CNP1. Spectrum of Renal Injury in Pregnancy Induced Hypertension: South Indian Experience

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Introduction: Pregnancy Induced Hypertension is an important complication of late pregnancy and is an important cause of maternal and fetal morbidity and mortality. Data on clinical profile, especially renal profile of preeclampsia and eclampsia in Indian women is lacking

Materials and Methods: In this prospective, observational study 347 patients with diagnosis of preeclampsia-eclampsia, who were undergoing treatment at M.S. Ramaiah Medical College, were included in the study. The study duration was from 2010-2014. Details regarding epidemiologic data, obstetric data, laboratory parameters, maternal, renal and fetal outcomes was noted. Patients with pre-existing hypertension, diabetes mellitus or chronic kidney disease were excluded from analysis.

Results: Three hundred and forty seven patients satisfied the criteria for preeclampsia-eclampsia with overall incidence of 3.4%. HELLP syndrome was seen in 31 patients (9%). 56 patients (19%) had acute kidney injury (AKI) with mean serum creatinine of 3.2mg/dl with mean proteinuria of 2.8 gm/24 hours. 19 patients required dialysis. Persistent renal failure was seen in 2.5% of the cohort. Maternal mortality was 2.5%, largely secondary to sepsis. Primiparity was the major risk factor for PIH.

Conclusions: We report a low rate of preeclampsia in a low-moderate risk cohort with an incidence of AKI and maternal mortality consistent with reported literature.

CNP2. Unusual case of Polyuria - case report

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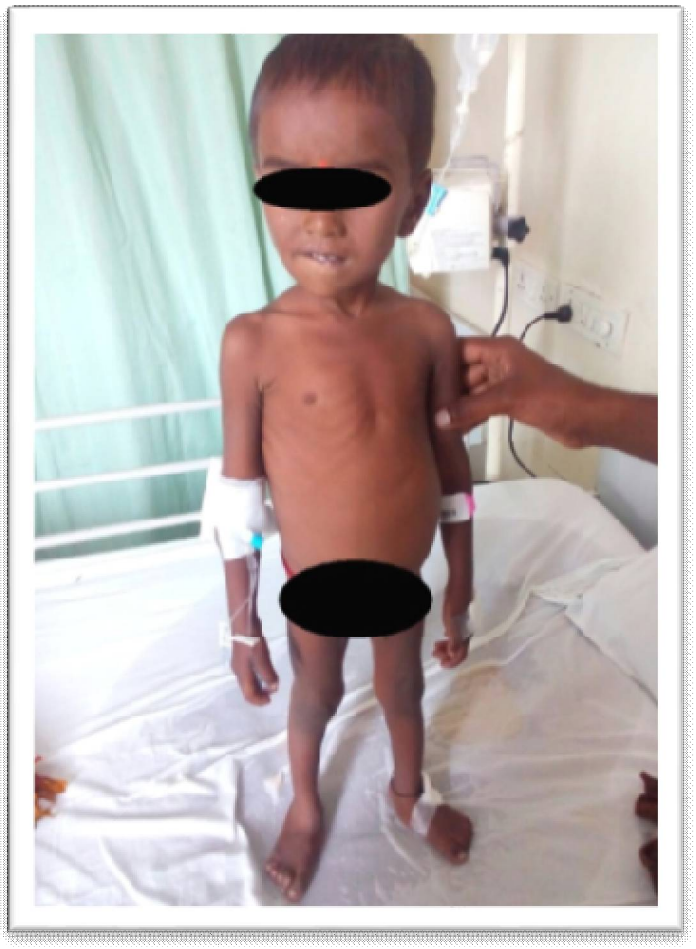
Introduction: A3 year old male child, born of consangious marriage. No co-morbidities. Apparently well till now, presented with polyuria (6 liters/d), polydipsia (4-5litres/d) and failure to thrive.Initially treated at local hospital with Tablet Desmopressin with provisional diagnosis of Diabetes Insipidus in view of polyuria. He later became unresponsiveness to Desmopressin. Referred here.Examination revealed pallor, severe dehydration, hypotension, lethargic with severe malnutrition. USG abdomen suggested no renal calculi or reflux disease.Serum osmolality was low (264 mosm/kg) associated with polyuria. Patient had persistent hypokalemia 1.8 meq/L and hypocalcemia 8.0 mg/dL and hypophosphatemia 1.8 mg/dL. Metabolic acidosis was present. Findings were against Diabetes Insipidus. Urine Potassium and Urine Bicarbonate levels could not be calculated because of very dilute urine.

Polyuria defined as increase in total daily urine output of urine >40 ml/kg/day or 2000ml/m2/day. Persistently low bicarbonates, metabolic acidosis and severe hypophosphatemia - suggestive of proximal acidosis. Persistent hypokalemia can cause urine concentrating defect causing polyuria.Achievement of milestones were suggesting acquired cause. Water deprivation test was difficult because of the irritable child and severe dehydration. In absence of glucose-induced osmotic diuresis in uncontrolled diabetes mellitus, most common causes of the polyuria are Primary Polydipsia, Nephrogenic Diabetes Insipidus and Central Diabetes Insipidus.

Persistently severe hypokalemia (plasma potassium concentration usually below 3 meq/L) can impair urinary concentrating ability. The distinction between pituitary and renal causes of polyuria may be made by the plasma ADH concentration and the urinary response to D-amino-arginine vasopressin. Child was started on hydration and potassium supplement (Potassium Citrate) in the dose of 1ml/kg/day in 3-4 divided dosages and sodium Bicarbonate replacement 5-10 meq/kg/day in 4 divided dosages. Over 2 months periodchild improved, urine quantity decreased, urine was concentrated. Now the serum and urine osmolality is normal and child is comfortable with appropriate weight gain.

Table 1 Laboratory findings

	1/5/16	13/8/16	17/8/16
Hemoglobin	8.3		
Serum calcium	6.82		8.0
S phosphorus			1.9
S sodium	138	127	132
S potassium	5.5	1.8	2.1
S bicarbonate		15.6	
urine Ph			6.2



Presentation of Child getting better with treatment

CNP3. Trail of diffuse proliferative glomerulonephritis

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Introdiction: This poster is presented to impress on the need to rigorously look for a focus of infection in all cases of infection related glomerulonephritis(IRGN).

Case Summary: A 22 year unmarried lady presented to our department with fever,edema and occasional haematuria of four weeks duration.Urine analysis revealed a nephritic sediment with protein creatinine ratio 2.8.She also had mild renal failure (Se.Cr -1.7).Complements levels were normal.She had no sites of active infection,sore throat,oral ulcers,hair loss,skin rash or photosensitivity.However her blood counts were elevated with neutrophilic leukocytosis on the peripheral smear.(TC-23,360 Neutrophils-82%).A renal biopsy was performed and it revealed endocapillary and mesangial proliferation in all glomeruli,dense inflammatory infiltrate within an edematous interstitium and IgG(+3),C3(+3) and C1q(+1) on immunoflorescence consistent with a diagnosis of diffuse proliferative glomerulonephritis.In the background of fever,elevated blood counts and DPGN without symptoms of autoimmunity, infection related glomerulonephritis was diagnosed but no focus could be identified.On repeated probing,the patient revealed irregular menstrual cycles and pain over the right breast with milky and foul smelling discharge.Breast examination showed a 4*4 cms abscess.Incision and drainage was done and antibiotics prescribed based on pus sensitivity reports.Her prolactin levels were checked as a further workup for irregular menstrual periods.Serum prolactin was markedly elevated.(460ng/ml).MR imaging of the brain revealed a pituitary microadenoma(3mm size).She was started on bromocriptine and had a complete recovery from all symptoms.

Conclusion: In this case we had a strong clinical diagnosis of infection causing glomerulonephritis and so we persevered with history taking inspite of normal complement levels.It is well documented that complements can be normal in the presence of visceral abscess as seen in our patient.Thankfully our efforts were rewarded and we were able to diagnose a microadenoma early and surprisingly use bromocriptine as a treatment for IRGN.

CNP4. Clinical And Laboratory Parameters Used In Decision Making For Renal Biopsy In Patients With Diabetes Mellitus.

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Introducction : To Critically Evaluate The Clinical And Laboratory Parameters Used In Decision Making For Renal Biopsy In Patients With Diabetes Mellitus, And To find the discriminatory value of these parameters.

