate examination of non-neoplastic renal parenchyma is an important tool in recognizing theoretically any non-neoplastic kidney disease at risk for progressive renal disease after nephrectomy and could be an essential step in early providing preventive and treatment measures.
CR-15. Multiple Myeloma Related Amyloidosis: A Rare Manifestation

Authors: Dr. Arpitha, Dr. Sainath, Dr. Ravindra, Dr. Jaishree

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Introduction: Multiple myeloma is a clonal malignancy of terminally differentiated B lymphocytes characterized by uncontrolled proliferation of abnormal plasma cells. Amyloidosis is found in up to 30% of patients who present with multiple myeloma; conversely, multiple myeloma is present in up to 20% of patients who present with amyloidosis. But multiple myeloma presenting primarily as systemic amyloidosis without any other manifestations is rarely reported. We report a case of a woman who presented with nonoliguric acute kidney injury as the initial manifestation of multiple myeloma.

Case Report: A 41yr/f came with vague complaints of reduced appetite, nausea. Systemic examination revealed no abnormality. The blood investigation revealed deranged renal function tests with hypochromic anemia. USG abdomen showed medicorenal disease. Xray chest, spine, skull were all normal. Urine showed marked proteinuria without bence jones proteins. ANA was negative. Serum protein electrophoresis was normal. Kidney biopsy revealed amyloidosis with chronic interstitial inflammation. Serum and Urine immunofixation electrophoresis had increased free kappa light chain and free lambda light chain without M band. Bone marrow biopsy showed diffuse interstitial infiltration by plasma cells (60-70%) confirming the plasma cell dyscrasia and these cells express CD138 and showed kappa light chain restriction. This confirmed multiple myeloma then patient was started on chemotherapy and advised for bone marrow transplantation.

Conclusion: The signs and symptoms of multiple myeloma are nonspecific. Patients can present in a variety of clinical settings, which may delay the diagnosis and result in additional disease related complications. This case indicates that multiple myeloma can present as renal amyloidosis without any skeletal leisions, M protein levels elevation or bence jones proteins.
CR-16. Amyloidogenic cast nephropathy with intratubular amyloidosis

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Institution: Department of histopathology and Nephrology*, PGIMER, Chandigarh

**Background:** Myeloma cast nephropathy is commonest renal manifestation of multiple myeloma. Rarely casts are composed of amyloid and may be associated in few cases with intratubular amyloidosis (ITA), characterized by presence of amyloid within tubular epithelial cytoplasm. Both were present in current case.

**Case report:** A 48 year old male presented with pain over upper end of right humerus. Imaging revealed lytic lesion. He had renal dysfunction with serum creatinine of 5.7mg/dL. Monoclonal band was detected on serum (0.36g) and urine protein electrophoresis. With clinical diagnosis of rapidly progressive renal failure, ultrasound guided kidney biopsy was done.

Kidney biopsy revealed 7 normal glomeruli. The distal tubules were dilated and distended by presence of eosinophilic casts having lamellated central part with peripherally radiating spicules. These casts were periodic acid Schiff (PAS)-negative, Congophilic with apple green birefringence under polarizing microscopy. Few tubules contained PAS-negative protein reabsorption-like droplets in epithelial cell cytoplasm. Congo-red stain showed positive reddish-brown amyloid with apple-green birefringence in droplets of these tubules. Immunofluorescence revealed lambda light chain (LC) restriction. Ultrastructural examination revealed large collections of amyloid fibrils within tubular epithelial cells and in lumen forming amyloid droplets. A diagnosis of amyloidogenic light chain cast nephropathy with ITA, $\lambda$-LC type was made. Bone marrow biopsy done subsequently showed 30% $\lambda$-LC restricted plasma cells without any amyloid deposition. Patient succumbed to pulmonary infections before initiation of chemotherapy.

**Conclusion:** We report a rare case of amyloidogenic light chain cast nephropathy with ITA.
CR-17. Heavy and light chain Amyloidosis (AHL) in the background of multiple myeloma, rare subtype with an atypical presentation - case report

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Background: Amyloidosis is an uncommon disease characterized by deposition of proteinaceous material in the extracellular matrix which results from abnormal protein folding. Even though more than 25 precursor proteins are identified, majority of systemic amyloidosis results from deposition of abnormal immunoglobulin light chains; either kappa or lambda. In heavy chain Amyloidosis (AH) deposits are derived from immunoglobulin heavy chain alone whereas heavy and light chain amyloidosis (AHL) the deposits are derived from immunoglobulin heavy chains and light chains. Both AL and AHL are extremely rare diseases.

Case report: 57 year old gentleman known case of hypertrophic obstructive cardiomyopathy (HOCM) presented with history of worsening pedal oedema and bilateral symmetric peripheral neuropathy. His serum creatinine was 2.2 mg/dl 24 hour urine protein excretion was 2208 mg/day, urinary sediment was inactive. Serum electrophoresis revealed a monoclonal band in the region of gamma globulins; free light chains were elevated with a kappa/lambda ratio of 0.29. Light microscopy of the kidney biopsy showed a marked mesangial widening with prominent nodules composed of glassy, homogenous, eosinophilic material which was congophilic with apple green birefringence on polarised microscope. Scattered tubules show fractured casts. Many smaller and larger vessels showed similar material causing luminal occlusion. Immunofluorescence microscopy showed strong positive staining for heavy chain IgG and light chain lambda in the mesangium, along the glomerular basement membrane, tubular basement membrane as well as along the vessels. IgA, IgM, C3, C1q and kappa were negative. Electron microscopy confirmed as amyloid. Bone marrow aspirate showed more than 30% plasma cells along with plasma cell nodules in biopsy.

Conclusion: To the best of our knowledge this is the first report of AHL from India with an atypical presentation of complete heart block, which is a rare initial manifestation of AHL.

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Background: Amyloidosis is one of the important differentials for Nephrotic syndrome. We are hereby presenting a case of Amyloidosis secondary to active koch’s infection.

Case Report: 17 year old male was admitted with c/o fever and cough with expectoration since 15 days and pain abdomen since 7 days. On examination he was moderately built and poorly nourished. There was a lymphnode mass in the right axillary region. Review of history revealed the presence of right axillary mass since 3 years, and gradually increased in size. His investigations revealed mild azotemia, severe hypoalbuminemia, and nephrotic range proteinuria. After 2 human albumin infusions, he was subjected for renal biopsy which was called out as amyloidosis, without any restriction for kappa or lambda light chains on IF. A bone marrow aspiration and biopsy was done, serum was sent for immunofixation electrophoresis. Lymphnode mass was excised and was sent for histopathology. It was called out as tuberculous lymphadenitis. Meanwhile bone marrow aspiration / biopsy and immunofixation electrophoresis reports were available and unremarkable. IHC was positive for AA stain in renal biopsy. Hence it was concluded that this was a case of renal amyloidosis secondary to tuberculous lymphadenitis. Patient was started on ATT with modified doses and discharged. Later patient was lost for f/u and on telephonic conversation with the family it was learnt that patient had expired at home.

Conclusion: The purpose of presenting this case was that although Amyloidosis is commonly seen in our country secondary to pulmonary tuberculosis, Amyloidosis secondary to tuberculous lymphadenitis has rarely been reported in literature.
CR-19. Fibrillary Glomerulopathy- A Case Series

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Institution: Department of Pathology, Christian Medical College, Vellore

Background: In 1977, Rosenmann and Eliakim were the first to report a glomerulopathy, characterized by infiltration of glomeruli by “amyloid like” fibrillary deposits containing IgG and C3, which resembled amyloid but did not stain with Congo red. Fibrillary glomerulopathy is characterized by glomerular accumulation of non-branching, randomly arranged fibrils, ranging in diameter from 18 to 30 nm. Fibrillary and Immunotactoid glomerulopathy were grouped into a single disease category but they differ from each other pathogenetically , where Fibrillary glomerulopathy occurs in association with autoimmune diseases, monoclonal gammapathy and Hepatitis C and Immunotactoid glomerulopathy is associated with B cell neoplasms. Ultrastructurally, the fibrils are 18-30 nm in Fibrillary glomerulopathy and 30-60 nm in immunotactoid glomerulopathy.

Case report: We describe three cases of fibrillary glomerulopathy. On light microscopy, there was variable increase in mesangial cellularity, mesangial expansion by acellular eosinophilic deposits and capillary wall thickening. These deposits were PAS negative and showed a moth-eaten appearance on silver stain. On immunofluorescence there were mesangial and capillary wall deposits of predominantly IgG. Ultrastructurally, the mesangium was expanded by organised deposits characterised by haphazardly arranged fibrils of diameter 15-20nm.

Conclusion: Fibrillary glomerulopathy presents with persistent and progressive disease that results in early onset of end stage renal disease. Histologically, this condition has to be differentiated from other diseases with non immune organised deposits which include collagenofibrotic and fibronectin glomerulopathy and immune organised deposits such as amyloidosis, immunotactoid glomerulopathy and cryoglobulinemia. Hence, electron microscopy plays an important role in diagnosis and categorisation of these organised deposits.

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Institution: Departments of Pathology¹, & Nephrology²; Manipal Hospital, Bangalore.

Background: Pathophysiologic mechanisms of acute kidney injury in HIV-infected patients are diverse. Identification of the same is achieved by careful examination by light microscopy & immunofluorescence. However, Immune complex-mediated disease poses management dilemma- to increase immunosuppression or not! We present one such case diagnosed ultrastructurally.

Case Report: 55- yrs female, a k/c/o DM, HTN, DR, Recurrent UTI, severe anemia, CKD with baseline creatinine of 3.1mg% 3 months ago, presented with complaints of episodic hypoglycemia, dyspnea, reduced appetite and gross hematuria. At admission, serum creatinine was 12 mg/dL, Hb- 6.5 gm/dL, platelet count of 78000 and active urine sediments. Screening for HIV, HBsAg & HCV was done prior to hemodialysis & found to be positive for HIV-1. In view of the recurrent anemia, thrombocytopenia and severe renal failure, a renal biopsy was performed, and showed Immune-complex mediated focal mesangial proliferative glomerulonephritis with focal (25%) active crescents. IF showed diffuse and global finely granular deposits with IgG (3+) and C3 (2+) in the mesangial region. EM showed tubulo-reticular inclusions in the endothelial cells frequently, along with granular transformation of nuclei of mesangial cells, endothelial cells and podocytes. Latter showed nuclear bodies, and cytoplasmic multilamellated inclusions. Granular deposits were seen in the mesangium. CD4 count was < 50 cells /mm³, hence she was started on HAART therapy without adding any further immunosuppression. However, she succumbed to infections and sepsis.

Conclusion: Ultrastructure study is of utmost importance in identification of direct cytopathic effects and HAART-induced tubulo-interstitial injury. It helps in guiding treatment plan, and hence should be done in all cases of HIV infected patients with acute kidney injury.
CR-21. A case of fibronectin glomerulopathy

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Background: Fibronectin glomerulopathy is a rare hereditary disease with an autosomal dominant mode of inheritance, presents with proteinuria and slowly progressive loss of renal function. Glomerular fibronectin deposits that sometimes form organized deposits.

