the ability for low viral load, untreated hepatitis B to cause proliferative glomerulonephritis (GN) or the significance of anti-Ro in absence of other biochemical, histological or clinical classical lupus manifestations.

**Methods:** We present a case of a sixty-three-year-old female who presented with acute on chronic renal dysfunction; she was treated naively for hepatitis B positive with low viral titre, anti-Ro antibody positive and had known IgM kappa monoclinal gammopathy with stable titres. Importantly cryoglobulins and the rest of her autoimmune panel was negative.

**Results:** Kidney biopsy light microscopy showed proliferative GN with membranoproliferative pattern. There was moderately severe arteriolar sclerosis and arteriolosclerosis with few vessels showing intimal fibrinoid necrosis with hyperplasia of tunica media (onion skinning). Six out of twenty glomeruli were obsolescent and there were two fibrocellular crescents. Immunofluorescence showed 4+ granular mesangial and peripheral capillary wall reaction with IgM and kappa, 3+ granular mesangial and peripheral capillary wall reaction noted with IgG, lambda and C3 only. Electron microscopy showed variable foot process effacement and a few cryoglobulin-like cylindrical organised deposits. The patient experienced improvement in renal function with mycophenolate and prednisone, however there was debate of the use of chemotherapy agents such as rituximab or traditional myeloma therapy. We are planning to do a repeat biopsy in the future to evaluate regression of renal lesions.

**Conclusions:** This case demonstrates the increasing evidence that many patients with proliferative glomerulonephritis do not fit neatly into one pathophysiological box and thus treatment must be tailored to the individual patient.

**MON-022**

**STEROID RESISTANCE IN CHILDHOOD NEPHROTIC SYNDROME IN MAPUTO, MOZAMBIQUE**

**KAKAR, S***1, Faktor, K*, Monteiro, Y*, Buck, WC*, Taunde, S*, Gonzaga, E*, Puliyananda, D**

1Hospital Central De Maputo, Pediatrics, Maputo, Mozambique, 2David Geffen School of Medicine, medical school, Los Angeles, USA, 3David Geffen School of Medicine, Pediatrics, los angeles, USA, 4Hospital Central De Maputo, Statistics, Maputo, Mozambique, 5Cedars-Sinai Medical Center, Pediatric Nephrology, los angeles, USA

**Introduction:** First line treatment for childhood nephrotic syndrome, characterized by proteinuria, hypoalbuminemia and edema, is corticosteroid therapy. An association between African ethnicity and steroid resistance has been reported, but published data from sub-Saharan Africa is limited, and no data available to date from Mozambique. The aim of this study was to determine the clinical profile, to assess the level of steroid resistance, and to determine the benefit of corticosteroid treatment in childhood nephrotic syndrome at Hospital Central De Maputo, Mozambique.

**Methods:** A Retrospective evaluation of patient charts and logbooks was performed to collect demographic and clinical data for a cohort of children (<15 years) diagnosed with nephrotic syndrome, between 2010 and 2015, and followed for at least 6 months, to better ascertain the disease burden and characterize the outcomes and clinical management of nephrotic syndrome in Mozambique. Patients were characterized as sensitive and resistant based upon response to corticosteroid therapy.

**Results:** Of 27 included patients, 17 (62.9%) were steroid sensitive and 10 (37%) were steroid resistant. 16(59.2%) were boys and 11 (40.7%) were girls, with age group of <5years corresponding to 12 (44.4%) and >5 years 15 (55.5%). Steroid resistance (60%) was noted higher in age group <5 years, as compared to 40% in >5years. No cases of plasmodium malariia were isolated in our series. HIV status was negative in 24 of these patients, and positive in one, with two patients who were not tested.

**Conclusions:** Incidence of corticosteroid resistance is higher than those reported in Asian or Western countries. Based on these findings, improved access to second line treatment is needed. Considering the care of children with nephrotic syndrome is greatly compromised by very limited resources and limited laboratory facilities, more than half of these patients may benefit from the judicious use of corticosteroids.

**MON-023**

**HYPOKALEMIC NEPHROPATHY IN NEPHROTIC SYNDROME PATIENT**


Centers Study in Bangladesh Cresecntic Gomerlonephritis, A Single Immunohistological Spectrum of Crescentic Glomerulonephritis, A Single Center Study in Bangladesh

**ISLAM, J***

1Armed Forces Institute of Pathology, Histopathology, Dhaka, Bangladesh

**Introduction:** Crescentic glomerulonephritis (GN) is defined as presence of crescents in >50% of glomeruli sampled for renal biopsy. This is a critical diagnosis with clinical implications that ideally is communicated to the referring physician immediately. There is limited data on the etiology, clinical and histopathological spectrum of crescentic glomerulonephritis (CrGN) in Bangladeshi population.

**Methods:** All the crescentic glomerulonephritis diagnosed histologically at Armed Forces Institute of Pathology, Dhaka irrespective of age and sex are included in the study. The study duration was from January 2017 to June 2018. The renal biopsy samples were undergone H&E, PAS, Masson trichrome and Methanamine silver staining. For each case direct immunofluorescence study was carried out for IgG, IgM, IgA, C3, C1q, Kappa and Lambda. Depending on clinical, biochemical, serological, histological and immunofluorescence evaluation crescentic glomerulonephritis was classified.