Material and Methods: A prospective single centre study between March 2015 to March 2016.All Diabetic kidney disease patients who underwent a renal biopsy were included in this study. The clinical and investigation parameter(s) were noted in the file before the biopsy. The histopathology was categorized as 1.Non Diabetic Renal Disease(NDRD).2.Pure Diabetic Nephropathy(DN) and 3.Mixed (DN and NDRD).The demographic data was collected. Clinical parameters (presence of diabetic retinopathy and short duration of diabetes) and investigations (nephrotic proteinuria and hematuria) which are often used in the decision making for renal biopsy were noted. The collected data was analyzed to note the discriminatory value of these parameters in avoiding unnecessary biopsy i.e. histopathology result of category 2.

Results : Total of 62(male 54, Female 8) biopsies were done. The mean (sd) of the cohort was 52.3(10.1). The mean(sd) serum creatinine and 24 hour protein excretion was 4.0(3.28) mg /dl and 4.7(4.4) g/day. There were 25 (43.%) NDRD , 34(53%) DN and 3 (5%) mixed biopsies. The clinical parameter which had the highest discriminatory value was the absence of diabetic retinopathy ($p=0.003$)which predicted the presence of NDRD and mixed lesions in the biopsy. Neither nephrotic range proteinuria ($p=0.8$) and hematuria($p=0.08$) had any predictive value. Also the clinical presentation of RPRF negatively predicted the presence of NDRD in the biopsy.

Conclusion: The absence of diabetic retinopathy is still the strongest predictive parameter in the histopathologic diagnosis of nondiabetic renal disease.

Rapidly progressive real failure as a presentation did not predict a non diabetic renal disease in the biopsy.

CNP5. Renal doppler resistive index as a non invasive modality in predicting interstitial fibrosis in kidney biopsies

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Introduction: Renal biopsy is quintessential for diagnosis and in deciding therapy in renal diseases. In some of the cases renal biopsy is done exclusively for prognostication. It behooves us to avoid invasive procedures like kidney biopsy to identify these patients. Anecdotal reports suggest RI correlates well with interstitial fibrosis. We conducted a prospective blinded study to assess relation between RI and significant interstitial fibrosis, so a biopsy could be avoided altogether when planned for prognostic purposes.

Materials and Methods: In this study; 38 patients underwent Ultrasound color doppler testing just before renal biopsy and Resistive index were calculated. Clinical data and histopathological data were recorded and evaluated.Here we explored the relationship between pre-biopsy resistive index by renal Doppler and its ability to predict interstitial fibrosis on kidney biopsy. Statistical analysis was done using appropriate tests. ROC curves were plotted for mean RI in predicting moderate to severe interstitial fibrosis on renal biopsy.

Results: We observed inverse correlation between RI and eGFR (r_s = -0.79, p<0.01) and positive correlation between RI and interstitial atrophy (r_s =0.787; p value<0.05), and glomerulosclerosis (r_s =0.728; p value<0.05). ROC analysis showed the best cut-off RI value to predict moderate to severe interstitial fibrosis was 0.64 (area under the curve=0.90; 93.3% sensitivity and 87% specificity; Fig: 1).

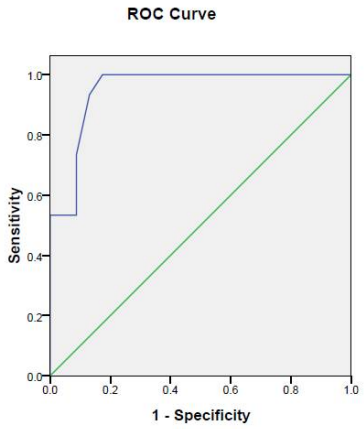

Fig. 1 ROC curve of predictive value.

Conclusions: Resistive Index, using doppler, is an excellent modality in predicting fibrosis in kidney biopsies. Considering cost effective and noninvasive nature of doppler studies, our study suggests that RI can be used in place of kidney biopsy, especially when done for prognostication purposes.

CNP6. A comparative study of effectiveness of enalapril,losartan and their combination in reduction of proteinuria among patients with type 2 diabetic nephropathy

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Introduction: To compare the effectiveness of Enalapil,Losartan and their combination in terms of achievement of 50% reduction in proteinuria among patients with type 2 diabetic nephropathy, and to compare their adverse reaction profile.
Materials and methods:62 patients were included in the study. Duration of study was one year. Inclusion criteria was newly diagnosed patients with type 2 diabetic nephropathy with proteinuria>500mg/day and S.Cr< 3mg/dl. Exclusion criteria included diastolic BP<80mm Hg, serum potassium > 5.5meq/L and bruit over arteries.30 patients were included in the enalapril group and 32 in the losartan group. Enalapril was started at 2.5mg/day and losartan at 25mg/day.Blood pressure,24 hour urine protein, S.Cr andS.Potassium were assessed every 2 months. If 50% reduction in proteinuria was not attained, dose was doubled every 2 months till a maximum dose of 40mg/day for enalapril and 100mg/day for losartan was achieved. Data was analysed using SPSS version 16.
Results:The median age group,sexratio,systolic and diastolic BP,FBS, baseline creatinine, potassium and 24 hour proteinuria, and comorbidities such as hypertension,dyslipidemia and coronary artery disease were compared between two groups.No statistical difference was noted (p value >0.05).At 6 months follow up 83.3% in enalapril group and 84.4% in losartan group achieved 50% reduction in proteinuria with no statistically significant difference between the 2 groups(p value 1.000).There was no significant difference between the two groups in frequency of total adverse events.
Conclusion:Enalapril and losartan were equally effective in reduction of proteinuria in type 2 diabetic nephropathy without significant difference in adverse reaction profile.

CNP7. Pattern of non-diabetic renal diseases [NDRD] in patients with diabetus mellitus at state run tertiary care center

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Introduction: A wide spectrum of non-diabetic nephropathy,including both glomerular and tubulo-interstitial lesions are reported in patients with Diabetes mellitus. Their precise diagnosis requires histological examination of kidney tissue. We carried out this study to find the clinical, laboratory, and pathological features of NDRD in DM patients. We also examined if any significant differences in clinical profile between the NDRD and DMN groups

Materials & methods::The demographic, clinical, and biochemical data of patients with DM (defined by ADA) who underwent renal biopsy in this institute for a duration of from 2012 august to september 2016 were analyzed prospectively. 280 patients were included in the study. Data were collected from inpatient file, monitor sheets, histopathological reports.

Discussion:In this study, incidence of NDRD was 38%, DMN 42% AND combined was 20%. CKD, NS and AKI were the most frequent clinical presentation. AIN, PIGN and CTID are commonest NDRD. These results suggest that prevalence of different category of biopsy-proven renal disease in diabetic patients depends on the usual prevalence of renal disease in the total population, according to the geographical area and ethnic characteristics and NDRD is merely a coincidental in DM. 58% of patients in the study had NDRD [either isolated or combined]. This study showed isolated NDRD in 38%, this result is similar to that reported in India and other regions with incidence of isolated NDRD were less than 50%[1,2]

Conclusion: Kidney biopsy is an important diagnostic tool to define underlying renal disease other than diabetic nephropathy in DM patients with prognostic value.

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CNP8.Evaluation of serum Lipoprotein (A) levels and novel lipid indices in patients with chronic kidney disease

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Introduction: Cardiovascular diseases are the most common cause of morbidity and mortality in non-dialytic CKD patients. In countries like India owing to the cost factors, measuring the lipoprotein levels are undermined. Studies have shown that novel lipid indices can be used to assess the CVD risks in CKD patients.

Aim & Objectives: To evaluate Serum Lipoprotein (a) levels and assess the significance of novel Lipid Indices in patients with chronic kidney disease.

1. To estimate fasting lipid profile in chronic kidney disease subjects and controls.
2. To calculate lipid indices: (Atherogenic index of plasma (AIP), Lipid tetrad index (LTI), Castelli risk index-I (CRI-I), Castelli risk index-II (CRI-II), Atherogenic co-efficient (AC).
3. To estimate serum lipoprotein (a) levels and correlate with novel lipid indices across all stages of CKD.

Materials & Methods: The study was conducted in Nephrology Clinic. 70 CKD patients from stage 1 to 5 were enrolled. Age and sex matched 70 healthy controls were studied. Patients with history of smoking, lipid lowering therapy, drugs causing dyslipidemia, and cardio vascular diseases were excluded. Serum urea, creatinine and the eGFR (MDRD formula), Serum total cholesterol, triglyceride, HDL and lipoprotein (a) levels were estimated. Serum LDL, non-HDL, VLDL and lipid indices were calculated from above.The results were compared by using excel software. Student unpaired “t” test, and Pearson coefficient of correlation were used for statistical analysis.