Case Report: An 18 year old young lady presented with recently detected hypertension, oedema and dyspnoea of 3 days duration. Urine protein : creatinine ratio was 17.65 and serum creatinine 11 mg/dl. A renal biopsy was done with a diagnosis of rapidly progressive renal failure. The renal histology showed enlarged glomeruli with prominent eosinophilic, PAS positive, silver negative deposits in the mesangium and along the capillary basement membrane. The deposits were red in colour on the trichrome stain, prompting a diagnosis of fibronectin glomerulopathy. There was focal glomerular obsolescence (4/14), moderate tubular atrophy and vessel changes of hypertension. IF study showed no deposits. Immune stains with anti-fibronectin antibody showed strong positivity confirming the diagnosis of fibronectin glomerulopathy. The girl is born to non-consanguineous parents with no history of renal disease in the family. Investigations of the parents and the single girl sibling (urine and blood) showed no abnormality. It is thereby assumed that this is a de-novo mutation and genetic studies in the family for fibronectin gene mutations are being attempted.

Conclusion: This case documents a rare case of Fibronectin glomerulopathy with possible de-novo mutation.
CR-22. Paroxysmal nocturnal hemoglobinuria and acute renal failure – report of 3 cases

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Background: Paroxysmal nocturnal hemoglobinuria (PNH) is an acquired clonal disorder due to stem cell mutation resulting partial or complete absence of glycosylphosphatidylinositol anchor, leading to dysfunction of CD59 and CD55 causing increased susceptibility to lysis by complement. Renal disease in PNH is mainly related to intravascular hemolysis which may be due to direct tubular toxicity of heme released from hemoglobin.

Case Reports: 3 patients with demographics: 18yrs/M, 40yrs/F and 23yrs/M presented with recent onset of macroscopic hematuria and severe anemia. Laboratory findings in all 3 revealed high serum creatinine levels, and proteinuria. Peripheral smear showed features of Microcytic hypochromic anemia and Coombs test was negative in all the 3 patients. Renal biopsy in first 2 patients showed normal glomeruli, the third patient showed normal sized glomeruli with focal proliferation of mesangial cells and increased mesangial matrix. Tubules in all 3 patients showed moderate acute tubular injury with presence of coarse brown pigment within the cytoplasm staining positively with Prussian blue. Renal hemosiderosis was diagnosed seeking work-up for hemolysis. Immunofluorescence (IF) was negative in first 2 patinets. In the third, IF showed diffuse/global granular deposits of IgA (3+) and C3 (2+), suggesting IgA nephropathy and renal hemosiderosis. Subsequent bone marrow study in first 2, revealed no significant abnormality with normal iron stores. Flow cytometry study revealed- deficient CD14 in granulocytes; and deficient CD59 in RBCs confirming diagnosis of PNH. Patient was managed with supportive management including dialysis for a week; followed by normalization of renal function.

Conclusion: Morphologic finding of iron pigment in renal tissue is crucial. A diagnosis of PNH should be considered in patient presenting with acute kidney injury associated with severe anaemia despite lack of peripheral evidence of intravascular hemolysis.
CR-23. Hemosiderin Cast Induced Acute Kidney Injury Secondary to Paraquat Poisoning

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Institution: Dept. of Pathology, Basic Sciences Block, Kasturba Medical College, Manipal.

Background: Paraquat is a broad spectrum herbicide known to be highly lethal to man and animals. Paraquat produces toxic effects through the generation of the superoxide anion, including little known and reported activity on RBC membrane stability, causing hemolysis, besides conversion of hemoglobin into methemoglobin. Multiorgan failure is characteristic, with literature review reporting rare cases of acute kidney injury.

Case Report: A 24 year old male was brought to the hospital with a history of paraquat consumption. On admission, his hemoglobin levels were low (8.4 gm %) with deranged liver and renal function tests and respiratory acidosis. Pseudocholinesterase was within normal levels. Urine examination revealed sugar, ketone bodies and bile pigments. Urine blood was 3+ with > 200 RBCs/ µL (1-2/hpf). Urine paraquat was negative. Inspite of supportive treatment, he succumbed. Autopsy revealed pale and yellowish kidney with firm and congested lungs. Liver also showed yellowish discoloration. Microscopy from kidney revealed normal glomeruli with an occasional one being hemosiderin stained. Tubules showed extensive intraepithelial hemosiderin deposits, with features of acute tubular injury & granular, epithelial, hyaline, hemosiderin and bile casts.Liver showed acute intrahepatic cholestasis, with lung showing intrapulmonary hemorrhage. A final diagnosis of acute kidney injury – toxic, secondary to paraquat induced hemolysis with superimposed cholemicnephrosis, acute intrahepatic cholestatis (drug induced) and intrapulmonary hemorrhage was given.

Conclusion: Organophosphorous compounds can cause acute kidney injury in humans, apart from other well-known effects on various other organs. Awareness of this is essential for early diagnosis and appropriate treatment in this direction.

Author: Dr Suma Raju MD

Institute: Nephrology, Narayana Multispeciality Hospital, (Former Assistant Professor, University of Southern California, USA)

Introduction:
Ciprofloxacin is a commonly used fluoroquinolone inspite of evidence of increased resistance. Acute interstitial nephritis secondary to ciprofloxacin usage is a well known entity at therapeutic dosages. Literature review shows scant reports of renal failure secondary to overdose of ciprofloxacin.

Case report:
Here we report a 50 year old male who presented as an emergency with anuria and renal failure 4 days after ingestion of 20 tablets of ciprofloxacin. Renal biopsy revealed granulomatous interstitial nephritis, acute tubular necrosis and crystals in the tubular lumen.

Conclusion:
Acute Interstitial nephritis is the most common biopsy feature of Ciprofloxacin induced renal failure. Isolated findings of acute tubular necrosis, granulomatous interstitial nephritis and crystals in the tubular lumen have also been reported. This case is unique for the presence of all three pathological findings.
CR-25. Acute Patchy Renal Cortical Necrosis following Paracetamol Overdose

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Background

Acute renal cortical necrosis is a rare cause of acute renal failure, secondary to ischemic necrosis of renal cortex. It accounts for 2% of all causes of acute renal failure in developed countries and more frequent in developing countries. The obstetric complications like abruptio placenta, severe pre-eclampsia/eclampsia are the most common causes and the other causes like infections, sepsis, shock, trauma, snake bite, drugs like NSAID & contrast media, glycol poisoning, heavy metal poisoning amount to 20-30% of the cases.

The incidence of acute renal failure in patients with paracetamol poisoning is less than 2%. Renal effects of paracetamol overdose are less common than hepatic effects. Acute renal failure occurs in 10-40% of the patients with severe hepatic necrosis. Acute renal toxicity may occur as a direct primary event.

Case Report

A 30-year-old female patient with a history of deliberate overdose of paracetamol presented with renal failure and oliguria. The patient was started on hemodialysis and remained dialysis dependent even after twelve days for which a renal biopsy was done. Renal biopsy showed patchy cortical necrosis. The liver function tests were normal throughout the hospital stay. Eight months after the biopsy, the patient is stable with a serum creatinine of 1.4 mg%.

Conclusion

In addition to hepatotoxicity, the clinical significance of nephrotoxicity in paracetamol overdose and the importance of monitoring the renal function must be recognized. Its severity and course may not be closely related to those of hepatotoxicity and it may also occur in low-risk patients.
CR-26. Pattern of histopathological changes of kidney in phosphorus poisoning – An autopsy finding

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Background

Toxic tubular injury can be caused by a number of agents which results in a more widespread form of injury than the ischaemic form. Yellow phosphorus, a toxic substance found in pesticides and firecrackers, is an unusual agent to cause tubular injury as it mainly affects the liver.

Case history

An 18-year-old male patient was admitted with a history of consumption of rat poison. His liver enzymes and serum creatinine levels were raised. The patient expired two days after admission, following which his liver and kidney were sent for histopathological examination. Multiple bits from kidneys and liver were received. Grossly, the kidney showed yellow discoloration. Microscopically, proximal tubular epithelial cells of kidney were predominantly affected and showed diffuse cytoplasmic vacuolation. Liver showed bile ductular proliferation with sinusoidal congestion, canalicular and hepatocellular cholestasis with vacuolar degeneration of hepatocytes. Thus, the toxic effects on liver and kidney were the causes of mortality in this individual.

Conclusion

As seen in the literature, marked histomorphological appearance, correlated with the clinical hepatic and renal findings, supported by the fact that yellow phosphorous showed a greater accumulation in liver and kidneys. Although the hepatotoxic effects of phosphorus are well recognized, literature provides limited information on effects of phosphorus on kidney. It is important to know these changes, in view of high incidence of rodenticide poisoning and high death rates with no effective antidotes available for treating these victims.
CR-27. Histopathological changes of the kidney in Sjogren’s syndrome

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Institution: Kasturba Hospital, Manipal University, Manipal

Background

Sjogren's syndrome is a progressive autoimmune disorder involving the exocrine glands, typically presenting with keratoconjunctivitis and xerostomia. Tubulointerstitial nephritis remains the most common presentation of renal involvement and often characterized by a distal (type I) renal tubular acidosis.

Case report

A 39-year-old female patient who was apparently normal 9 years back came with complains of bilateral leg swelling associated with red itchy lesions over both the lower limbs. She also had a history of bilateral knee joint pain since 1 year. She had noticed hyperpigmentation of the lips and buccal mucosa since 2 months. A skin biopsy done 1 year ago revealed leukocytoclastic vasculitis. On examination hyperpigmentation was noted on the lips and bilateral lower limbs. Laboratory investigations showed elevated 24hr urine protein with reduced serum C3, C4 levels. ANA profile showed Anti SS-A, Anti SS-B and Anti RO positivity. On renal histology, 3 glomeruli showed global sclerosis and 1 with fibrocellular crescent. Tubules showed microvacuolation and hobnailing, atrophy, tubulitis, hyaline casts and peritubular fibrosis. Interstitium showed dense aggregates of lymphocytes, with focal granulomas composed of epitheloid cells, scattered histiocytes and occasional neutrophils. Immunofluorescence was negative for IgG, IgM, IgA and C3. Based on the above findings a diagnosis of Sjogren’s syndrome was suggested and clinical co-relation requested

Conclusion

The varied histopathological changes that can be found in the kidney in patients with Sjogren’s syndrome need to be kept in mind while diagnosing renal involvement in connective tissue disorders. The most common pathological lesion in sjogren’s syndrome is tubulointerstitial nephritis.

**Authors:** Suvradeep Mitra, Kiran K, Manish Rathi*, Ritambhra Nada

**Institution:** Dept of Histopathology & Nephrology*, PGIMER, Chandigarh

A 28 year female diagnosed to have Systemic Lupus Erythematosus (SLE) presented with fever, generalized lymphadenopathy and pancytopenia. On examination, she was pale, had oral ulcers, generalized lymphadenopathy and skin rashes. She had subnephrotic range proteinuria (1gm/day) with active urine sediments and renal dysfunction (Blood urea nitrogen- 30mg/dL and serum creatinine- 2.60mg/dL). Serology revealed 3+ speckled pattern of antinuclear antibody and high titres of dsDNA. Rheumatoid factor, cryoglobulin, antineutrophil cytoplasmic antibodies and viral markers were negative. Patient had pancytopenia (Hemoglobin-8.3gm/dL, total leucocyte count-2500 /mm$^3$ and platelet count- 90×10$^3$/mm$^3$). Fine needle aspiration cytology of axillary lymph node and kidney biopsy were done.