**Results:** During stipulated period 21 crescentic glomerulonephritis were diagnosed which comprises 2.56% out of total 819 non transplant renal core biopsy patients. Mean age was 27.238 years, 5years corresponding to 12 (44.4%) and 5 years, as compared to 40% in >5years in 21 cases 01 could not be classified due to lack of glomeruli during direct immunofluorescence (DIF) study. Pauciimmune crescentic glomerulonephritis (PI-CGN) was 10 (50%). Immune complex mediated crescentic glomerulonephritis (IC-CGN) was 9 (45%) and anti glomerular basement membrane disease (anti GBM disease) was 01(5%). Among the PI-CGN, 5 (50%) were ANCA positive and 03 (30%) were ANCA negative, for other 02 PI-CGN cases ANCA report was not available. Among ANCA positive PI-CGN 01 had associated ANA positivity. Among IC-CGN, 02 were crescentic IgA, 01 was crescentic lupus nephritis, 01 was isolated C3 glomerulonephritis, 03 were membranoproliferative glomerulonephritis with diffuse crescent formation and rest 02 were IC-CGN of unknown reason. The glomerular crescents formation was 80.33% in PI-CGN while it was 61.82% among IC-CGN cases and 66.67% in anti GBM glomerulonephritis. Among IC-CGN, 4/9 (44.4%) cases needed dialysis on admission while 5/10 (50%) PI-CGN cases were managed during admission by dialysis. The only anti GBM disease was tried to be managed by frequent dialysis but failed and plasmapheresis was advised but could not be instituted who ultimately succumbed to death following severe haemoptysis.

**Conclusions:** In our study commonest cause of crescentic glomerulonephritis was pauci-immune crescentic glomerulonephritis followed by immune complex mediated glomerulonephritis. While one case of anti GBM glomerulonephritis was found that was possibly a case of Good pasture syndrome.
Introduction: Hypokalemic nephropathy (HN) caused by prolonged K+ deficiency is associated with metabolic alkalosis, polydipsia, polyuria, growth retardation, hypertension, and progressive tubulointerstitial injury. Histopathologic report of HN in nephrotic syndrome (NS) patient is rare.

Methods: A 27-year-old male suffered from weakness, especially lower leg, since two weeks prior to admission. The patient had a history of NS since childhood and cared by nephrologist. Recent condition was hemodynamically stable, laboratory examinations showed potassium serum 1.85 mmol/L, albumin plasma 1.8 g/dL, serum creatinine 2.45 mg/dL, and marked dyslipidemia. The patient was clinically assessed with hypokalemia, relapse nephrotic syndrome, and renal insufficiency. Intravenous potassium was given and renal biopsy was performed. Histopathologic findings showed global sclerosis. The specific findings related to HN were mesangial proliferation and specific lesions on tubules including atrophy, vacuolization, intratubular deposition of amorphous and laminated hyaline materials and colloidisation of tubular epithelial and interstitial nephritis. The patient was discharged at 10th day of hospitalization with potassium level 2.93 mmol/L, and serum creatinine 1.95 mg/dL. The patient was treated with oral methylprednisolone, mycophenolate mofetil, ACE-inhibitor, simvastatin and oral potassium.

Results: Hypokalemic nephropathy may associate with chronic hypokalemia. Hypokalemia, especially if persistently occurs, can induce a variety of changes in renal tissue, impairing tubular transport and possibly inducing chronic tubulointerstitial disease and cyst formation. HN also associated with alterations in intrarenal vasoactive substances, leading to vasoconstriction, salt sensitivity, and progression of interstitial fibrosis. Histopathological changes in HN, including severe tubular dilatation, intratubular deposition of amorphous and laminated hyaline materials, intratubular cellular casts, and tubular atrophy is found. More attention is paid to atrophy and vacuolar changes in renal tubular epithelium accompanied by inflammatory interstitial changes in patients with potassium losses. One function that is not impaired is the ability to appropriately conserve potassium, which can be important in distinguishing between extrarenal and renal sources of potassium losses when the cause of hypokalemia is not clear.

Conclusions: A case with nephrotic syndrome associated with HN is reported. Chronic hypokalemia may involve in the development of hypokalemic nephropathy. Hallmark of histopathologic finding are tubular epithel atrophy, intratubular amorphous deposition and vacuolization of tubular cells, and interstitial nephritis to fibrosis.

MON-024

UNUSUAL PRESENTATION OF DENSE DEPOSIT DISEASE.

RASTOGI, P*1, Obediat, M1, Holanda G, D1
1University of Iowa, Pathology, Iowa City, USA

Introduction: Complement factor 3 glomerulopathy (C3G) results from the glomerular accumulation of C3 with no accompanying immunoglobulin. Patients with C3G may have acquired factors that cause C3 dysregulation through acquired pathway, e.g. C3 nephritic factors (C3NeF), anti-complement factor H autoantibodies and/or Complement factor B autoantibodies. Two morphological and clinically distinguishable subtypes have been described and include C3 glomerulonephritis (C3GN) and dense deposit disease (DDD).

Methods: The patient is a 17-year-old Caucasian male, who presented with a vague history of “Stomach virus”. This episode was followed by periorbital edema, which rapidly (Over the course of a