Introduction: Post-infectious acute glomerulonephritis (PI-AGN) is very common among Moroccan children. It can be defined as an acute, non-suppurative, diffuse, generalized inflammation of the glomeruli of both kidneys. It occurs after an infection of the skin or the upper respiratory tract. Group A beta-haemolytic (GABH) streptococcus is the most incriminated infectious agent. PI-AGN prognosis depends on the severity of renal involvement, with the possibility of chronic renal disease in the worst cases.

Methods: The aim of our study is to determine the epidemiological, clinical, histological, and evolutive features of PI-AGN in children treated in our center. For that reason, we gathered retrospective data of 83 children. The study took place from January 2016 to June 2020.

Results: The median of age was 9 years. Distribution by sex was in favor of males with 65%. Most cases (91%) were seen in winter. The most involved infectious sites were the upper respiratory tract (74%), and the skin (24%). The infection was still active at diagnosis in 30% of cases, while the remaining cases had it at least one week before. Microscopic hematuria and proteinuria were always present. Gross hematuria, generalized edema, and hypertension were also very common (seen respectively in 77%, 82%, and 65% of the cases). Other symptoms reported at diagnosis of PI-AGN were abdominal pain and vomiting (71%), convulsions (17%), and oliguria (13.3%). Acute kidney failure was found at onset in 25 children, from whom two had dialysis. The C3 complement fraction was low in 68 patients, and the ASLO titers were high in 53% of cases. All patients were under water and salt restriction. Furosemide was the first-line treatment of hypertension and oliguria patients, and nicardipine was added when blood pressure targets were not achieved. Renal biopsy was performed on 14 children. The main indications were nephrotic syndrome, persistent proteinuria, rapidly progressive glomerulonephritis (respectively in 22, eight, four, and two patients). The renal biopsy findings were as follows: endocapillary GN with C3 deposits in 14 children, minimal change disease in four children, membranous GN in one patient, crescent GN in two children, and glomerulosclerosis with C3 deposits in one child. Oral steroids (nephrosis regimen) were prescribed in 40%. Meanwhile, intravenous methylprednisolone (1g/1.73m² three days in a row) and cyclophosphamide (500 mg/m²/month for six months) was indicated in severe cases (respectively 23% and 9.6% of cases). All our 83 children had a good prognosis, and no cases of chronic kidney disease were noted at follow-up.

Conclusions: PI-AGN is still very common in Morocco, mainly because GABH-streptococcus upper respiratory tract infections are still frequent in our children. However, the prognosis remains good if the appropriate management is started early.

No conflict of interest

POS-158
D GALACTOSE IN STEROID RESISTANT NEPHROTIC SYNDROME- OUR EXPERIENCE

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Introduction: A proportion of patients with FSGS have a permeability factor which has not been well characterized but is known to be of molecular weight 30 to 50 Kilodaltons and is removed by plasmapheresis, which is often done in such patients who have proteinuria after a renal transplant. The permeability factor has been shown to bind to the glyocalyx in the vicinity of the podocyte. The glyocalyx is rich in galactose and exogenous galactose has been tried in such patients and the permeability factor has been presumed to bind to the galactose and to be taken up by the Kupffer cells of the liver removing it from circulation. D galactose has minimal toxicity (raised blood sugars and sometimes osmotic diarrhea) and is available as a powder. We have tried in some patients of steroid resistant minimal change disease on biopsy (unsampled FSGS presumed) and patients with primary FSGS and the clinical course of these patients will be outlined here.

Methods: 1. White caucasian female aged 38 with nephrotic syndrome and primary FSGS on biopsy with approx 30% glomerulosclerosis and moderate tubulointerstitial fibrosis, given D galactose 15 grams twice daily after failed trials with other drugs. All patients except number 5 were given ACE ARBs and bloods and urine were tested monthly for sugars, renal function, eGFR and urine ACR.

Results: 1. Patient number one has shown a response with some improvement in renal function and reduction of proteinuria (see chart). 2. No response in MCD but subsequently responded with long term remission with Orfatumumab. 3. No response in FSGS with end stage renal disease. Patients 4 and 5 are still being followed up.