Renal biopsy showed lupus nephritis-class IV (Diffuse proliferative glomerulonephritis with wire loops) with full house immunofluorescence positivity along with extraglomerular staining along tubular basement membranes with IgG and tissue ANA. No tuft necrosis or crescents were noted. There was focal moderately dense (30%) interstitial inflammation comprising of plenty of histiocytes along with few lymphocytes and plasma cells. Histiocytes showed karyorrhectic debris and haematoxylin bodies. Lymph-node aspiration showed findings of histiocytic necrotizing lymphadenitis. Tubulo-interstitial inflammation had similar morphologic characters Histiocytic necrotizing lymphadenitis and similar cutaneous involvement in SLE have been described in literature. However, to our knowledge the present case documents the first case of SLE with histiocytic necrotizing tubulointerstitial nephritis in renal biopsy with lupus nephritis. Patient was treated with Mycophenolate mofetil (2gm/day), Prednisolone (1mg/kg body weight-tapered at 2 months) and Hydroxychloroquine (200mg/day) with partial remission on 6 months of follow-up. Pancytopenia was alleviated and creatinine level came down to 1 mg/dL.

Identification of histiocytic necrotizing tubulo interstitial nephritis is clue towards histiocytic lymphadenitis. The impact of such lesion on overall prognosis of patient needs further investigation.

Authors: Debasis Gochhait, Kiran K, Suvradip Mitra, Ashwani Kumar, Raja Ramachancharan*, M Minz**, Ritambhra Nada.

Institution: Dept. of Histopathology, Nephrology* and Transplant Surgery**; Post Graduate Institute of Medical Education and Research, Chandigarh.

Background

Tumors like masses in kidneys with chronic kidney disease are adenomas or renal cell carcinomas especially with crystal formation. IgG4 related disease involves kidneys mostly as tubulo-interstitial nephritis with or without glomerular involvement. Mass forming fibro-inflammatory lesions in kidney constitutes only 26% of renal presentation and generally tend to have lower serum creatinine values (1.4 mg/dl) as compared to those who present with acute renal dysfunction and show tubulointerstitial nephritis.

Case report

A 44 year male patient presented in the nephrology OPD with recurrent urinary tract infections. On evaluation, he was found to have chronic renal failure- stage V along with a heterogeneous space occupying lesions in the cortex of left kidney. To characterize the nature of mass before transplant, a diagnostic biopsy was done. Histopathology of mass revealed plasma cells rich fibro-inflammatory lesion with many eosinophils along with expansile storiform type of fibrosis along with tubular destruction and atrophy. Few glomeruli included were sclerosed and blood vessels showed arteriosclerosis. On Immunohistochemistry with IgG4 > 30 plasma cells were positive staining. Serum IgG4 levels done were 400 mg/dl patient was started on steroids and reduction in mass was documented.

Conclusion

We have documented this case to highlight that mass lesion in background of chronic kidney disease is not always epithelial lesion; It can be IgG4 related fibro-inflammatory mass.
CR-30. Hypokalemic paralysis secondary to Renal Tubular Acidosis

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Institution: General Medicine, MGM Medical College, Kamothe, Navi Mumbai

Background: Renal Tubular Acidosis (RTA) is a multifactorial disease with multiple etiologies and varied manifestations. Association between Hypokalemic paralysis, RTA and Sjögren's syndrome is sparsely reported.

Case Report:

Case 1: 28yr female, presented with episodic weakness in 4 limbs, back pain, not gaining weight since 5 yrs. Treated as idiopathic Hypokalemic Periodic Paralysis (HPP) earlier. No signs of SICCA syndrome. On further evaluation serum potassium 2.1, serum bicarbonate low, thyroid function normal, urine potassium high, Anti-Ro & Anti-La positive. Diagnosis- Hypokalemic paralysis with distal RTA secondary to Sjögren's Syndrome (without any clinical signs of Sjögren's Syndrome). Patient is asymptomatic and on Shoal’s solution.

Case 2: 35yr male, presented with acute onset flaccid quadriplegia, difficulty in swallowing & difficulty in opening mouth, dryness of mouth & eyes. On investigation Serum potassium 1.3, serum bicarbonate low, urine potassium high, thyroid function normal. Diagnosis- hypokalemic paralysis with distal RTA. Responded to IV potassium supplementation.

Conclusion: As neither the history nor the clinical examination can differentiate between hypokalemic paralysis caused by RTA and that of familial hypokalemic paralysis, and because the emergency as well as prophylactic treatments of the two disorders are quite different, a simple differential diagnostic workup is emphasized.
CR-31. Candidal Renal Papillary Necrosis Conquered

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Renal papillary necrosis due to Candida albicans is a rare but treatable cause of acute renal failure. A middle aged male with history of type 2 diabetes mellitus presented with infected right lower extremity and right lower lobe pneumonitis with hypotension. Though, he improved initially, during his stay in the hospital, he developed acute renal failure. Blood culture grew Candida albicans, but was overlooked as possible contaminant. As the patient’s condition was deteriorating, a renal biopsy was done and it revealed candidal renal papillary necrosis. Subsequently ophthalmoscopic evaluation revealed that he also had candidal retinitis. He was treated with parenteral fluconazole for two weeks followed by oral fluconazole for a total of 4 weeks. Following treatment, the patient improved symptomatically and his renal parameters returned to normal. This case illustrates the need to consider candidal papillary necrosis as a differential in an immunocompromised patient with acute renal failure. It also highlights the crucial role of renal biopsy in diagnosis and management of the patient. Candidal renal papillary necrosis is potentially reversible, nonetheless a diagnostic and therapeutic challenge.
CR-32. Two unusual cases of microvascular thrombosis in renal allograft

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Introduction: Renal allograft thrombosis may be responsible for 2–7% of early allograft losses in adults.

Case – 1: A 20 year old lady with ESRD of unknown cause received a cadaveric donor kidney. The donor was a 23 year old lady, with multiple head injuries, who was declared brain dead and the transplant was done after 48 hours. Perfusion of the kidney was difficult after harvest and at anastomosis, the kidney was pale blue and flaccid with no urine output. A wedge biopsy showed diffuse glomerular microthrombi with no leucocyte margination; C4d and DSA were negative. A repeat biopsy on 6th day showed patchy cortical necrosis. The patient’s urine output had improved but at 6 weeks post transplant, she is still dialysis dependant.

Case – 2: A 23 year old man with ESRD of unknown cause received an unrelated live renal transplant. The urine output decreased abruptly post-anastomosis. On re-exploration, the kidney was blue, flaccid and anuric, and removed with a diagnosis of hyperacute rejection. The biopsy showed diffuse glomerular thrombosis with neutrophilic margination, a rare focus of mild peritubular capillaritis and minimal C4d deposits. Luminex cross match done 2 days and 15 days prior to transplant were negative with HLA class I and class II antibodies in both instances being <500 MFU. Antiendothelial antibodies were positive. History of blood transfusion in 8 months preceding transplant for unexplained hemoptysis was obtained.

Conclusion: Though the histology in both cases are similar, in the first case, the thrombosis was donor-related DIC (tests prior to transplant on donor showed a positive DIC profile) whilst the second case was probably a hyperacute rejection due to circulating non–HLA antibodies.
CR-33. Disseminated intravascular coagulation (DIC) in cadaver kidney donor: does it affect graft function?

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Institution: Department of Pathology, Department of Nephrology, Department of Surgery, St Johns National Academy of Health Sciences, Bangalore.

Introduction: DIC in kidney donor is considered as a relative contraindication for transplantation. Though delayed graft function (DGF) has been reported in some case studies, the long term graft function remains unaffected. We report two cadaveric renal transplant recipients where donors had DIC.

Recipient 1: 45 year old female with CKD on maintenance dialysis underwent cadaveric renal transplantation. Donor kidney was from diseased patient with DIC. Allograft biopsy showed glomerular fibrin thrombi and mild acute tubular necrosis (ATN). Post operatively she required dialysis for 22 days due to DGF. Allograft biopsies done on day 5 and 15 to rule out rejection showed normal glomeruli and mild ATN. Creatinine level was gradually normalized. Now after 10 months of follow up she is doing well on immunosuppression, without requiring dialysis.

Recipient 2: 48 year old female with hypertensive nephropathy on maintenance dialysis underwent cadaveric renal transplantation. The donor was a 23 year old male died of DIC following RTA. During retrieval, donor kidney appeared swollen. Day zero allograft kidney biopsy showed extensive glomerular fibrin thrombi and moderate ATN. Post operatively she was oliguric and required dialysis for 16 days indicating DGF. Day 9 allograft biopsy done to rule out rejection, showed normal glomeruli and pus cell casts in the tubules. Creatinine levels gradually normalized, but she expired on day 24 due to sepsis.

Discussion and conclusion: In both recipients DGF was observed. However recipient allograft biopsies done to rule out rejection showed normal glomeruli and mild ATN suggestive of ischemic injury. In both our cases despite of DGF, good graft function recovery was observed.

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Department (s) and Institution: Department of Pathology and Nephrology, Nizam’s Institute of Medical Sciences, Hyderabad

Background: Plasma cell-rich acute rejection (PCAR) is a morphologic type of acute rejection with prominence of plasma cells in the interstitial infiltrate and it indicates poor outcome. Though not included in Banff as a separate category; PCAR has been published in literature. Most of these PCARs are cellular rejections. However plasma cell infiltrates are also seen as a part of acute humoral rejection (AHR).

Case report: Total 166 allograft biopsies were received from January 2013 to May 2014 and four cases of plasma cell rich AHR were identified. Acute rejections were seen in 20.4% of the biopsies and plasma cell rich AHR accounted for 11.6% of these. All were male patients with a mean age of 36 years. The patients received live related allografts and were on triple immunosupression. Two of the patients had history of noncompliance. All of these were late AHRs seen with a mean transplant duration of 17 months. The mean serum creatinine at biopsy was 3.4mg/dl. Plasma cells constituted >10% of the interstitial infiltrating cells and c4d was positive in all 4 biopsies. All the biopsies had an associated tubulitis. The biopsies did not show Cd20 positive lymphoid aggregates and were negative BK virus immunohistochemistry. Three of the patients did not respond to the antirejection therapy and are dialysis dependent.

Conclusion: Plasma cell rich acute humoral rejection is a distinct clinicopathologic entity and a component of PCAR. It denotes poor outcome and resistance to routine antirejection treatment. Accurate recognition is necessary for appropriate management.
CR-35. Acute Antibody Mediated Rejection presenting as Nephrotic Syndrome – A report of two cases.

Authors: Nachiketa Mohapatra, Nisith Kumar Mohanty, Bibekananda Panda, Manas Ranjan Baisakh

Institution: Department of Nephrology and Histopathology, Apollo hospitals, Bhubaneswar, India

Abstract: Early diagnosis and treatment is important in acute antibody mediated humoral rejection (AMR). The most common presentation of AMR is impaired graft function characterized by rise in creatinine. Associated nephrotic range proteinuria is well established with rejection and needs clinical evaluation and alertness. There is no clear cut etiologic mechanism established for AMR presenting as nephrotic syndrome till date and the renal function is normal in previously reported cases. Therefore, the documentation in literature is limited for which most of the time ignored ending in chronic rejection, transplant glomerulopathy and interstitial fibrosis and tubular atrophy (IF/TA). One of the cases did not have renal dysfunction and the other presented with rise in Serum creatinine. Hence, rise in creatinine is not always an indictor of AMR and a clinical alertness of proteinuria should always be kept in mind.

Keywords: Late onset acute humoral rejection, Nephrotic syndrome, Renal transplantation.
OR-01. Histopathological Study of Renal Lesions in Pediatric Age Group

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Institution: Department Of Pathology and Nephrology, Institute of Medical Sciences, Banaras Hindu University, Varanasi (U.P) -221005

Introduction: Renal medical disease in pediatric patients, our experience in a tertiary care centre.