Results: Prevalence of diabetes and hypertension was 51.4%, 67.1% in CKD group and 17.1%, 21.4% in controls. Mean calculated eGFR was 34.72 ($\pm$16.7) ml/1.73m^2 and 105.8($\pm$17.08) ml/1.73m^2 in cases and controls respectively. Elevated triglyceride levels and lipoprotein levels noted in 74.3%, 48.6% of cases and 61.4%, 35.7% of controls. Statistically significant difference noted in triglyceride levels (227.41 vs 156.31), VLDL levels (45.48 vs 31.66) and lipoprotein (a) levels (53.26 vs 17.88) between cases and controls. But no significant difference noted in the total cholesterol, LDL, HDL levels.

Among the lipid indices, significant statistical difference was observed in Atherogenic Index of Plasma (AIP) (0.35 vs 0.21) and Lipid Tetrad Index (LTI) (73532.92 vs 12712.65) between cases and controls. No statistically significant difference observed in the other lipid indices. Significant positive Pearson correlation noted between the lipoprotein (a) and Atherogenic Index of Plasma (0.388) & Lipid Tetrad Index (0.799). While using lipid indices for risk stratification, early CKD stages (II, III) had highest CVD risk scores.

Discussion: There is significant lipoprotein (a) level difference noted between cases and controls, but poor correlation noted between lipoprotein (a) levels and eGFR. Lipid tetrad index had best correlation with lipoprotein (a) levels. Also LTI had highest positive and negative predictive

values for CVD risks (77.8% & 72.7) compared to AIP (65.3 &66.2). Other lipid indices are may not be appropriate for CAD risk assessment in CKD patients.

Conclusion: Lipid indices, especially atherogenic index of plasma and lipid tetrad index can be used to measure the burden of dyslipidemia especially when individual parameters are normal. Using lipid indices can complement to the routine lipid profiles to identify the individuals at high risk of CVD in early CKD.

CNP9. Acute pyelonephritis in type 2 diabetes mellitus – a single centre experience

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Introduction : Diabetes mellitus is a common predisposing factor for UTI. These patients are more prone for severe forms of UTI caused by resistant pathogens compared to the general population.

Aims : To analyze the clinical features, microbiological profile and treatment outcome of pyelonephritis in type 2 diabetic patients at our centre.

Materials& Methods: This was a hospital□based prospective study. Patients hospitalized from March to October 2016 with a diagnosis of Type 2 DM with suspected pyelonephritis were subjected to symptom analysis, appropriate laboratory investigations, imaging with ultrasonography and CT Abdomen. Results on continuous measurements are presented as Mean +/- SD and results on categorical measurements are presented in percentage. Chi-square test has been used to find the significance of study parameters on categorical scale between two groups. p <0.05 was considered significant. SPSS version 20.0 used for data analysis.

Results:

Parameter	**Emphysematous Pyelonephritis**	**Non Emphysematous Pyelonephritis**	**P**
N (Total 102)	21	81	
Age	57.0± 9.72	54.74± 11.16	
Gender Male **Female**	3 18	19 62	
Unilateral **Bilateral**	71.4% 28.6%	67.9% 32.1%	
Pyuria	19%	12.3%	< 0.05
Altered sensorium	14.3%	3.9%	NS
Shock	14.3%	2.5%	NS
Leukocytosis >11,000 cells/mm^3	90.5%	69.1%	<0.05
Serum creatinine **At admission (>1.5 mg/dl)** **Peak (> 1.5 mg/dl)**	 90.5 % 95.2 %	 53.1% 65.4%	 < 0.05 < 0.05

At discharge (>1.5)	61.9%	40.7%	NS
Need for RRT	14.3%	16%	NS
Recurrence	23.8%	17.3%	< 0.05
Mortality	9.5%	2.4%	NS

Urine culture was positive in 88.2 % and Gram negative bacilli were the most common organism detected**.** 17 patients with Stage I, II and IIIa EPN were managed with antibiotics with or without Percutaneous Cutaneous Drainage(PCD). 4 patients with Stage IIIb/IV patients were managed with antibiotics, PCD/ DJ stenting.Two expired due to sepsis.None required nephrectomy.

Conclusions:

1. A higher prevalence of culture positive Non emphysematous pyelonephritis noted among Diabetic females.
2. Gram negative bacilli were the most common organisms implicated.
3. Majority had reversible renal dysfunction.
4. Patients with emphysematous pyelonephritis had poor glycemic control, high recurrence rate and mortality.

CNP10. Clinico –biochemical microbiological profile of emphysematous pyelonephritis: a single centre study

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Introduction: EPN is a rare life-threatening necrotizing infection characterized by accumulation of gas in the renal parenchyma, perirenal tissues and/or collecting system. It's common in diabetics with mortality rate of up to 20%. Common organismsincludeEscherichia Coli, Klebsiella, Enterobacter, and Proteus spp.Management alternatives include medical treatment with or without DJ stenting.

Aims: To determine the clinical characteristics, imaging findings, microbiological patterns,primary and secondary outcomes of patients with EPN.

Materials and Methods:A retrospective study conducted at MS RamaiahHospital between Jan 2011 and May 2016. Patient's inclusion and exclusion criteria include;

INCLUSION CRITERIA	EXCLUSION CRITERIA
• All age groups • All patients proved as emphysematous pyelonephritis by ultra sound abdomen or CT abdomen	• Incomplete data • Mortality with suspected diagnosis before confirming the diagnosis • Early transfer to other centre after discontinuing treatment at the study centre

Clinical and laboratory data, imaging findings, microbiological patterns of 50 patients chosen for the study were recorded. The data was analysed to identify the prognostic variables that could predict the morbidity and mortality of patients with EPN. Primary end points were successful treatment and all-cause mortality. Secondary end points included need for Haemodialysis and specific treatment group

Results: Therewas equal incidenceamongst both sexes with median age of 59 years. CommonsymptomswereAbdominal Pain (93.18%), Fever (81.81%), Dysuria (75%), Vomiting (72.72%), Polyuria (77.27%), Oliguria (65.9%), and Breathlessness (68.18%). 97.72% (n=43) were diabetics.Commonest organismcultured was E.□coli (38.63%). 36.3% (n=16) required dialysis. Their mean age was60.25 +/- 11.74 years. Male sex, diabetes mellitus, shock, high serum creatinine at presentation and uremic symptoms showed no statistically significant association. 12.5% required haemodialysisindefinitely.

The antibiotic treated group had 100% success rate while DJ stenting group had 95.65% success rate (22/23). Early diagnosis and broad spectrum antibiotics along with appropriately timed intervention resulted in decreasedmortality.

Conclusion: Incidence of EPN was equal in both sex with no differences in clinical and laboratory parameters between dialysis and non-dialysis group. Pain abdomen and renal angle tenderness were commonest signs and symptom. E.coli was the commonest organism and early use of Broad spectrum antibioticsdecreased the mortality.

CNP11.IgA Nephropathy: Clinicopathological Correlates And Response To Immunosuppression

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Introduction: Prevalence, clinical course and outcomes of IGA nephropathy are highly variable with varied histology.Definite improvement in proteinuria after 6 month course of immunosuppression with steroid and MMF has been a major observation in previous studies in our institution. This study attempts to correlate clinical features and observed treatment response, to the histological characteristics.

Aim Of Study: To study the clinical characteristics, course of the disease and its histological correlates,

To assess the profile of immune deposits and their influence on clinical behavior and treatment response,

To assess the response to immunosuppression on follow up.

Materials and Methods: 104 patients attending nephrology OPD of our institution with biopsy proven IgAN were chosen. Their clinical characteristics, with their course on follow up, was noted in detail. Patients given steroid/ MMF immunosuppression were followed up. Further the clinical characteristics, follow up and response to treatment were assessed against their histological correlates and immune deposits

Results : Most common clinical presentation noticed was microscopic hematuria and hypertension; gross hematuria occured in younger age groups. T scores were significantly associated with hyperuricemia, hypertension, renal dysfunction and GFR deterioration. C3 and C1q deposits were associated with crescents.

74 patients in the study group received immunosuppression ,83 % having proteinuria >1 gm. Substantial reduction of proteinuria noted at 6 months(mean reduction 64%). Reccurence of proteinuria to more than 1g m/day was noticed in 20%.