POS-159
CLINICO PATHOLOGIC SPECTRUM OF ALTERNATE COMPLEMENT PATHWAY MEDIATED GLOMERULAR DISEASES – AN EXPERIENCE FROM A TERTIARY CARE CENTRE IN NORTH KERALA

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Introduction: Abnormal alternate pathway can result in C3 glomerulopathy (C3GN and DDD) and atypical HUS. Our attempt was to study the clinico pathologic spectrum of these diseases in our set of patients. Very few similar studies from our country are available in literature.

Objectives: (1) To study the clinical spectrum and renal pathologic findings of C3 glomerulopathy and aHUS (2) To study the factors associated with prognosis of the disease.

Methods: This was a prospective study conducted at department of nephrology, Govt Medical College Kozhikode, Kerala, India from December 2017 to December 2019. Patients meeting the inclusion
criteria were prospectively followed up for 6 months with relevant clinical data and laboratory investigations. Electron microscopy and genetic testing was done wherever feasible. At 6 months of follow up the outcomes were analyzed. Factors affecting disease progression were studied.

**Results:** We had 33 patients out of which 27 had C3GN and 6 had atypical HUS. Majority were in age group 31-40yrs. 57.6% were males. The most common clinical syndrome was acute nephritic illness (30.3%). Positive family history was present in 9.09%. 29 patients had hypertension at onset of disease. 27 patients had proteinuria > 1 g per day. Micro hematuria was present in 91%. Abnormal GFR at presentation was seen in 25 patients. The mean serum creatinine at onset was 3.8±2.76 mg/dl. 93.9% had low serum C3. The mean hemoglobin of 6.3±±0.63g/dl and mean platelet count of 0.56±0.30 lakhs/mm3 in aHUS group. The most common pattern on light microscopy was MPGN (8 patients). Crescents in biopsy was seen in 26.66% and 23.3% had severe IFTA. The most frequent deposits were subendothelial in electron microscopy. Genetic study was done for 13 patients and 10 of them (76.9%) had positive mutation. The most frequent mutations were in CFHR genes. 87.8% of patients (29/33) received steroids. 9 patients (27.27%) received MMF while 4 patients (12.12%) received CNI and 3 patients with Crescentic GN (9.09%) received cyclophosphamide. aHUS patients received therapeutic plasma exchange and 1 received only plasma infusion in addition to steroids. Most of the patients received at least 4-6 months of immunosuppression. 21% of patients had dialysis requiring renal failure at onset.

At 6 months 29 patients survived out of which 37.9% progressed to ESRD. 3 of them underwent transplant. 4 patients died during follow up. 44.4% of patients with no ESRD had persistent micro hematuria and hypertension. The total remission with steroids only was 71.4%, with steroids plus MMF was 66.6% and with steroids plus CNI was 75%. In atypical HUS only 40% achieved remission with TPE and immunosuppression. Multivariate regression analysis showed serum creatinine at diagnosis and IFTA on biopsy as predictors of disease progression.

**Conclusions:** Alternate complement pathway dysregulation in this study resulted in C3GN and atypical HUS. There was 33% progression to ESRD. We have limited access to complement therapeutics and hence less costlier but effective treatment strategies are definitely the need of the hour.

No conflict of interest

**POS-160**

**CLINICAL AND HISTOMORPHOLOGICAL SPECTRUM OF CRESCENTIC GLOMERULONEPHRITIS FROM SOUTHERN INDIA-A SINGLE CENTER EXPERIENCE**

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**Introduction:** Proliferative extracapillary (crescentic) glomerulonephritis on histology portends a poor clinical prognosis and is commonly associated with etiologies such as anti-glomerular basement membrane disease (anti-GBM), anti neutrophil cytoplasmic antibody (ANCA) vasculitis, Infection related glomerulonephritis (IRGN). There is a heterogeneity in etiologies reported from South Asia. This study evaluates clinical and histological spectrum of crescentic glomerulonephritis (CgN) presented to our institute.