Objective: Our present aim was to see distribution among age, sex and percentage of various glomerulonephritis in children up to 16 years of age, at our institution.

Methodology: Total 98 cases of paediatric age group (less than 16 years) diagnosed between January 2011 to May 2014 were taken and analysed.

Results: Males predominated (53%) over females (47%). Percentage of male and female in 5-10 year age group are 5.1% and 3.1%, while in 11-16 year group they are 48.9% and 42.9%. Minimal change glomerulonephritis was seen in 14.3%, while membranous glomerulonephritis in 4.1%, focal segmental glomerulosclerosis in 3.1%, focal segmental mesangioproliferative glomerulonephritis in 39.8%, endocapillary glomerulonephritis in 15.3%, crescentic glomerulonephritis in 5.1%, lupus nephritis in 12.2%, end stage kidney in 3.1%, tubulo-interstitial nephritis in 1%, hemolytic uremic syndrome in 1% and amyloid nephropathy in 1% were found.

Conclusions: Focal segmental mesangioproliferative glomerulonephritis is the most common type of glomerulonephritis among paediatric cases found in our institution.
OR-02. Immature Glomeruli in renal biopsies - could they be the culprit in paediatric Steroid-Resistant Nephrotic Syndrome?

Authors: Suvradeep Mitra, Ashwani Kumar, Debasis Gochhait, Deepti Suri, S. Singh, Ritambhra Nada

Introduction

Nephronogenesis is known to complete by birth. However, immature glomeruli alone or in presence of focal segmental glomerulosclerosis (FSGS) are seen in a subset of paediatric renal biopsies done for steroid-resistant nephrotic syndrome (SRNS). Literature is largely silent on this subject; hence we planned to determine their significance by comparing with age matched controls from paediatric autopsy sections where cause of death was unrelated to kidney disease.

Material and Methods

Thirty cases of SRNS which had immature glomeruli (peripheral crowning of podocytes in H/E stain, absence of WT1 in IHC) were sub grouped in 4 categories (infants, 1-5 years, 6-10 years and 11-15 years) and percentage of immature glomeruli present were noted. These were compared with 55 paediatric kidney sections sub grouped into similar 4 groups. Almost 500 glomeruli were available in each autopsy section, equivalent to nearly 50 renal biopsies. Average percentage of immature glomeruli in each section was compared with corresponding age matched biopsy group by appropriate statistical test (chi square test).

Results

Significant nephronogenesis was observed till the age of 5 years. The percentage of immature glomeruli reduces with the age (neonates- 97.5%, n=29; infants- 80.8%, n=11; 1-5 years- 80%, n=5; 6-10 year- 3%, n=6; and 11-15 years-0.13%; n=4). Paediatric renal biopsies of cases of SRNS also show the presence of immature glomeruli (infants-73.5%, n=2; 1-5 years-28.7%; n=21, 6-10 years- 8.8%, n=5; 11-15 years- 4.5%, n=2). However, significantly more number of immature glomeruli persisted in SRNS patients with age groups beyond 5 years. (p value<0.05).

Conclusions

Immature glomeruli are present in paediatric kidneys in a significant proportion till the age of 5 years with reduction with increasing age. Persistence of glomerular immaturity is associated with SRNS in patients after 5 years of age.
OR-03. Paediatric Lupus Nephritis in a tertiary care centre

Authors: Deepasha Garg, Vineeta V Batra, Pallavi Agarwal

Institution: Department of Pathology, G.B. Pant Hospital, New Delhi.

Background: Lupus nephritis is not an uncommon presentation in paediatric population with an estimated 10 - 20% of SLE patients presenting before adulthood. It has been postulated that complement deficiencies predispose to development of nephritis in childhood. We herein present the prevalence and presentation of lupus nephritis in paediatric population in a tertiary care hospital in north India.

Aims and Objectives: 1] To examine the prevalence and clinical presentation of paediatric lupus nephritis in a referral tertiary care centre in northern India in a time span of 5 years and 3 months (Jan 2009 – March 2014). 2] To compare microscopic features with the varied clinical presentation.

Materials and Methods: The Kidney biopsies of patients with lupus nephritis were examined by LM and IF. The diagnosis and activity was confirmed by ANA, dsDNA, ANCA and Nuclear antigen line assay.

Results: A total of 39 no of cases of paediatric lupus nephritis were examined. The male to female ratio was 0.4:1. The biopsies were classified according to ISN/RPS classification. The various diagnosis rendered were class II (n=3), class III (n= 8), class IV (n=12), class V (n=30, mixed class IV +V (n=3). ANA was positive in 32 of these cases and dsDNA was positive in 20 of these cases. Associated ANCA positivity was seen in 2 cases.

Conclusions: SLE involves the kidney in a significant number of paediatric patients. The morbidity and mortality in these patients is high due to high activity of disease and poor drug compliance. It is therefore important to study these patients and keep them under close follow up.
OR-04. Evaluation of Complement Regulation in C3 Glomerulopathy: An Experience at PGIMER, Chandigarh

Authors: Ashwani Kumar¹, Ritambhra Nada¹, Raja Ramachandran², Vivekananda Jha², R.K. Vashistha¹, Kusum Joshi¹.

Institution: Department of Histopathology¹, Department of Nephrology², PGIMER, Chandigarh, India.

Background

Dysfunction of alternate complement pathways results in C3 glomerulopathy (C3GP) and atypical Hemolytic uremic syndrome. We investigated patients of C3GP for functional assay and regulators of alternate complement pathway.

Aims & Objective

To study complement factors (C3, C4), alternate complement pathway regulators i.e. factor H(FH), factor B(FB) and alternate pathway functional assay(APFA) in patients with C3GP.

Material & Methods

Serum of 51 patients of C3GP was collected for this study. Complement levels (C3&C4), FH, FB and APFA was tested in 51, 37, 41 and 44 patients respectively with 40 controls. C3 and C4 measurement was performed by nephelometer (Binding Site Minineph Human C3 & C4 kits). ELISA was performed for APFA (Wieslab assay kit), FH and FB levels (Assaypro). Clinical details were taken from department of Nephrology.

Results

In patients of C3GP, C3 levels were low and C4 levels were normal in most of the cases. APFA showed very low complement activity in all cases and deficiency were noticed in 7% (n-3).

Decreased levels of FH were found in 45% (n-17) whereas 55% (n-20) had normal levels. In contrast, FB was normal in 97% (n-40) and was decreased in only 3% cases (n-1).

Conclusions

Decreased level of complement components and complement functional activity confirms participation of alternate complement pathway in pathogenesis of C3GP. Point of dysfunction in half of these patients possibly results from factor H involvement. This lower factor H level can be due to autoantibody formation or genetic abnormality which needs to be further evaluated as it will have therapeutic implication.
OR-05. Spectrum of biopsy-proven renal disease – A Study from North East Region of India

Author: Dr. Ronica Baruah MD, DCP & Dr Rohit Goel MD.

Institute: Ekopath Metropolis Laboratory, Guwahati

The spectrum of renal diseases has a changing pattern with time and depends on age, ethnicity and geographical variation. Unfortunately renal biopsy data is insufficient, particularly from north east India. The aim of the study was to investigate the pattern of renal diseases as seen on biopsy in Guwahati, which is a referral centre for North East India. This study included patients from a wide range of ethnic groups from Assam and neighboring states. A total of 414 renal biopsies were analyzed from January 2011 to October 2013. Approximately 90% of the biopsies (342 cases) had adequate samples, and majority of them had tissue for both light microscopy and direct immunofluorescence. The most common pathology seen was Lupus Nephritis (23%), followed by Minimal Change Disease (22%), and IgA Nephropathy (12%), - these three accounted for 57% of the total cases. Regarding the ethnic distribution of Lupus Nephrites, Bodo- Kachari Communities showed a significantly higher incidence (28%) as compare to its total population (5-6%) in North East region of India. Majority of Lupus Nephritis cases were ISN/RPS Class IV (45%) and had active lesions. The incidence of Lupus Nephritis and IgA Nephropathy were higher than seen in other Indian studies, while the incidence of FSGS was lower than others in our group.
OR-06. Spectrum of Biopsy proven Renal disease and its clinical presentations in MS Ramaiah Medical Hospitals.

Authors: Dr Dinesh Kumar A, Dr Gurudev KC, Dr Mahesh E, Dr Vijayanyasorekar, Dr Gireesh MS, Dr Vijay Varma, Dr Rakesh.

Institution: M S Ramaiah Medical College/Hospitals, Bangalore

Background: Renal biopsy data analysis is essential to study the prevalence of biopsy-proven renal disease. We have completed 5 years of collection of renal biopsy data at our center and reported the pattern of BPRD.

Aims & Objectives: 1] To know the incidence of biopsy proven Renal diseases. 2] To study biopsy proven renal diseases and their clinical presentations

Material & Methods: All the kidney biopsies performed in our institute from October 2008 to November 2013 were retrospectively analyzed. We recorded the all data for each patient.

Results & Conclusions: A total of 754 biopsies were analyzed. The most common indications of renal biopsy were nephrotic syndrome (36.87%), followed by chronic kidney disease (22.01%) and Acute kidney Injury (17.77%). Primary glomerular disease comprised 465 (61.67%) of the total patients. Among the PGD cases, the most common one was minimal change disease (13.39%), followed by chronic glomerulonephritis (13.26%), membranous nephropathy (6.89%), focal segmental glomerulosclerosis (6.1%), membranoproliferative glomerulonephritis (4.90%), crescentic glomerulonephritits(4.77%), postinfectious glomerulonephritis (4.50%), IgA nephropathy (4.11%), diffuse proliferative glomerulonephritis (2.91%), and IgM nephropathy (0.79%).Secondary glomerular disease accounted for 96 (12.73%) of the cases. The most common SGN was diabetic nephropathy (7.29%), followed by lupus nephritis (5.03%), multiple myeloma (0.39%), amyloidosis (0.26%) and hemolytic uremic syndrome (0.13%). Tubulointerstitial disease [135 (17.29%)] and vascular disease [55 (7.29%)] were also common. This study provides descriptive biopsy data and highlights the changing incidence of renal disease which is probably contributed by an increase referral due to increased awareness together with increased manpower and infrastructure.
OR-07. Histopathological Patterns of Adult Renal Disease in a Tertiary Care Centre in Manipal, Karnataka – An 8 Year Epidemiological Review

Authors: Dr. Manna Valiathan, Dr Anuradha CK Rao

Institution: Kasturba Medical College, Manipal University, Manipal, Karnataka

Background: The pattern of adult renal disease in the Dakshina Kannada-Udupi districts is not known.

Aims and objectives: The study was conducted to analyze the patterns of adult kidney disease in a tertiary care centre (Kasturba Hospital, Manipal University, Manipal, Karnataka).

Materials and methods: A retrospective analysis of kidney biopsy reports from 2005 to 2012 was done. Pediatric, inadequate, renal allograft and post mortem biopsies were excluded from the study.

Results: 1126 renal biopsies were included in the study. Focal segmental glomerulosclerosis was the most common lesion (14.4%) followed by Acute Postinfectious glomerulonephritis (7.8%) Membranoproliferative glomerulonephritis (7.54%), End stage renal disease (7.37%) IgA nephropathy (6.45%), Membranous glomerulonephritis (6.21%), Minimal change disease (5.59%), Crescentic glomerulonephritis (2.93%). Among secondary glomerular disease Lupus nephritis was most common (8.88%) followed by diabetic nephropathy and hypertensive nephropathy. Chronic tubulointerstitial nephritis constituted 5.77% of cases.