Conclusions:E and T scores in biopsy has an influence on the clinical behavior, course and response to immunosuppression. Immune deposits have no correlation with clinical behavior or treatment response. Immunosuppression shows definite improvement in short term, but proteinuria relapsed in a subset of patients after withdrawal of therapy.

CNP12. Clinicopathologic profile of patients presenting with Acute Nephritic Syndrome

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Aims:To assess the clinicopathologic profile of patients presenting with acute nephritic syndrome.

Materials and methods:This is a prospective study to analyse the etiologic diagnosis of patients presenting with clinical diagnosis of acute nephritic syndrome. Renal biopsy specimen were analyzed.Baseline clinical and laboratory evaluation was noted down in a predesigned proforma.

Results: 31 patients were included in the study. 65% were males and 35% were females. The mean age was 38.8 years. MeanS.Cr of 4.8mg/dl. Serum C3 was low in 35% of cases. Of the total 31 cases 14 cases of IgA nephropathy, 8 were infection related glomerulonephritis, , 4 cases were lupus nephritis, 1 case each of ANCA vasculitis, anti GBM and HCV related MPGN and 2 biopsies showed sclerosed glomeruli.28 out of the total 31 patients (90.32%), presented with macroscopichematuria, and among these 13(46.4%) were later found to have IgA Nephropathy on renal biopsy,25% had IRGN, 10.71% LN. 21 patients (67.74%) had hypertension.Of these 11 (52.38%) had IgA Nephropathy while 5(28.81%)had IRGN. 12 out of 31 (38.70%) had Nephrotic range proteinuria and out of this 3 patients each (25%each) had evidence of IgA Nephropathy, IRGN and Lupus nephritis.Only one of four patients with lupus nephritis had extra renal manifestation.21 (67.74%) had renal failure, with 9 out of them i.e 42.85% showing features suggestive of IgA Nephropathy and 4(19.04%) showing features of IRGN. Among patients with macroscopic hemeturia and hypertension,39% had IgAN and 30% had IRGN.

Discussion:IgA nephropathy was the most common etiology in the study population. 21.4% of patients with IgAnephopathy had nephroticproteinuria. extrarenal manifestations were present in only 25% of lupus nephritis.

CNP13.Case Report of Chylous Ascites in Lupus Nephritis

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Aim: To report rare case of chylous ascites in patient with lupus nephritis.

Introduction: 20 yr female presented to nephrology opd with chief complaints of pedaledema, facialpuffiness, abdominal distension from 1month.H/o frothy urine present. H/o recurrent oral ulcers present.h/o primary amenorrhea present. No h/o hypertension,diabetesmellitus,hypothyroid.family h/o –nilsignificant. For the above complaints, pt got evaluated and found to have malnutrition with BMI- 15 Kg/m2 ,pallor,anasarca,all peripheral pulses normal,normal bloodpressure.CVS examination normal.respiratory examination showed diminished breath sounds bilaterally.abdomen examination showed massive ascites.no organomegaly.

Investigations:

CBP- Hb%- 7gm%,TLC- 4550, Plateletcount- 1.8 lakh/mm3
CUE- PH- 6,S.G-1008,Alb-3+,RBC- 4-5,Puscells- 5-6
LFT- SGOT-14,SGPT-10,ALP-110,TSP-3.8gm%,Alb- 1.8gm%
Urea-28mg%,s.creatinine-0.8mg%
Lipidprofile- total cholrsterol- 114mg%,HDL-30,LDL- 58,VLDL-16,TG-60mg%.
S.Calcium- 6.8mg/dl
24 hr urine proteins- 3.2gm/day
ANA,dsDANA-POSITIVE, C3,C4-LOW
ASCITICFLUID ANALYSIS- milky white,TC- 4, Protien- 2.3gm%,cholesterol-230,triglycerides- 350.

U/S abdomen- grossacites,hypoplastic uterus
Lymphatic scintigraphy –normal
Renal biopsy- class 3+4 lupus nephritis.

Based on above findings presence of chylous ascites in lupus nephritis confirmed.

Review of literature-Chylous ascites or chylothorax is characterized by a milky-appearing fluid containing high levels of triglycerides [1, 2]. The causes of chylous ascites or chylothorax can be categorized as nontraumatic and traumatic [1–3]. Chylous ascites is a rare form of ascites most often seen in women, especially those of child-bearing age, and usually in those of non-European descent. A triglyceride concentration >110 mg/dL supports the diagnosis; a level <50 mg/dL excludes a chylous effusion . During the course of SLE, a patient may develop inflammation of one or more serous membranes, resulting in pleural, peritoneal, or pericardial effusion. It is possible that inflammation of the lymphatic vessels and cisterns provokes an increase in their endoluminal pressure and permeability of the walls leading to extravasation of chyle.

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CNP14.To study the spectrum of biopsy proven renal disease (BPRD) and to look for any changing trends in renal diseases

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Introduction: Due to lack of renal biopsy registry there is lack of renal biopsy data and also not able to know the pattern of disease in India. This study analyzed the biopsy proven renal diseases (BPRD) and also compared it to pattern of disease in India and worldwide.

Materials and Methods - This is an observational retrospective cohort study done at Department of Nephrology Christian Medical College, Vellore. 1704 native kidney biopsies were included in the study excluding the transplant renal biopsies in 2 year period from January 2012 to august 2014.. This data was analyzed and compared with our previous published cohort (1986 and 2002) (n=5405), and with registries worldwide.

Results: The indications for renal biopsy included nephrotic syndrome (48.5%), nephritic syndrome (19%) and chronic kidney disease (10.9%). Primary glomerular disease accounted for 49.11% of all biopsies. Non-IgA mesangioproliferative glomerulonephritis as a group was the predominant pathology (24.80%), followed by IgA nephropathy (22.50%), membranous glomerulopathy (MN) (14.90%), idiopathic focal segmental glomerulosclerosis (FSGS) (9.31%), minimal change disease (MCD) (8.61%), and membranoproliferative glomerulonephritis (MPGN) (4.30%). Of the patients with secondary glomerular diseases, lupus nephritis (51.40%) and diabetic nephropathy (28.60%) were the most common. Among tubulointerstitial nephropathy (5.86%), the most common cause was chronic interstitial nephritis (59.1%) and among vascular nephropathies (9.35%), the common cause was benign nephrosclerosis (75%).

During the study period, there was a steady increase in IgA nephropathy prevalence and lupus nephritis in comparison to the previous reports from the same centre. A reduction in the frequency of MPGN and MCD was observed.

Conclusions: This study provides important descriptive epidemiological biopsy registry data and highlights some observed trends in changing prevalence of renal disease. This report represents the basis for the long-pending need of a prospective renal biopsy registry in India.

Keywords – renal biopsy, nephrotic syndrome, primary glomerulonephritis, non diabetic renal disease, indication for renal biopsy, biopsy registry.

CNP15.A Case of Proliferative Glomerulonephritis withmonoclonal IgG deposits(PGNMID)

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Introduction :A Case of Proliferative Glomerulonephritis with monoclonal IgG deposits butwithoutunderlying myeloma

Materials and methods: An adult female with unexplained nephrotic proteinuria with normal serum creatinineunderwent renal biopsy in august 2016 which showed Monoclonal immunoglobin mediated glomerulonephritis. Light microscopy showed diffuse global proliferation of endothelial, mesangial cells with capillary lumen obliteration, immunofluorescence showed IgG/Kappa chain linear 3+ deposits in capillary walls, interrupted linear c3 deposits, Electron microscopy showed extensive subendothelial and mesangial electron dense deposits.Myeloma workup was negative with only mild increase of serum kappa light chains. Oncologist opined lesser benefit on antimyeloma therapy considering cumulative drug side-effects. Hence empirically she was started on oral steroids(1mg/kg/d)

Results: There was no proteinuria remission even after 3months of steroid therapy, but proteinuria had increased. Serum creatinine was normal. Clinically stable. Option of using CNI was given, but patient lost for follow-up.

Conclusion:Because the majority of patients do not have M-spike or plasma cell dyscrasia, renal biopsy with careful attention to light-chain and IgG isotype staining is essentialfor diagnosis. Larger multicenter studies of PGNMID will be required to define the optimal therapeutic approach.