**Methods:** We performed a retrospective observational cohort study of patients aged ≥14 who underwent renal biopsy at our institute from January 2014–June 2020 with evidence of crescents (involving ≥50%). Renal syndrome at presentation, clinical history, laboratory parameters, requirement of renal replacement therapy were noted. All biopsies included light microscopic, immunofluorescence examination and selected cases underwent electron microscopic examination. Biopsy samples with inconclusive diagnosis on histology were excluded. Patients were categorized into three etiological groups, group I (complex mediated), group II (anti GBM disease), and group III (Pauci-immune). Complex mediated group included infection related glomerulonephritis, C3 glomerulopathy, IgA nephropathy, and Lupus nephritis.

**Results:** A total of 2879 native kidney biopsies were performed during the study period. Ninety three (5.23%) patients had Crescentic glomerulonephritis of which group I had 50 (53.76%), group II had 16 (17.21%) and group III had 23 (24.78%) patients respectively (Figure 1). Four patients (4.30%) had a dual pathology anti-GBM with IgA nephropathy. Mean age of the study population was 42.23±16.63 years however pauciimmune CrGN was more prevalent in relatively older patients (46±17.32) years. Women predominated the study cohort 48 (51.63%) and similar distribution was noted across the groups I, II, and III respectively (49.6%, 53.3% and 57.2%). Two thirds of the patients 63 (66.7%) presented as rapidly progressive glomerulonephritis (RPGN), 28 (30.13%) as acute nephritic syndrome and two patients presented with only nephrotic range proteinuria. Hypertension was seen in 46 (60.21%) patients and 14 (15.02%) patients had associated Diabetes. Mean creatinine on presentation was 5.67±3.4mg/dl in immune complex GN, 8.45±3.94mg/dl in anti GBM disease and 6.34±2.82mg/dl in pauciimmune GN. Requirement of renal replacement therapy at presentation was highest in patients presenting with anti GBM disease followed by pauci-immune GN (93.3% &68.7%). Median number of glomeruli for evaluation were 11 (range 5–34). Morphology of crescents were cellular in 60 (64.51%) patients, fibro-cellular in 12 (12.93%), fibrous in 8 (8.6%), cellular to fibro-cellular in 13 (13.06%) patients. Viable glomeruli showed 100 percentage crescents of uniform morphology in 15 (93.7%) of patients with antiGBM disease. Glomerular tuft necrosis and vascular necrosis were noted in 18 (19.35%) and 14 (15.05%) patients respectively and were predominantly associated with anti GBM disease and pauciimmune glomerulonephritis. Four patients who underwent electron microscopic examination had features consistent with Immune complex glomerulonephritis.

![Figure 1. spectrum of crescentic glomerulonephritis](image)

**Conclusions:** Immune complex glomerulonephritis was the leading cause of Crescentic GN followed by pauciimmune GN in our cohort with female preponderance. Majority of the patients who presented as RPGN required renal replacement therapy.

No conflict of interest

**POS-161**

**A CASE OF EGPA THAT DEVELOPED DURING REMISSION OF IGA NEPHROPATHY**

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**Introduction:** Eosinophilic granulomatosis with polyangiitis (EGPA) is a systemic necrotizing vasculitis that affects small and medium sized vessels. EGPA is also characterized by allergic rhinitis, asthma, and prominent peripheral blood eosinophilia. EGPA is a rare disease, and its prevalence is about 11-13 cases per million. On the other hand, IgA nephropathy is the most common disease found to cause primary glomerulonephritis all over the world. There has been no case report of EGPA who had a history of IgA nephropathy.

**Methods:** We conducted a PubMed literature search using key words (eosinophilic granulomatosis with polyangiitis or EGPA or Churg-Strauss) and (IgA nephropathy), from 1986 to 2020. No case report was identified.

**Results:** A 54-year-old woman was diagnosed to have IgA nephropathy by a kidney biopsy when she was 25 years old and had 1.0 g/day of proteinuria. A remission was achieved using prednisolone, and the remission had been maintained under no immunosuppressant for 25 years. 7 years ago, she moved and follow up was started at our hospital. At the first visit, serum creatinine (scr) was 0.53 mg/dl. eGFR was 95 ml/min/1.73m2. MPO-ANCA was negative in the blood test. Urine