Conclusion: The commonest glomerular lesion in this series was FSGS, followed by Post Infectious glomerulonephritis. A significant percentage was diagnosed in end stage, emphasizing the need for early diagnosis and intervention. This study underscores the need for a renal biopsy registry which could reveal significant information on the patterns of adult renal disease in this region.
OR-08. Renal Biopsy in the Elderly: The Spectrum of Diseases

Authors: Sumita Shrivastava, Swarnalata Gowrishankar, Meenakshi Swain, Michelle De Padua

Institute: Department of Pathology, Apollo hospitals, Jubilee hills, Hyderabad

Background: Aging leads to changes in the renal morphology and a compromise in renal function, further complicated by diseases like diabetes and hypertension. Kidney biopsy has emerged as an important diagnostic tool to help disease management.

Objective: To establish the frequency of varying histological diagnoses in the elderly undergoing kidney biopsy, with follow-up of patients presenting as acute kidney injury (AKI) and a comparison of nephrotic syndrome (NS) causes with other age groups.

Material and Methods: A retrospective assessment of renal biopsy reports of patients aged 60 and above (Elderly) along with follow-up of AKI cases and comparison of causes of NS with those in other age groups. The study period was from 2011 to 2013. Transplanted kidneys and nephrectomies were excluded.

Results: Of 566, 432 patients were between 60-69 years, 116 between 70-79 years and 16 were above 80 years. There was a male predominance 2.4:1 (Male 399; Female 167). The most common clinical presentation included chronic kidney disease (CKD) (33.2%) followed by AKI (27.6%); NS (26%); RPRF (12%) and nephrotic-nephritic syndrome (1.2%). Diabetic nephropathy (DN), obstructive uropathy and hypertensive nephrosclerosis were the major etiologies of CKD. Most of the cases of AKI were due to obstructive uropathy and sepsis. The cases of AKI are being followed up. The commonest cause of NS was membranous nephropathy (MN) (34%) followed by minimal change disease (MCD) in the elderly. In 0-5yrs and 6-15yrs age groups, commonest cause was MCD (68% and 40% respectively). However, between 18-59yrs, it was MN (24.4%) followed by MCD (21.6%).

Conclusions: CKD was the commonest presentation and DN, the commonest cause of the same. Causes of NS vary according to age. The great diversity of diagnoses in the elderly is further complicated by age-related co-morbidities.
OR-09. Renal biopsy findings in patient with hypothyroidism (report on seven cases)

Authors: Dr. Varnika Rai, Dr. Usha Singh, Dr. R.G. Singh

Institution: Department Of Pathology and Nephrology, Institute Of Medical Sciences, Banaras Hindu University, Varanasi (U.P) -221005

Background: Hypothyroidism is a common form of thyroid disorder is highly prevalent in India but data regarding histopathological findings in renal biopsies of patient with hypothyroidism are very hardly accessible in our country.

Aims and Objectives: Our present aim is to see histopathologic findings in renal biopsies of patient with hypothyroidism in our institution.

Material and Methods: Total 7 diagnosed cases of hypothyroidism with renal disorders between June 2012 to April 2014 were taken. Paraffin sections were stained for H&E, PAS and AFOG stain.

Results: 5 out of 7 cases (71.43%) were males and only 2 were females (28.57%). Age varied from 25 to 51 years. All cases were having hypothyroidism for last 5 years and were taking irregular treatment. All had microscopic hematuria. Two had renal failure (28.57%) and 5 had nephrotic syndrome presentation (71.43%). (Out of 7 cases, 3 cases (42.85%) were diagnosed as focal segmental glomerulosclerosis with tubulointerstitial nephritis. 2 cases out of 7 (23.57%) were diagnosed as mixed glomerulonephritis in which predominantly there were membranous glomerulonephritis with focal segmental mesangial cell proliferation, another 2 cases (23.57%) had membranous glomerulonephritis. One of these cases had features of SLE and was positive for ANA and Anti-dsDNA. Two patients diagnosed as FSGS were taking antitubercular treatment also.

Conclusion: Thus our study concludes that hypothyroidism cases give rise to renal vascular diseases in the form of focal segmental glomerulosclerosis or membranous glomerulonephritis.
Abstracts: 7th International CME in Renal Pathology & Annual Conference of ISRTP  

Aug 1-3, 2014, Mysore  

OR-10. Primary Focal Segmental Glomerulosclerosis: A Clinicopathological Study and Correlation with Histopathological subtypes  

Authors: Mary Mathew, Sakhi Anand  

Institute: Department of Pathology, Kasturba Medical College, Manipal University, Manipal, Karnataka, India  

Background: Focal glomerulosclerosis (FSGS) has been classified into five subtypes according to the Columbia classification. This study is a classification of the morphological variants and its correlation with prognostic histopathological features with clinical and laboratory findings.  

Aims & Objectives: 1) Reclassify 41 renal biopsies of primary FSGS according to the modified Columbia Classification by D’Agati. 2) Establish the frequency of FSGS variants in the present cohort. 3) Correlate clinical and laboratory features with histopathologic features and variants of FSGS.  

Materials and Methods: 41 cases of primary FSGS diagnosed in 5 years were re-classified and clinical and laboratory parameters were correlated with histopathological subtypes.  

Results: FSGS (NOS) was the most common subtype (51.2%), followed by perihilar (24.4%), tip (19.5%) and cellular lesions (4.9%). Nephrotic syndrome was most commonly associated with the cellular and the perihilar variant and least in the tip variant. 50% of the tip and cellular variant presented with renal insufficiency. A significant correlation was observed between serum creatinine and mesangial hypercellularity, podocyte hyperplasia, arteriolar hyalinosis, intimal sclerosis and medial hypertrophy. Associations between interstitial fibrosis, intraglomerular foam cells and hematuria and degree of proteinuria, adhesion and intimal sclerosis were statistically significant on univariant analysis.  

Conclusions: This comprehensive study reiterates that the variants of FSGS have substantial differences in clinico-histopathological features, however larger studies are required to elucidate the prognostic significance.
OR-11. Clinico-Pathological Study of Membranous Glomerulonephritis in a Tertiary Care Centre

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Institution: Department of Pathology and Nephrology, Institute of Medical Sciences, Banaras Hindu University, Varanasi (U.P) -221005

Background: Membranous glomerulonephritis is a common cause of nephrotic syndrome in adult but in our country detailed work up on membranous glomerulonephritis is hardly available.

Aim and Objective: Our present aim was to see clinical, pathological and immunofluorescence finding in membranous glomerulonephritis at our institution.

Material and Methods: Total 60 cases of membranous glomerulonephritis diagnosed between July 2011 to May 2014 was taken. Paraffin sections were stained for H&E, PAS and AFOG stain. Direct immunofluoresence was done.

Results: Male female ratio is 2.1:1. Only 5 cases (8.3%) were below 20 years and 50% cases were between 20 to 40 years. Nephrotic range proteinuria was seen in 37 cases (61%). Nineteen patients (31%) presented with microscopic hematuria. Out of 60 cases 3 cases had clinical features of SLE with ANA and anti-ds DNA positivity. On microscopic examination, uniform GBM thickening seen in 50 cases (83%) and focal GBM thickening in 10 cases. Twenty nine cases showed focal mesangial cell proliferation and 10% cases showed intraglomerular thrombi. Tubular atrophy was present in 29 cases. Interstitium showed mild mononuclear infiltrate in 60% of cases. Hyaline thickening of blood vessel was seen in 48% of cases and vasculitis in 8 cases. Direct immunofluoresence microscopy showed linear granular deposit of IgG, C3 and C4 in all cases.

Conclusion: More than 90% cases were of primary membranous GN and SLE was the common cause of secondary membranous GN.
OR-12. Clinicopathological Study of Mesangiocapillary Glomerulonephritis: Our Experience in a Tertiary Care Centre

Authors: Dr Ashish Kr Gupta¹, Dr Usha¹, Dr R G Singh², Dr S Singh², Dr Jai Prakash²

Institutions: ¹Department of Pathology, ²Department of Nephrology. Institute of Medical Sciences, Banaras Hindu University, Varanasi

Introduction: Mesangiocapillary glomerulonephritis, also known as membranoproliferative glomerulonephritis (MPGN) is the commonest cause of nephrotic range proteinuria in the adults.

Objectives: Our present aim was to study clinicopathological spectrum and immunofluorescence findings in the MPGN.

Methodology: Total 36 cases of MPGN diagnosed by renal biopsy since 2011 were included in the study. Paraffin sections were stained for H&E, PAS and AFOG stains. Direct immunofluorescence was done.

Results: M:F ratio was 1:1. 18 cases (50%) were below 20 years of age and 27 cases (75%) were below 40 years of age. Major symptom of swelling of the various body parts presented in 29 cases (80%). Nephrotic range proteinuria was observed in 29 cases (80%), microscopic hematuria in 15 cases (42%) and gross hematuria in 2 cases. Serologically, eight patients (22%) had reduced level of C₃ and three cases were found to be positive for ANA & ds-DNA. On light microscopy, all 36 cases showed variable degrees of mesangial cell proliferation, GBM thickening with enlargement of glomeruli in 33 cases (92%). 22 cases showed increase in mesangial matrix and mesangial cell interposition. Proliferation of endothelial cells and duplication of GBM in seven cases. Tubules in 26 (72%) cases found to have cloudy swelling and necrosis, 18 cases had tubular atrophy and 8 cases had dilated tubules. 20 cases (55%) had focal to severe mononuclear cell infiltration in interstitium. Hyaline thickening of blood vessels seen in 18 cases and five cases showed features of vasculitis.

Conclusion: MPGN mainly involves the young adults. Most of the cases are idiopathic in nature. SLE and diabetes mellitus being the secondary causes of MPGN.

Authors: Dr Deepak M Nadig¹, Dr Suchitha S¹, Dr Mahesha Vankalakunti³, Dr Manjunath S Shetty², Dr Manjunath G V¹

Institutions: ¹Department of Pathology, ²Department of Nephrology, JSS Medical College, JSS University, Mysore, ³Department of Histopathology, Manipal Hospitals, Bangalore.

Background: Although kidney disease has been a recognized complication of HIV infection since the beginning of the HIV epidemic, its epidemiology, underlying causes and treatment have evolved in the era of highly active antiretroviral therapy (HAART). Early recognition and treatment of kidney disease are imperative in lessening morbidity and mortality in this patient population.

Aims & Objectives: To study the clinico-pathologic spectrum of renal diseases in HIV positive patients.

Materials & Methods: This study analysed the renal biopsy findings and clinical features at presentation in 27 symptomatic retroviral positive patients over a period of two years (2012–2014). All retroviral positive patients with renal dysfunction undergoing renal biopsies between July 2012 and June 2014, were included in this study. For each biopsy, the slides were stained with haematoxylin and eosin, periodic acid Schiff, masson’s trichrome and Jones silver stains. Immunofluorescence pattern was noted.

Results: Of the 27 patients, seven (25.9%) were females and 20 (74.1%) were males, the male: female ratio was 2.86: 1. Patient age ranged from 24 to 70 years (mean ± SD; 44.77 ± 11.79 years). The most common indication for renal biopsy was rapidly progressive renal failure (33%), followed by nephrotic syndrome (26%) and chronic renal failure (23%). The glomerular histologic lesions included focal segmental glomerulosclerosis (with one of collapsing variant), diffuse mesangiproliferative glomerulonephritis, membranous glomerulonephritis, immune mediated glomerulonephritis, IgA nephritis and thrombotic microangiopathy. The nonglomerular histologic lesions included acute tubular injury, chronic interstitial nephritis and urate induced chronic tubulointerstitial nephritis. Five of the cases showed comorbid lesions of diabetic nephropathy.