CNP16.Atypical presentation of kidney disease intype 2 diabetes mellitus; importance of renal biopsy

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Introduction: Diabetic nephropathy (DN) constitutes a majority of cases of kidney disease in diabetics, but Non diabetic renal disease (NDRD) is seen in a number of cases. Biopsy is performed if hematuria, absence of DR and proteinuria, rapid onset or progression of kidney disease are present. However, NDRD is often diagnosed during these biopsies. The clinicians need to consider renal biopsy in diabetics with atypical presentations as they have better response to therapy

Aims: To study the spectrum of NDRD in atypical presentations of renal disease in Type 2 diabetics. The clinical and pathological profile of NDRD would be documented and utility of renal biopsy assessed.

Materials And Methods:Type 2 diabetics with atypical presentation (Active urine sediment, rapid deterioration of S. Creatinine, rapid onset proteinuria, renal dysfunction without proteinuria) who were subjected to renal biopsy to rule out NDRD from January 2012 to June 2016 were included in the study. The patients with Type 1 DM, malignancy, immunologic disease, obstructive uropathy and infections were excluded from the study. Clinical details like age, sex, duration of diabetes, HTN, diabetic retinopathy, 24 hour urine for proteins and urine microscopy were noted for all the patients. The patients were grouped into three categories based on the biopsy results; Isolated NDRD, combination of NDRD and DN and isolated DN.

Results:Isolated NDRD was seen in 32.7%(n=40) , NDRD with DN in 11.4%(n=14) and isolated DN was seen in55.7%(n=68). NDRD was seen in total 54 patients(44.2%).
Mean age in 3 groups (Isolated NDRD, NDRD with DN and DN alone) was 47.2±10, 50.1±9 & 50.1±8yrs respectively.
Duration of DM was 3.2, 6.1 and 7yrs($p < 0.05$). S.Creatinine was (mg/dl) 4.9, 5.2 and 4.7 respectively.
24 hour urine proteins were .9g, 1.7g and 1.6g per day.
Active urine sediment was seen in 27.7% in patients with NDRD (n=15).
DR was present in 22.5% with isolated NDRD compared to 46.4% in DN.
The most common presentation in NDRD patients was AKI (33.3%) followed by NS (25.9%), RPRF(22.2%), AGN(7.5%), CKD and Asymptomatic urine sediment were seen in 3 cases each (5.5%).

The biopsy diagnoses were ATIN(20.3%), ATN(12.9%). PIGN and IgAN (9.2%),MN and CTIN (n=4, 7.4%). FSGS, Crescentic GN, CKD and MCD (3.7% each), cholesterol embolism and amyloidosis(1.8%).
Active urine sediment and duration of DM were better predictors of NDRD.

Conclusion : The overall incidence of NDRD) was 44.2%. The NDRD patients were younger than patients with DN and had a shorter duration of diabetes. The clinicians need to consider renal biopsy in diabetics with atypical presentations as they have better response to therapy .

CNP17. A comparative study of acute pyelonephritis in diabetics vs non-diabetics

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Introduction: This study was conducted to understand the clinical, biochemical and microbiological characteristics of patients with Acute Non-Emphysematous Pyelonephritis in diabetics vs non-diabetics.

Materials and Methods: It is a retrospective study conducted at M.S. Ramaiah hospitals from January 2012 to August 2016. The hospital medical records were searched electronically with the key words of Acute Pyelonephritis . Patients satisfying the inclusion – exclusion criteria were chosen for data analysisand categorized into diabetics and non diabetics.

Results:A total of 177 patients with diagnosis ofAcute Non Emphysematous Pyelonephritis were included in the study.96 being diabetic and 81 being non – diabetic.The mean age of patients in the diabetic group (51.9 years) was slightly higher than the non-diabetic patients (45.7 years). About 19.8% in diabetic group and 14.8% in the non-diabetic group were over 65 years. There was no sex preponderance noted in either group in our study (M:F=~1). Non Diabetic patients had longer duration of fever with chills (6.38 days mean in non-diabetics against 4.7 days mean in diabetics) at the time of admission.Recurrent UTI was also noted as one of the predisposing conditions for acute non-emphysematous pyelonephritis with 11.5% in diabetics against 5.8% in non-diabetics.The urine culture in the diabetics and non-diabetics showed positive growth in 53% and 43.2% cases respectively, with E.coli, ESBL and Klebsiella being commonly identified organisms. Blood culture specimens in both the groups had high negativity, 86.5% in diabetics and 98.8% in non-diabetics.Over 90% of patients in either group responded to treatment with either antibiotic alone or in combination with DJ stenting. Temporary hemodialysis was required in 8% in diabetic group and 6% in non-diabetic group. Maintenance hemodialysis was required in approximately 3% of diabetic patients.

Conclusion: Incidence of Acute Pyelonephritis was found to be higher in patients in their fifth decade of life in diabetics. The incidence among non-diabetics showed double peak with first one in second decade and the second peak in fifth decade. The incidence was found to be equal in males and females. Higher mean age, shock, depressed level of consciousness, higher serum creatinine both at the time of admission and discharge, elevated serum potassium and low serum sodium at admission were more commonly reported among the diabetics over non diabetics. E. coli and ESBL E. coli were the most common organisms identified in urine culture specimens of diabetics and non-diabetics respectively. Antibiotic treatment was the most effective way of treating patients in either cohort. DJ stenting in addition to antibiotics was required in significant proportion in either cohort. A close monitoring following initiation of treatment is required to consider additional interventions in non-responsive groups. The need for maintenance hemodialysis was more common in diabetic population.

CNP18.Polypharmacy, Medication adherence and Clinical Outcomes in patients with Diabetic Nephropathy attending Nephrology Department of A Tertiary Care Hospital

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Aim: To assess prevalence and patterns of polypharmacy, the factors affecting medication adherence and the clinical outcomes at 6 months in patients with diabetic nephropathy (DN).

Materials and Methods: We collected the data of 150 outpatients with DN from Nephrology department of St. John's Hospital, Bangalore prospectively and followed them up at 6 months. Data on disease characteristics and treatments were analyzed using Chi-squared tests and multivariate logistic regression.

Results: Mean age was 58.14 $\pm$10.44 years with a male preponderance (72.7 %). Hypertension was the commonest co-morbidity (98.7%). 64 % of patients were in DN stage 4 – 5. 58% of patients were undergoing hemodialysis.

Mean no. of drugs prescribed was 9.25 ± 2.5. Polypharmacy ($\geq$5 drugs) was noticed in 95.3% patients and high level polypharmacy ($\geq$10 drugs) in 46.7%. Presence of $\geq$ 3 co-morbidities was a significant predictor of high level polypharmacy (OR – 1.414: 95% C.I (1.008, 1.982), p = 0.045).

71% of patients were adherent to medications with no significant change in overall adherence pattern over 6 months. However, adherence to statins reduced from 63% at baseline to 39.1 % at 6 months. Major reasons for non adherence were medication cost (55%) and polypharmacy (36%). Higher stage of DN was a significant determinant of non adherence (OR = 4.726, 95% C.I (1.279 – 17.462), p = 0.020)

At the end of 6 months, 11 patients died. Other clinical outcomes were uremia (24.2%), hospitalization due to infection (14.8%), progression of DN stage (12.5%), initiation of dialysis (10.9%), heart failure (7.8%) coronary artery disease (5.5%) and others (15.7%).

Discussion: Clinical and demographic characteristics were similar to previous studies. Adherence to statins reduced over 6 months. Higher stage of DN was found to be a significant determinant of non adherence. About one third of patients developed clinical outcomes at the end of 6 months

CNP19 . Toxic epidermal necrolysis and acute kidney injury due to glyphosate ingestion

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Abstract: The literature, particularly from India is scarce on the renal effects of glyphosate poisoning.Glyphosate causes toxicity not only after its ingestion, but also after dermal exposure, by inhalation route and upon eye exposure. We present a patient report of glyphosate consumption which resulted in toxic epidermal necrolysis– the first report after glyphosate consumption and acute kidney injury.

Introduction : The literature, particularly from India is scarce on the renal effects of glyphosate poisoning. Commercial glyphosate-based formulations range from 41% or more concentration of glyphosate to 1% glyphosate formulations. The latter is marketed for domestic use. The formulations consist of an aqueous mixture of the isopropylamine (IPA) salt of glyphosate, a surfactant, and various minor components. Polyoxyethyleneamine is commonly used as a surfactant.[1]Glyphosate causes toxicity not only after its ingestion, but also after dermal exposure, by inhalation route and upon eye exposure. We present a patient report of glyphosate consumption which resulted in toxic epidermal necrolysis– the first report after glyphosate consumption and acute kidney injury.