Conclusion: This study provides descriptive biopsy data and highlights the prevalence of renal disease among retroviral positive patients in a south Indian population. In contrast to other studies where classic HIV associated nephropathy (HIVAN) was the predominant lesion, the current study depicts a variety of glomerular and tubulointerstitial disorders.
OR-14. Renal Biopsy with crescents – A 5 year clinicopathological study in a tertiary care hospital

Authors: Gayatri Ravikumar¹, Pritilata Rout¹, Isha Garg¹, Prashant G Kedalaya²

Institution: Departments of Pathology¹ and Nephrology², St. John’s Medical College and Hospital, Bangalore

Background: Glomerular crescents are associated with rapidly worsening renal function and may be found in various glomerulopathies. This study aims to categorise the causes of crescents at our centre.

Aims and Objectives: To study – 1] the incidence of crescents and crescentic glomerulonephritis (CGN) in renal biopsies; 2] To categorise the causes for crescents in these biopsies.

Material and Methods: Renal biopsies with crescents over a five year period were studied by light microscopy and direct immunofluorescence, wherever available and were correlated with relevant clinical and laboratory findings.

Results: The number of renal biopsies was 1629 and 158 (9.69%) showed crescents. Thirty four (2.08%) showed CGN. Twenty two percent were paediatric age group. The clinical presentation varied with common ones being nephritic (30%), acute renal failure (17.8%) and SLE (15.2%). Investigations recorded were proteinuria (99%), hematuria (74%), hypertension (63%) and increasing creatinine (89%). ANA positive in 28% and ANCA in 17.6%. The crescents were seen in the following: Lupus nephritis (LN)(29%), post infectious glomerulonephritis (PIGN)(18.58%), IgA nephropathy IgAN (17.3%), vasculitis (11%), membrano-proliferative glomerulonephritis (MPGN) (3.84%), Anti GBM antibody disease (2.5%), HSP nephritis (1.9%) and one case each of membranous glomerulonephritis (MGN), TMA and IgAN with MGN. In 11% the cause of crescents were undiagnosed due to lack of IF. The causes of CGN are: Anti GBM (75%), vasculitis (47%), PIGN (17%), LN (9%), IgAN (3.7%) and unclassified (41%).

Conclusion: Crescents in renal biopsies are rare findings. AntiGBM antibody disease and vasculitis are known to present with CGN, but other glomerulopathies like PIGN, LN and IgAN harbour crescents frequently, therefore mandating a careful scrutiny of the biopsy for crescents.
OR-15. IgA crescentic glomerulonephritis - Closer to ANCA positive or negative crescentic cohorts?

Authors: Debasis Gochhait, Ashwani Kumar, Suvaradip Mitra, Nandkrishna, Raja Ramachandran*, KL Gupta*, Ritambhra Nada.

Institution: Dept of Histopathology & *Nephrology, Post Graduate Institute of Medical Education and Research, Chandigarh.

Background: IgA nephropathy is the commonest cause of glomerulonephritis. However, Indian patients generally present with progressive disease and more often have crescents (50%) than reported from western literature (5%).

Aims & Objectives

• To classify crescentic IgA nephropathy using classification system for ANCA associated pauci-immune glomerulonephritis
• To compare morphology with ANCA positive and negative glomerulonephritis

Material & methods: 32 cases (23.8%) crescentic IgA nephropathy were retrieved from records of 134 cases of IgA nephropathy (5 years) and compared with ANCA positive (27) and negative (18) cases.

Results

Crescentic IgA had 68% cellular (n-22) and 32% mixed (n-10) category lesions which was different from ANCA positive and negative crescentic (85: 15% and 83:17%). Glomerular tuft necrosis was occasional (n-1) in crescentic IgA whereas it was seen in 48% and 38% cases of ANCA positive and negative crescentic respectively (p<0.05). Glomerulitis was present in all cases of crescentic IgA and also common in ANCA positive and negative crescentic (63% and 67% cases respectively). Severe Interstitial fibrosis and tubular atrophy was present in 4 case each of crescentic IgA and positive ANCA crescentic and 6 of negative ANCA crescentic. However, granulomatous glomerulitis (11%), vascuilitis (7%) and interstitial granulomas (15%) were present only in ANCA positive crescentic whereas tubulitis (25%) and thrombotic microangiopathy (19%) were present in crescentic IgA and not seen in other categories (p<0.05).

Conclusion

Crescentic IgA has closer morphologic similarities to ANCA negative pauci-immune glomerulonephritis than ANCA positive pauci-immune glomerulonephritis. TMA is more frequent in Crescentic IgA.
OR-16. How different are ANCA negative and positive pauci-immune crescentic glomerulonephritis: an experience at tertiary care hospital with a large ANCA negative cohort

Authors: Kiran K, Ashwani Kumar, Manish Rathee*, Shankar Naidu*, KL Gupta*, Ritambhara Nada.

Institution: Department of Histopathology and Nephrology*, PGIMER, Chandigarh

Background: Pauci-immune crescentic glomerulonephritis (PICrGN) is a common cause of rapidly progressive glomerulonephritis. In majority, PICrGN is a manifestation of antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis. However, our centre has documented 40% ANCA negative PICrGN.

Aims & Objectives:

1. Comparison of renal morphology of ANCA-negative PICrGN with those of ANCA-positive disease.
2. Identifying distinct morphological features present in the ANCA negative cohort

Material & methods: This was a retrospective analysis of diagnosed cases of PICrGN. Sixty three diagnosed cases of PICrGN between 2006-2012 were and compared for glomerular, tubulointerstitial and vascular pathology. Glomerular features included nature and number of crescents, tuft necrosis, glomerulitis, granulomatous glomerulitis and interstitial granulomas. Tubulointerstitial and vascular changes were graded as none, mild, moderate and severe.

Results: Among 63 patients with PICrGN, 39 (61.9%) were ANCA positive, 24 (38%) were ANCA negative. Compared with ANCA-positive patients, ANCA-negative patients were younger (mean- 43.6 versus 51.6 years). ANCA negative cases had less often focal disease (12%) which has better prognosis as compared ANCA positive (23%) cases. Whereas other categories were equally present. Cellular crescents were significantly more in ANCA-negative patients (mean-9) than in ANCA-positive patients (mean-6; \( P < 0.05 \)). Granulomatous glomerulitis and vasculitis were not seen ANCA-negative patients. \( P < 0.05 \). Chronic tissue damage with glomerulosclerosis and interstitial fibrosis was already present at diagnosis in ANCA-negative patients \( P < 0.05 \).

Conclusions: ANCA-negative PICrGN is not rare. Renal histopathologic changes are more like MPO-ANCA crescentic pauci-immune glomerulonephritis seen with microscopic polyangiitis in our cohort unlike similar histomorphology quoted in literature.
OR-17. Pauci-immune crescentic glomerulonephritis - a series of 21 cases.

Authors: Dr. Thingujam Bipin Singh MD

Institution: Babina Diagnostics, Manipur

Background

Pauci-immune crescentic glomerulonephritis is the most common cause of crescentic glomerulonephritis and is most prevalent in older patients with characteristic features of focal necrotizing and crescentic glomerulonephritis with little or no glomerular staining for Immunoglobulin by immunofluorescence microscopy examination. Most patients have antineutrophil cytoplasmic autoantibodies including patients with and without systemic vasculitis. 80%-85% of patients with active untreated pauci-immune crescentic glomerulonephritis have been reported to be ANCA-positive. Cases not associated with ANCA and do not have evidence for immune complexes on anti-GBM mediation is rare and has been known to account for no more than 5% of all crescentic glomerulonephritis.

Aims and objectives

To study the histological and laboratory features of pauci-immune crescentic glomerulonephritis.

Materials and methods

All pauci-immune crescentic glomerulonephritis cases diagnosed from January 2012 to May 2013, in Babina Diagnostics, were included in this retrospective study. The demographic and laboratory data were extracted for analysis. ANCA tests were performed by both IIF assay and antigen-specific ELISA. Renal specimens were evaluated using direct immunofluorescence and light microscopy as per protocol.

Results

Four of the twenty one cases (19%) of pauci-immune crescentic glomerulonephritis were ANCA negative. Patients with the negative ANCA had a lower percentage of normal glomeruli.

Conclusion

Among the patients with pauci-immune crescentic glomerulonephritis, ANCA-negative patients accounted for 19% of cases. Compared with ANCA-positive patients, ANCA-negative patients had a lower percentage of normal glomeruli and a higher level of proteinuria.
OR-18. Clinicopathologic spectrum of Renal Amyloidosis: 2 year hospital based study

Authors: Swati Sharma¹, Ranjini Kudva¹, Ravindra Prabhu²

Institute: ¹Department of Pathology and ²Nephrology, Kasturba Medical College, Manipal, Karnataka

Background: Amyloidosis is the term that defines a group of chronic infiltrative diseases characterized by beta pleated sheet configuration on X-ray diffraction examination and a fine, non branching fibril on electron microscopy. Histologically, amyloid appears as an amorphous, eosinophilic, hyaline and extracellular substance. The spectrum of renal symptoms and signs in amyloidosis is variable. Diagnosis of amyloidosis is confirmed by staining the section with Congo red and examining the stained section in polarized light.

Aims and Objectives: To study the spectrum of clinical and pathologic features in renal amyloidosis.

Materials and Methods: Renal amyloidosis(RA) cases were retrieved from pathology archivals for a period of two years.

Results: 6 cases of RA were identified out of a total of 60 renal biopsies performed during the study period. The age range of the cases was 28-56 years with a mean and median of 44.5 and 50 years respectively. The male to female ratio was 2:1. 5/6 cases presented with pedal edema. 1 case each was associated with history of hereditary sensory neuropathy, rheumatoid arthritis, pulmonary tuberculosis and hyperthyroidism. All cases had proteinuria and deranged renal function. Protein electrophoresis was done for all 6 cases. Paraffin sections stained with hemotoxylin and eosin, periodic acid-Schiff, trichrome, methenamine silver and Congo red were studied for pattern of amyloid deposition and other associated renal findings. Data pertaining to immunoflorescence pattern was taken. In follow up, 1/6 cases died due to renal failure.

Conclusion: Although renal amyloidosis has a low incidence in major studies done, it accounts for an important reason for dialysis and has high morbidity and mortality. As the treatment options expand, there is increasing need for early diagnosis.
OR-19. Renal Amyloidosis- An Indian Scenario

Authors: Thakral Divya, Batra Vineeta Vijay, Aggarwal Meetu

Institution: G.B. Pant Hospital, New Delhi

**Background:** Amyloidosis is a group of diseases in which proteins deposit as insoluble fibrils extracellularly in tissues. Renal disease is a frequent manifestation and often a major source of morbidity for individuals having systemic amyloidosis. In India, the incidence of secondary renal amyloidosis is much higher compared to the western world.

**Aims and Objectives:** To analyze cases of renal amyloidosis in a tertiary care centre and the causes.

**Materials and Methods:** A retrospective study carried out in the Department of Pathology, G B Pant Hospital, Delhi. A total of 110 cases were collected during the period from January 2008 to March 2014.

**Results:** Most patients with renal amyloidosis were in the age group of 31-40 years (26.4%) with male predominance (M:F 2.4:1). Patients presented with generalized anasarca. Out of the total 110 cases, 3 were of primary amyloidosis (2.7%) while rest were secondary in nature (97.3%). Majority gave history of secondary infection in the form of a recent tubercular infection (n=52). Other secondary causes included rheumatoid arthritis, bronchiectasis and COPD. On renal biopsy, the commonest site of amyloid deposition is peripheral capillary walls of the glomeruli. Amyloid deposition was also seen in the tubules, interstitium and blood vessels.

**Conclusion:** To conclude, secondary amyloidosis is much more common than primary amyloidosis in our country. The most likely commonest cause of renal amyloidosis in India is tuberculosis. Amyloid deposition causes renal damage due to severe protein loss and is difficult to treat; therefore accurate diagnosis of subtype of amyloidosis is extremely important.
OR-20. The Spectrum of Histology in Renal Biopsies Taken During Pregnancy & in the Post Partum Period

Authors: Sarika Karthik, Swarnalata Gowrishankar, Meenakshi Swain, Michelle De Padua

Institute: Department of Pathology, Apollo hospitals, Jubilee hills, Hyderabad.

Introduction: Pregnancy causes physiological changes in kidney function. A complicated pregnancy can exacerbate a preexisting pathological renal condition or a de novo renal disease can be observed during pregnancy.

Objective: To study the spectrum of pathological findings in renal biopsies done during pregnancy and in the immediate postpartum period, with a follow up of cases wherever possible.

Materials and Methods: 120 cases were identified in a five and a half period from Jan 2009 to June 2014. The clinical presentation and the renal biopsy findings in each of the above cases were reviewed and classified.

Results: Out of 120 cases, varied degrees of renal failure were noted in 85 cases. Nephrotic range proteinuria is seen in 26 cases, nephritic presentation is seen in 40 cases. 6 cases were evaluated for asymptomatic proteinuria. Histologically, cortical necrosis (40 cases) was the most common diagnosis. The next common diagnoses were acute tubular injury and acute tubulo-interstitial nephritis, each of them seen in 20 cases. A smaller number of primary and secondary glomerulopathies were encountered, including minimal change disease (8 cases), membranous nephropathy (5 cases), membrano-proliferative glomerulonephritis (4 cases), FSGS (2 cases), IgA nephropathy (7 cases), Thrombotic microangiopathy (3 cases), Lupus nephritis (4 cases) and a single case of chronic glomerulonephritis. Out of the 13 cases that were followed up, 7 progressed to end stage renal failure and 6 cases showed complete recovery. All these cases had variable histopathological findings. Remaining cases are being followed up.

Conclusion: Predominantly, the cases presented with acute kidney injury, having cortical necrosis as the most common histomorphological picture with a variable prevalence of de novo and preexisting glomerulopathies.
Abstracts: 7th International CME in Renal Pathology & Annual Conference of ISRTP

Aug 1-3, 2014, Mysore

OR-21. Acute renal failure following snake envenomation: The spectrum of morphological features on kidney biopsy

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Introduction: Acute renal failure following snake bites is common in South Asia but uncommon in rest of the world. Acute tubular necrosis and cortical necrosis are the common findings, with acute interstitial nephritis being rare. Prolonged renal failure with oligo-anuria is observed in patients with cortical necrosis or acute tubular necrosis.

Aims/Objectives: To study the patients with acute kidney injury following snake bite.

Materials and methods: 30 kidney biopsies of snake envenomation with acute renal failure were identified in a 5 year period from 2009 to 2013. The clinical and histological details retrieved from medical records. Follow up obtained from the concerned consultants.

Results: Proteinuria (1+) was seen in 12 cases, hematuria seen in 5 cases The Serum creatinine is elevated in all cases with the mean being 7.7 mg/dl. The most common histological findings on kidney biopsies were acute tubular necrosis (ATN) (50%), followed by patchy cortical necrosis (23%) of which three had associated features of a thrombotic microangiopathy (TMA). Acute tubulointerstitial nephritis (ATIN) was seen in 20%, and diffuse cortical necrosis in 6%. The follow up showed recovery of renal function in cases of ATN and ATIN as expected, and progression to dialysis dependent renal failure in the cases of cortical necrosis whose details of follow up were available.

Conclusion: Snake envenomation causes a spectrum of renal changes ranging from ATN, ATIN, cortical necrosis and TMA which are responsible for the renal failure. The renal biopsy is an important diagnostic tool for predicting renal function recovery.
OR-22. Renal cortical necrosis-Clinico-pathological analysis of a catastrophic entity

Authors: Pavneet Kaur Selhi, Vikram Narang, Harsimran Kaur, Harpreet Kaur, Neena Sood, Aminder Singh

Institution: Dayanand Medical College & Hospital, Ludhiana, Punjab, India

Background: Renal cortical necrosis (RCN) is a rare cause of acute renal failure secondary to ischemic necrosis of the renal cortex. The high incidence of RCN in developing countries, including India, is related mainly to obstetric complications. Non-obstetric causes include sepsis, snakebite, hemolytic uremic syndrome etc.

Aims & Objectives: To analyze the clinico-pathological profile of renal cortical necrosis in our center.

Material & Methods: All renal biopsy records of acute renal failure patients who were biopsied amid January 2012 to December 2013 at the Dayanand Medical College & Hospital, Ludhiana, India were reviewed to identify patients with histologically proven renal cortical necrosis. Clinical data was also reviewed for demographic profile, to identify the cause and outcome of renal cortical necrosis.

Results: In a retrospective analysis of 33 biopsied patients of ARF on dialysis; 03 were found to have acute renal cortical necrosis on histopathological evaluation. Patients presented with non-obstetric conditions including snakebite, hyper-acute renal allograft rejection and post-hysterectomy septic shock. Different clinical course and outcomes were observed in all three cases with spontaneous recovery of renal function in one patient after prolonged oliguria. The second patient eventually required chronic hemodialysis. The third patient of renal allograft transplant underwent graft nephrectomy followed by thrombosis, CVA, septicemia and succumbed to the illness with a fatal outcome.

Conclusions: Renal cortical necrosis is rare cause of acute renal failure. Early diagnosis by strong clinical suspicion and histopathological examination can reduce the high mortality rate of this catastrophic entity.

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Background: Thrombotic microangiopathy is associated with many disease entities like Microangiopathic haemolytic anemia, haemolytic uremic syndrome and various autoimmune disorders eg. SLE etc. The basic underlying mechanism includes endothelial injury and thrombus formation involving various organs in the body, the kidney being one of the most common, with glomerular / arteriolar involvement or both. The process can be both chronic and acute with varied clinical responses.

Aims & Objectives: 1) To analyse the renal morphological features of thrombotic microangiopathy. 2) To analyse the clinical and biochemical parameters of these cases.

Materials & Methods: A retrospective study was carried out on renal biopsy specimens from the archives of the department of pathology. 7 cases thus retrieved over a 6 year period, were reviewed for morphological and clinical features. Clinical diagnoses (1 each), included HUS, infection, hypertension, SLE, IgA nephropathy, postpartum renal failure and Burns. The glomerular and vascular characters were individually studied in each case.

Results: Of the 7 cases, 6 were a part of the clinical workup for renal failure, and one was post-mortem. The male to female ratio was 4:3. 5 cases showed only glomerular involvement; one showing chronic lesions, 4 acute and one both. Exclusive vascular involvement was noted in 2 cases and 1 showed both; all the 3 showed acute lesions, chronic changes were noted in one.

Conclusion: Thrombotic microangiopathy has protean manifestations, presenting as a precipitating factor for myriad disorders. Affecting glomerulus and vessels with acute and chronic lesions, its significance in the renal biopsy should not be underscored.
OR-24. Cytological diagnosis of subcutaneous fungal infection in renal transplant recipients

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Background: Renal transplant recipients (RTRs) are at increased risk of the development of a variety of skin infections that can result from graft-preserving immuno-suppressive therapy.

Aim: To determine cytomorphological findings of fungal subcutaneous swelling in RTRs

Material and method: A retrospective review of fine needle aspiration cytology (FNAC) smears of subcutaneous swelling with positive fungal elements in RTRs over last 2 years.

Results: Seven cases (all males; age range, 34-58 years, mean, 46.3 years) of positive fungal elements in RTRs over last 2 years were retrieved. The time interval between the renal transplantation and appearance of swelling ranged from 8 to 19 months. The swelling was located on lower limb (six cases) and arm (one case). The lesion was solitary (six cases) and multiple (one case). The cytology of aspirated material showed branched septate fungal hyphae in six cases. These were well delineated on Periodic acid schiffs (PAS) and chromic silver methenamine (CSM) stains. One case showed presence of faint, thin walled, broad ribbon like hyphae. Culture of aspirated material was performed in four cases which grew phaeohyphomycosis in all. Histology of excised tissue showed numerous septate, branched, pigmented fungal elements suggestive of pheohyphomycosis in four cases and broad ribbon hyphae suggestive of zygomycosis in one case. All of our cases responded well with anti-fungal treatment.

Conclusion: Fungal infection can manifest as subcutaneous swelling in RTRs. It is often severe, rapidly progressive and difficult to diagnose. FNAC is an important diagnostic tool which is simple, cost effective and rapid method.
OR-25. Detection of red blood cell dysmorphism in routine urine analysis using UF1000i (Sysmex)

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Institution: Anand Diagnostic Laboratory, Infantry Road, Bangalore.

Dysmorphic red blood cells are seen in glomerulonephritis and other renal disorders where the cells incur a mechanical damage in the tubules. These cells characteristically are acanthocytes (mickey mouse’s head shaped cells)

We subjected all routine urine analysis cases to chemical examination followed by analysis with UF1000i (Sysmex). This instrument has a specification for detection of RBC morphology which includes isomorphic and dysmorphic RBC's. As a part of our slide review criteria, the cases flagged for dysmorphic RBCs by this instrument were examined under phase contrast microscopy.

Our study includes a total of 297 routine urine analysis cases for which the UF1000i (Sysmex) flagged for dysmorphic RBCs. A microscopic examination under phase contrast was done for all of them. Thirty five cases out of 297 were positive for dysmorphic RBCs (12%). Only acanthocytes were considered as dysmorphic RBCs. Crenated RBC's and microcytes, target cells were excluded. Ten out of the 35 cases positive for dysmorphic RBCs showed albuminuria with chemical examination (28.6%).

The least number of RBC count for which dysmorphic cells were identified was 4 / hpf.

Sixteen cases out of the 35 cases showed nil RBCs on chemical analysis thus indicating a high sensitivity of RBC detection by UF1000i(Sysmex)

Detection of dysmorphic RBCs in routine urine examination cases plays a useful diagnostic tool in ‘renal hematuria’. Our study has shown that nearly 12% of patients for whom routine urine analysis flagged for dysmorphic RBCs showed "true" dysmorphism under phase contrast microscopy. Therefore UF1000i(Sysmex) complemented with phase contrast microscopy can be utilized in diagnosis of renal hematuria.

Authors: Dr. Hardeep Singh, Dr. Bhargav Reddy, Dr. Jaishree Ghanekar

Institute: Mahathma Gandhi Mission Hospital, Navi Mumbai.

Background: Hematological manifestations are one among the wide spectra of changes seen in CKD (Chronic kidney disease) patients. Anemia is a cardinal feature of chronic renal failure and is nearly universal in patients with End stage Kidney Disease (ESRD).

Aims and Objectives: To find the incidence and severity of anemia in CKD and ESRD; To compare the hematological changes in CKD and ESRD patients; The response to treatment of anemia in CKD and ESRD had been been studied.