Case report :A 30-year-old gentleman has consumed glyphosate (Hexagor 71%) with an intention to self-harm. He consumed 15mL.At a primary health centre gastric lavage was done withina hour. On the same day evening he developed body pains, oliguria and generalized erythema. The skin lesions later evolved into multiple discrete, closely set sterile pustules on flexures like elbows, axillae, and nape of neck. There was erythematous maculopapular rash on lower abdomen and upper thigh. The mucosae were normal. Within a day he developed anuria and pulmonary oedema. The skin lesions also progressed to widespread large bullous lesions on limbs and trunk. There were bleeding lip and oral ulcers, redness and watering of eyes and scrotal moist erosion. The Nicolsky sign was positive. At admission the blood pressure was 130/80 mm Hg. The patient had normotension during entire hospital stay. The investigation were, serum creatinine: 10.1 mg/dL, blood urea: 201 mg/dL, serum sodium: 135 mEq/L, serum potassium: 6.2 mEq/L, haemoglobin: 13.2 g/dL, total leucocyte count: 6800/mm^3 and platelet count: 60,000/mm^3, total bilirubin: 0.7 mg/dL, SGOT: 77 U/L, SGPT: 46 U/L, serum alkaline phosphatase: 75 U/L, serum creatinine phosphokinase: 215 IU/L, serum lactate dehydrogenase: 2579 U/L, serum cholinesterase: 1589 U/mL, serum pH: 7.2, serum bicarbonate 11.5 mmol/L and urine examination: albumin: 2+, pus cells: 1-2/hpf, red blood cells: 0-1/hpf and tubular cast

present. Ultrasound abdomen revealed, right kidney: 10.2 x 3.4 cm and left kidney: 10.4 x 3.2 cm. A 4 mm punch biopsy from right forearm revealed stratified squamous epithelium with spongiosis and exocytosis, vacuolar alterations of basal keratinocytes and there was moderate perivascular mononuclear infiltration in papillary dermis. It was consistent with toxic epidermonecrolysis. He was initiated haemodialysis and received eight sessions of haemodialysis before the urine output improved. He was discharged on day 24 after the consumption with normal serum creatinine and blood urea. At a follow up consultation after three months the serum creatinine and blood urea were 1.0 and 24 mg/dL respectively.

Conclusion : It is difficult to separate the toxicity of glyphosate from that of the formulation as awhole or to determine the contribution of surfactants to overall toxicity. Experimental studies suggest that thetoxicity of the surfactant, polyoxyethyleneamine (POEA), is greater than the toxicity of glyphosate alone andcommercial formulations alone. Ingestion of >85mL of the concentrated formulation is likely to cause significanttoxicity in adults.[1] In Lee series[2] the mean estimated fatal ingestion was330 mL, while survivors had a mean dose of 122 mL.

In plants, glyphosate disrupts the shikimic acid pathway. It results in deficiency of 5-enolpyruvylshikimate-3-phosphate production which leads to reductions in protein synthesis and plant growth and death of the plant occurs in 4-20 days.[3]The mechanism of toxicity of glyphosate in mammals is thought to be uncouplingof oxidative phosphorylation.[4]

Based on animal studies, it was found that only 30% is absorbed from gastrointestinal tract. The peak plasma concentrations of glyphosate are attained at1–2 hours.[5, 6, 7] The small intestine, colon, kidney and bone are thesites of distribution.[7]The major quantity of glyphosate is excreted unchanged in the urine.In two reports of human poisoning the peak plasma glyphosate concentration reached within 4 hours, the concentrations being almost undetectable by 12 hours.[8] The plasmaglyphosate concentrations 1000 mg/L[8]to 1600 mg/L have been encountered. [9]

Gastrointestinal corrosive effects, with mouth, throat and epigastric pain and dysphagia arecommon. Respiratory distress, impaired consciousness, pulmonary oedema, cardiogenic shock, arrhythmias might appear. Bradycardia and ventricular arrhythmias are often present pre-terminally.

Cardiovascular collapse is a major cause of death after glyphosate exposure,[2, 10, 11]and patients respond poorly to conventional fluid and vasopressor therapy.[12, 13]

Skin exposure resulted in several different manifestations. Skin contact with glyphosate had caused irritation[14]and contact dermatitis. [15]Chemical burns that later led to appearance of erythematous macules that developed into bullae within 24 hours was reported.[16] Facial swelling, paresthesiae andperiorbitaloedema and ageneralisedpompholyx were all reported.[17]Our patient had ingested the glyphosate. It is possible, ours is the first report of toxic epidermal necrolysis after consumption of glyphosate.

Renal and hepatic impairment are also frequent and usually reflect reduced organ perfusion, although a direct toxic effect of glyphosate or surfactant may contribute. Similarly, hypovolaemia, cardiogenic shock and/or acidosis may also give rise to acute kidney injury.

The precise contributions of both the surfactant and glyphosate to herbicide toxic effects in humans remain unknown.[18] The clinical features attributed to surfactant toxicity include vomiting, diarrhea, hemolysis, hypotension, alteredmental status, and pulmonary edema. [19]The clinical features like metabolic acidosis and CNS depression, nephrotoxicity, may be primarily attributed to the glyphosate itself.[20]

Management is symptomatic and supportive. Gastric lavage may be considered if a life-threatening amount of a concentrated glyphosate formulation has been ingested within 1 hour.Activated charcoalmay adsorb the surfactant component of glyphosate.[21, 22]Decontamination with activated charcoal should beundertaken in those with a protected airway and who present early. Use of activated charcoal in patientspresenting > 1 h after serious ingestion should be considered but remains controversial.[23]

Early invasivemonitoring, earlyventilatory and haemodynamic support and maintenanceof euvolaemia should all be instituted with alacrity. Hypotension secondary to fluid loss should be treatedappropriate use of crystalloids, colloids and blood products. Ionotropes may also be employed. Metabolic acidosis should be treated with quickly with sodium bicarbonate infusion.

Haemodialysis has not been preferred in patients of glyphosate intoxication for the reasonhaemodialysiscould not remove the surfactant, which may be partially responsible for the toxicity,[23]due to its large molecular size. However, haemodialysis does remove glyphosate. [24] Haemodialysis may improveacidosis and hyperkaelemia. The literature search revealed, that in Lee's series three patients were commenced on haemodialysis for acute kidney injury. All of these patients died.[2] In Stella's series,[24] both the patients initiated on renal support died. There were only a few published reports of successful haemodialysis in patients who ingested glyphosate. In one patient the daily haemodialysis was instituted for the treatment of pre-renal failure consequent to a massive loss of gastrointestinal fluid three days after ingestion.[20]In another report, two patients exhibited unresponsive hemodynamic states despite aggressive treatment. Bothpatients progressed to an anuric state. Haemodialysis was conducted due to hyperkalemia in one patient and due to metabolic acidosis in second patient. The authors could not entirely dismiss that the hypovolemiawas secondary to gastrointestinal fluid loss as the cause of their hypotension. [18]

Our patient had neither gastrointestinal cause nor hypotension due to any other reason to account for acute kidney injury. We did not perform a renal biopsy; for there was no indication. The timing of rise and fall of serum creatinine and presence of tubular cast points towards a possibility of acute tubular necrosis. Our patient recovered completely from acute kidney injury highlighting a possible role of haemodialysis in the management of glyphosate poisoning.

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CNP20.Epidemiological profile,clinical presentation& pathological profile of IgA Nephropathy.

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Introduction: IgA nephropathy is considered to be the most common form of primary glomerulonephritis in the world.Available evidence suggest an increasing incidence in India. At presentation IgA nephropathy can be of any known renal syndrome.
Aim : To assessthe epidemiological profile,clinicalpresentation andpathological profile of IgA nephropathy.
Materials And Methods : Patients admitted to the Institute of Nephrology ,MadrasMedical College, Chennai from January 2014 to June 2016 whose renal biopsy revealed IgA nephropathy were included in the study. History, clinical examination findings,laboratory investigations and renal biopsy were recorded.
Results : Out of 1075 native kidney biopsies performed during the study period,91 patients (8.5%) had IgA nephropathy. Of them 60 (65.9%) were males ,with male to female ratio of 2:1 .The mean age of patients was 34 years (ranging from 13 to 68 years). Majority of the patients were in the 20 – 29 years age group(31.8%) followed by 30 – 39 yrs age group (28.6%). Among these, 7(7.7%) patients presented with hematuria, 73 (80.2%)presented with edema ,24(26.4%) presented with oliguria and 55(60.4%) had hypertension.