Materials and methods: Data collected from 50 cases of CKD (Predialysis) and 50 cases of ESRD on Haemodialysis were discussed. Patients with age > 18 yrs and with a baseline Haemoglobin <10 gm/dl were included in the study. In all cases of CKD and ESRD investigations for assessment of renal function and Hematological changes was done. The study was done over 1.5 yrs at MGM Medical College Navi Mumbai.

Results: The incidence was seen higher in males and in the age group above 51 in CKD (54%) and ESRD (60%). 44% of CKD and 56% of ESRD had hemoglobin maximum in the range 7.1 – 9. 68% of CKD and 82% of ESRD had serum Serum ferritin above the normal range. 34% of CKD and 44% of ESRD showed an increase in Haemoglobin by 1 gm/dl following treatment for 4 weeks with Erythropoietin.

CONCLUSION: There is no difference in the hematological manifestations of CKD and ESRD. The cause of anemia in CKD and ESRD are multifactorial, hence inspite our treatment with erythropoietin and iron supplements the desired improvement could not be achieved.
OR-27. Granulomatous Tubulointerstitial Nephritis: a ten year experience from a tertiary care centre in North India

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Institutions: Departments of #Pathology, *Nephrology and §Clinical Immunology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India and $Department of Microbiology, Government Medical College, Chandigarh.

Background: Granulomatous tubulointerstitial nephritis (GIN) is usually due to infections, drug or sarcoidosis. However, the cause is often difficult to establish and the studies are limited.

Aims: The study was planned to study the clinicopathological features of GIN over a period of ten years at our Institute.

Patients and Methods: Renal biopsies diagnosed as GIN from Jan. 2004 to April 2014 in the Department of Pathology, SGPGIMS were retrieved. Clinical data was recorded. ZN stain for AFB was performed in all biopsies. Diagnosis of TB and sarcoidosis was suspected on clinical features. TB was established by demonstration of AFB or tubercular DNA.

Results: Seventeen patients were diagnosed as GIN [Mean age 35±15 years; Males 11]. Fourteen patients (10 TB; 3 sarcoid; 1 fungal) presented as CRF, two as nephrotic syndrome and one as post-partum acute renal failure (3 idiopathic). AFB was demonstrated in 1/10 and necrosis in 3/10 granuloma in TB. Blood IFN-Gamma assay, performed in 6/10 was positive in only one, while urine culture for AFB was negative in all patients. Three had tubercular lymphadenitis. Multiplex PCR for tubercular DNA, done in six biopsies diagnosed as TB, was positive in all six, while it was negative in a patient of sarcoidosis. Fungal GIN was due to Zygomycetes.

Conclusion: GIN is rare and TB is the commonest etiology (59%) in the tropics. Demonstration of AFB in renal biopsy, blood IFN-Gamma assay and urine culture are not sensitive for diagnosis of TB. Multiplex PCR for tubercular DNA is promising. Despite treatment, patients of TB and sarcoid remain dialysis dependent.
OR-28. The morphology of non neoplastic kidney disease in tumor nephrectomy specimen.

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Institution: Dept of Pathology, Kasturba Medical College, Manipal. Manipal University, Udupi district, Karnataka.

Background: Neoplastic changes are often noted in nephrectomy specimen removed for renal neoplasms. These lesions are often not suspected clinically. However, their presence, even in indolent form should be documented to ensure closer patient follow up.

Aims and Objectives: To identify spectrum of non neoplastic changes in renal parenchyma containing tumors & correlate the same with postoperative function data when available

Material and Methods: Parenchymal changes in 46 tumor nephrectomy specimen were studied. The findings were evaluated in the light of preoperative & follow up renal function data using H & E along with special stains(eg:PAS,PASM).

Results: Vascular changes with or without associated parenchymal scarring accounted for 46 & 12 percent of cases respectively. These changes were seen more often in patients with history of diabetes & hypertension. Scarred foci frequently showed ischemic glomerular obsolescence. Other findings included diabetic glomerulosclerosis, segmental glomerulosclerosis, juxta glomerular apparatus hyperplasia & fibrocellular crescent. Among diabetics, patients on regular treatment & good glycemic control did not have glomerulosclerosis. Further, amongst patients with follow up data, renal function deterioration was noted in those with significant parenchymal changes.

Conclusion: Non neoplastic renal lesions are common & often overlooked in tumor nephrectomy specimen. Equal importance should be given to both tumor classification & concomitantly present non neoplastic kidney disease. The presence of diabetic nephropathy may be first documented on the nephrectomy specimen. The identification of concomitant non neoplastic disease in the kidney can be important for subsequent optimal patient management.
OR-29. Role of Electron Microscopy in Renal Biopsy Evaluation: Experience from a Tertiary Care Centre in South India

Authors: Smita Mary Matthai¹ ³, Santosh Varughese², Gopal Basu², Anjali Mohapatra², Sueeena Alexander², Anna Valson², Shibu Jacob², Anna Pulimood¹ ³, Tamilarasi V², Anila Korula³

Institution: ¹Central Electron Microscopy Unit, Depts of ²Nephrology and ³Pathology, Christian Medical College, Vellore.

Abstract

Electron microscopy (EM) is routinely used for evaluation of renal biopsies in most centers in developed nations. However, in developing countries, a more selective approach to EM utilization is adopted due to resource constraints. There is no data from India on impact of EM in renal biopsy interpretation or its cost benefit relationship. This study aimed to assess the diagnostic contribution of EM in the evaluation of native kidney biopsies at a tertiary care center in South India. A total of 110 native renal biopsies were subjected to EM study, during the latter half of 2013. Of these, 101 met criterion for inclusion in this study, which were ≥ 5 glomeruli on light microscopy (LM), ≥ 2 glomeruli for immunofluorescence (IF) and ≥ 1 glomerulus for EM, excluding globally sclerosed glomeruli. A preliminary diagnosis was made on LM and IF studies, followed subsequently by EM examination. EM was essential for the primary diagnosis in 24 cases, resulting in a revision of preliminary diagnosis based on additional or unrelated ultrastructural findings. EM provided important confirmatory data for establishing the diagnosis and clearing diagnostic uncertainties in 27 cases. In 22 cases, EM provided helpful clinical information without altering the primary diagnosis. This study highlights the crucial role and continued importance of EM in renal biopsy evaluation, even in resource constrained settings. It is recommended that EM is incorporated into renal biopsy protocols or at least tissue be set aside for ultrastructural studies in all cases.

Key words: Electron microscopy, Renal biopsy, Ultrastructural study, native kidney biopsy, glomerular diseases.
OR-30. “Apolipoprotien-E expression in dense deposit disease, C3 Glomerulonephritis and Immune complex mediated Membranoproliferative Glomerulonephritis”

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Institution: Department of Histopathology¹, Department of Nephrology², PGIMER, Chandigarh, India.

Background

Mass spectroscopic studies of deposits in C3 glomerulopathy (C3GP) i.e. dense deposit disease (DDD) and C3 glomerulonephritis (C3GN) were similar in composition and different from immune complex mediated MPGN(IC-MPGN). Presence of alternate compliment pathway proteins and apolipoprotein E(Apo-E) is noted in significant amounts in all C3GP but not in IC-MPGN.

Aims & Objective

To study the localization of Apo-E in patients of C3GP and IC-MPGN by immunohistochemistry (IHC).

Material & Methods

IHC for Apo-E(ABCam) was done on paraffin sections of kidney biopsies diagnosed as DDD(n-13), C3GN(n-14) and IC-MPGN(n-76).

Results

In normal glomeruli, Apo-E stained mesangium(1+ intensity). Luminal aspects of capillaries of both the glomeruli and peritubular capillaries stained with 2+ intensity. Cytoplasmic staining (2+) in proximal convoluted tubules and no staining in distal tubules was noted. Expression of Apo-E was altered in all the groups when compared with control kidney biopsies. There was increased mesangial expression in cases with expanded mesangial matrix and especially with nodular pattern. Expression was significantly much more in sclerosed glomeruli especially in cases of DDD and C3GN. Loss of mesangial staining was more often seen in proliferative dominant patterns. Gain in expression around tubular basement membranes and loss of peritubular capillary staining was seen in all groups.

Conclusions

Apo-E expression is altered in all the studied groups. Mesangial Apo-E increases with expansion of mesangial matrix and lost with mesangial cell proliferation. It may be possible that glomeruli microdissected for proteomic analysis had glomeruli with mesangial matrix expansion and proliferation yielding differential results.
OR-31. Value of urine sediment scoring system in staging of acute kidney injury

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Institution: JSS Medical College, Constituent College of JSS University, Mysore

Background: Acute Kidney Injury (AKI) is diagnosed by a thorough clinical history, examination supported by certain laboratory test, that mainly includes serum creatinine and urine sediment analysis. According to Acute Kidney Injury Network (AKIN), AKI is defined as either a 0.3 mg/dl or 50% increase in serum creatinine concentration above baseline. A new urine sediment scoring system for AKI which is based on the number of Renal Tubular Epithelial Cells (RTEC) and the granular casts, has been derived. The prognosis and treatment differs based on the cause and stage of AKI hence it is essential to diagnose AKI at the earliest.

Aims and objectives:

To study the value of urine sediment scoring system in predicting the stage and prognosis of AKI.

Material and methods:

Study includes 50 patients with a clinical diagnosis of AKI. The urinary sediment were scored based on the number of granular casts and RTEC which were correlated with the clinical stage of AKI (AKIN classification).

Results:

The urine sediment combined scores were lowest in those with stage 1 AKI and it was found to be highest in patients with stage 3. The urinary scoring was seen to be significantly associated with increased risk of worsening AKI.

Conclusion:

The urinary sediment score is a useful cost effective tool to predict the stage and worsening of AKI.
OR-32. Proteinuria prevalence, clinical correlates and its association with Anti Retroviral Therapy in HIV positive patients

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Institutions: Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow and University College of Medical Sciences, Delhi

Background: Renal lesions constitute a considerable cause of morbidity in HIV-positive patients. Few urinary screening studies have been performed to determine the prevalence of proteinuria in HIV-infected ambulatory outpatients.

Aims: To assess the prevalence of proteinuria and microalbuminuria in asymptomatic HIV-positive patients. To study the association of urinary protein excretion with anti-retroviral therapy.

Methods: Patients were enrolled from among HIV-positive patients registered at the University College of Medical Sciences, Delhi. CD4 cell count estimation, urinalysis and renal function tests were performed. Protein and microalbumin estimations were done on spot urine samples. Sediment Morphology was assessed microscopically on centrifuged urine samples.

Results: 472 urine samples from asymptomatic HIV-positive patients were analyzed. Of these 459 patients were on ART. 9.1% urine samples tested positive for protein and 12.32% of the proteinuria negative samples tested positive for microalbumin. 88.4% of patients exhibiting proteinuria and 98% of those exhibiting microalbuminuria were on ART. Males and those with heterosexual acquisition of HIV showed higher prevalence of microalbuminuria. Mean CD4 count in microalbuminuria positive patients was 324. 56.6% of patients with microalbuminuria were in WHO stage 3. Three patients with overt proteinuria underwent kidney biopsies; these showed Collapsing glomerulopathy (HIVAN), Crescentic glomerulonephritis and acute and chronic tubulointerstitial injury.

Conclusion: The prevalence of asymptomatic proteinuria and microalbuminuria in HIV-positive patients is considerable. Our findings suggest that ART may be a significant contributor to the development of proteinuria/ microalbuminuria state. Detection of protein/ microalbumin in the urine in patients not on ART suggests that renal involvement probably occurs as part of natural course of the disease.