On classifying the patients based on clinical syndromes ,69 (75.8%) presented with nephrotic syndrome, 21 (23.1%) presented with subnephrotic proteinuria,66 (72.5%) patients presented with renal failure. Of them 23(25.2%) had chronic kidney disease. Renal biopsy revealedadvancedIgA nephropathy in 23 patients. For remaining patients scoring was done based on Oxford MEST scoring system.Mesangialscoring M0 and M1was noted in 20 and 48 patients respectively.E0 in 45,E1 in 23 patients,S0 in 32,S1 in 36 patients ,T0 in 51,T1 in 15,T 2 in 2 patients were observed. Cellular crescents were noted in 16 patients (17.6%) , fibrocellular crescents present in 3 (3.3%). Renal biopsy showed isolated IgA deposits in 40 (43.9%) patients,IgA with C3 in 31 (34.1%), IgA with IgMand C3 in 8 (8.8%), IgA with IgG and C3 in 6 (6.6%), IgA with IgMin 1(1.09%), IgA with IgGin 1(1.09%) patient. The patient with IgA and IgGdeposits had linear IgGstaining consistent with anti GBM disease superimposed on IgA nephropathy.
Conclusion : IgA Nephropathy constituted8.5% of native kidney biopsies performed.
Majority of patients with IgA Nephropathy presented with renal failure.
Nephrotic syndrome was the commonest classical presentation (75.8%).

CNP21.Collapsing Glomerulopathy superimposed on diabetic nephropathy- a rare presentation

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Aim:Collapsing glomerulopathy represents severe podocyte injury with massive proteinuria, rapid progression and resistance to therapy.It is associated with multiple aetiologies. It's association with Diabetic nephropathy is rare. We present here a case of biopsy proven Diabetic nephropathy and collapsing glomerulopathy. A 44 year male, known diabetes for 15 years and hypertension for two months presented with increasing pedal edema for two weeks.

Material and methods: patient was evaluated. Renal biopsy done in view of recent onset of edema, hypertension, and massive proteinuria.

Results: Clinically blood pressure was 170/100mmhg, BMI-42, pedal edema. Fundus showed diabetic retinopathy. Haemoglobin-10.8g/dl. Bun-54mg/dl, serum creatinine-2.4mg/dl, urine showed RBC- nil, wbc-nil, protein-4+, 24 hr urine protein -12.5g. serum.albumin-3.8g, total cholesterol-170mg/dl, serum calcium-9mg/dl, phosphorus-3mg/dl, TSH-3.7. serology for HBsAg, HCV , HIV, and parvovirus were negative. serum ANA, ds DNA, ANCA-negative. Ultrasound showed kidneys of 10.5cm and 10cm size. ECHO was normal, EF-65%. Renal biopsy:Light Microscopy-7 glomerulus, of which 5 were obsolescent, 2 viable glomerulus showed mesangial nodular lesion (KW)of varying size surrounded by patent capillary loops. One of two glomerulus showed podocyte hyperplasia with collapse of underlying tuft . Tubules showed atrophic changes in approximately 40% with interstitial fibrosis and vessels showed features of both arteriosclerosis and arteriolosclerosis. IF showed 3 viable and 2 obsolete glomerulus which were negative for granular deposit for a panel of antisera(IgG, A, M, C3, C1q) and kappa, lambda chains showed no restriction. Overall features were of advanced Diabetic Nephropathy class 4 with focal segmental glomerulosclerosis, collapsing variant.

Conclusion: Patient is treated with antidiabetic and antihypertensives. Collapsing glomeruloapathy is a proliferative disease defined by segmental or global wrinkling of glomerular basement membrane associated with podocyte proliferation. These are poor responders to standard therapies. First described as idiopathic disorder or following HIV infection, it is now associated with a broad group of diseases and different pathogenic mechanism, which participate in podocye injury and mitogenic stimulation. Patients with both collapsing glomerulopathy and diabetic nephropathy have more severe disease and development of ESRD is more rapid than patients with diabetic nephropathy without collapsing glomerulopathy.

CNP22 A Rare but not a least diagnosis ofHemophagocytic syndrome-what Nephrologist need to know?

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Introduction : Hemophagocytic syndrome (HPS) is a rare condition which mainly involves as a part of hepatosplenomegaly renal involvement with high grade of fever. It is mainly due to dysregulated activation of immune system. We have diagnosed 7 cases of HPS from January 2015 to December 2016 in ICU admission. In this case series, we have described clinical features, treatment and outcomes of this diseases.

Materiasl and Methods We performed a retrospective study of all inpatients admission in ICU from January 2015 to December 2016 diagnosed with hemophagocytic lymphohistiocytosisHLHof various etiology.

Results : We have identified 7 cases of HLH on above mentioned time. Out of 4/7 were male (57.14%) & 3/7 were female (42.86%). Out of seven cases of HLH, cause beingviral (n=4,57.14%), bacterial (n=1,14.28%) and primary (n=2, 28.57%). There was no renal transplant case in present study. The most common HLH criteria were documented splenomegaly (n = 7) by ultrasound abdomen, fever (n = 7), ferritin >500 ng/dL (n = 9), triglyceride more than 300 mg/dl (n=5) and hypofibrinogenemia of less than 150 mg/dl (n=7), pancytopenia (n=7), bone marrow biopsy proven hemophagocytes (n=3). All seven cases were critically ill, multi organ dysfunction syndrome and various modalities of RRT(n=6). Majority of patients has received iv solumedrol (n=7) three doses andetoposide (n=2) after initial trial of diseases specific therapy. Overall 6 patients (85.71%) were died in hospital due to MODS being most common factor. Patient (n=2) who received etoposide, one was expired during hospital stay whereas another patient has received full course of etoposideas per HLH-94 treatment protocol. Till writing of this paper there was no relapse reported in one patient who has completed course of treatment.

Conclusions: HLH is lethal condition which affects adults and pediatric population. Clinical features such as splenomegaly, elevated ferritin, and cytopenia should prompt evaluation for HLH in this population.Survival benefit was seen in patient who doesn't requires RRT and early diagnosis. Despite considerable advances in the treatment of primary HLH, no specific therapy has demonstrated a consistent capacity to control reactive HLH when combined with suppression of the triggering factor with poor outcome.

CNP23.Clinical profile and outcomes of Crescentic IgA nephropathy- Experience from a tertiary care center

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Aim: To analyze the clinical features, histo-morphological parameters and outcomes of Crescentic IgA Nephropathy in adults in a tertiary care center in South India

Material and Methods: All Crescentic Ig A nephropathy cases (> 50% crescents) from Jun 2011 to Aug 2016 were selected from the database maintained in the department. All patients received protocol based immunosuppression. Biopsy specimen containing less than 5 glomeruli, and secondary IgAN (e.g., lupus, liver cirrhosis, or Henoch–Schonlein purpura) were excluded.The demographic characteristics, duration of symptoms, clinical features, and urine output, serum creatinine, proteinuria at presentationand histopathological details of crescentic Ig A nephropathy and follow up investigations were documented on a predefined proforma. Primary outcomes were defined as complete remission,partial remission,CKD,ESRD and death.

Results: 29 patients with crescentic IgA glomerulonephritis were included. Majorities were male-24 (82.75 %) and mean age was 31.42 ± 14.18 y. The median follow up period was 5 months(Range 3 to 93 months). All had hypertension at presentation and only 20.69% had oliguria (n=6). The mean 24 hr. urine protein was 1.82± 0.99 g/day;5(17.24%) patients presented with nephrotic range proteinuria. The median creatinine level was 8.3 (6.3-10) mg/dl and eGFR was 7 (6-11) ml/min/1.73m^2 BSA. 22(81.8%) patients presented with dialysis requiring renal failure . The mean percentage of crescents was 71±17%.Follow up was available for 27 patients . Total of 21 (72.4%) patients had adverse outcomes-ESRD (13) and Death (8). Only 5(17,2%) had partial remission, one remained in CKD III. Mean Renal survival time 12.88±9.59 months . The patients with adverse outcomes(death+ ESRD) had higher serum creatinine at admission, dialysis dependent renal failure, higher percentage of cellular crescents and tubular atrophy.Most of the deaths happened in the initial 6 months . Multivariate Cox regression revealed initial serum creatinine as the only independent risk factor for ESRD (hazard ratio [HR], 1.32; 95% confidence interval [CI], 1.10 to 1.57; P=0.002)

Discussion: Crescentic Ig A nephropathy was common in middle aged males, carrying a poor prognosis with majority reaching ESRD despite effective therapy. The serum creatinine at presentation is an independent predictor of adverse renal outcome.

CNP24.An unusual case of Renal Failure

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Aim: To study the clinical profile of a patient with Renal dysfunction with unusual presentation
Materials and methods: A 52 year old male – a social worker admitted with edema and decreased urine output. There was no fever. There was no feveror abdominal pain. There was history of joint pain for which one dose ofNSAIDwas taken. Patient needed three hemodialysis sittings since Renal function and urine output was deteriorating. Serum creatinine went up to 8.9. Renal biopsy was done after two hemodialysis in a view of suspected drug induced interstitial nephritis
Results: Renal biopsy was suggestive of GRANULOMATOUS interstitial nephritis. He needed totalthreehemodialysis sessions after which his Renal dysfunction started improving. His probable cause for renal dysfunction could be the use of NSAID which is one of the major cause for granulomatousinterstitial nephritis
Conclusion:: Major causes for granulomatous interstitial nephritis include infections like TB, sarcoidosis, drugs like NSAID....Athe probable causes were ruled out in this case to come to conclusion of NSAID induced granulomatous interstitial nephritis.

CNP25.Unusual presentations of Sickle cell anemia - 2 cases reports

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Aim: To study the clinical profile of patients of Sickle Cell Anemia presenting as AKI
Materials and Methods: Two patients of Sickle cell anemia are evaluated for their unusual presentation.
1)A 17 year old female patient with Chronic Kidney Disease presented with Seizures, Malignant Hypertension, found to have Posterior Reversible Encephalopathy Syndrome , subsequently developed Aphasia multiple cranial nerve palsy, CT Brain showed Cerebral Hemorrhage
2)A 35 year old male patient presented as Rapidly Progressive Renal failure , Renal Biopsy report is suggestive of Membranoproliferative Glomerulonephritis
Discussion: Renal disease associated with sickle cell disease includes gross hematuria, papillary necrosis, nephrotic syndrome, renal infarction, inability to concentrate urine, renal medullary carcinoma, and pyelonephritis.Microscopic or gross hematuria is likely the result of microinfarcts in the renal medulla. Glomerular lesions however, are less commonly encountered and may be seen in patients with HbSS, HbSC, and sickle cell-thalessemia.

CNP26.Spectrum of Renal manifestations in patients With Psoriasis

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Aim:To study the spectrum of renal manifestations in patients with psoriasis
Materialsand Methods:Patients with psoriasis and renal disease referred to Institute of Nephrology,Madras Medical College were included in this prospective study.The study period was from July 2015 to November 2016.
Results:Thirty seven patients were studied, of them 26 were men and 11 were women.Mean age was 49 years.Mean duration of psoriasis was 9.3 years and mean psoriasis area and severity index(PASI) was 4.9.Acute kidney injury[AKI] was seen in sixteen[43%] patients,chronic kidney disease[CKD] was seen in sixteen[43%]patients,nephrotic syndrome was seen in five[14%]patients.Seventeen[46%]patients had consumed alternative medicines and eight[22%]had taken analgesics.Fourteen[38%]patients were on methotrexate with a mean cumulative dose of 1918mg.Kidney biopsy findings:four were membranous nephropathy with negative for antibodies to M type phopholipase A_2 receptor and had consumed alternative medicines,one was unsampled FSGS,among two infection related glomerulonephritis[IRGN],one was IgA dominant IRGN,one was lupus nephritis,one was IgA nephropathy,one was thrompotic microangiopathy[TMA] and three were chronic interstitial nephritis.Out of sixteen AKI,five were analgesics related,three were alternative medicine related,three were due to hyperuricemia,two were IRGN,one was lupus nephritis,one was IgA nephropathy,one was TMA.

Conclusion:
AKI:Total sixteen[43%]
AKIN stage 1-five,stage 2-seven and stage 3-four
Outcome: Completerecovery-Ten
CKD4 - Two
CKD5D - Four
Membranous nephropathy:Total four
Outcome:Complete remission - Two
Partial remission - Two
Paper or poster presentation.

CNP27.Acute Kidney Injury (AKI) in Sick Neonates: Lessons from early screening and diagnostic classifications.

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Introduction : To study burden, associated risk factors and short term outcomes of AKI in sick neonates.To compare utility of modified KDIGO and nRIFLE classifications in early diagnosis of AKI in sick neonates.

Materials and Methods: This is a prospective longitudinal study including all sick neonates admitted to the NICU. All babies were screened for AKI by measuring urine output and serum creatinine on day of admission (beyond 48 hours of life)and once in 48 hrs till 1 week of age. Demography, clinical profile and course were noted. AKI was staged using modified KDIGO and nRIFLE classifications.Shortterm outcomes assessed were patient and renal recovery. Return of serum creatinine to normal,based on gestational age was defined as complete renal recovery.

Results: FromJanuary to October 2016, 163of 196sick neonates screened were recruited. AKI was diagnosed in 30.1 % (49/163) of which 46/49 were based on KDIGO and 24/49based on nRIFLE classifications.Therisk factors associated with AKI wereSGA (p <0.01), prematurity (p 0.04), shock (p <0.01), cardiac anomaly (p <0.01), use of NSAIDs (p 0.01), sepsis (p 0.08), dehydration (p 0.01) and presence of KUB anomaly (<0.01).Complete renal recovery was seen in 59% of babies. Among 71.5% of the neonatal AKI survivors, 14% had an abnormal creatinine on discharge.Urineoutput criteria alone, diagnosed 24 babies (49%) with nRIFLE compared to 10 babies (20.4%) with KDIGO.nRIFLEdetected more babies with early AKI. There was24.5% agreementbetween the two classifications.

Conclusion: Sick neonates need early screening for AKI with recognition of associated risk factors.KDIGO classificationis preferable to nRIFLE in diagnosing neonatal AKI, though nRIFLE can detect more babies with early AKI.Close monitoring of serum creatinine and a highurine output criteria could help in early diagnosis of AKI in sick neonates.

CNP28.Clinical spectrum of acute pyelonephritis in a tertiary care centre- a Retrospective study

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Aim : To analyse the clinical spectrum of Acute pyelonephritis
Materials and Methods: Retrospective analysis of Patient records with discharge diagnosis of Acute pyelonephritis with Ultrasonogram / CT scan to confirm the diagnosis
Period: 2014 to April 2016
The parameters : Hb, s.creatinine , electrolytes, Bl.sugar at admission, HbA1c, Urine analysis & culture, CT scan , procedures done.
Results:
A total of 101 discharges with diagnosis of acute pyelonephritis were found
94 had USG and CT scan reports
M:F 33:61
Age :27 – 76
ICU VS Ward : 11 : 83
Hemoglobin less than 10 was noted in 39/94
Hyponatremia noted in 70/94 patients
Pre-existing illness: CKD 10/94, Calculus disease 5/94, past h/o pyelonephritis 4/94
P.TB 1/94 and stricture urethra 1/94
Pregnancy in 1
Creatinine values at admission:
20/94 patients had S.creatinine <1
55/94 patients had Screatinine 1-4
19/94 patients had S.creatinine >4

Diabetes was noted in 81 out of 94
Duration of DM ranged from new-onset to 20yrs
HbA1c was available in 54 case records and the mean value was 10.1%

Right kidney, left kidney and bilateral involvement noted in 11:11:11 in males and 21:19:21 in females
Emphysematous pyelonephritis was diagnosed in
Bacterial Spectrum:
No growth: 33, Esch.coli 41, Candida in 7,klebsiella & enterococcus each 4 Pseudomonas 3 and mixed organisms in 2,

Procedures done:
Hemodialysis in 8
DJ stenting : 10
Percutaneous nephrostomy:6
DJ stenting+PCN :3
Conclusions: The prevalence of Acute pyelonephritis & Emphysematous pyelonephritis were increased in females compared to males (61/94 vs 33/94) &(10/61 vs 3/33)
*****Emphysematous pyelonephritis noted in 30% of females and 11% of males with pyelonephritis
*Bilateral involvement is highly prevalent in females compared to males (21/61 vs 11/33)
*Urologist consult is imperative in all cases of Acute pyelonephritis

CNP29.Renal Manifestations in patients with malignancy – An observational study

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Aim: To study renal manifestations requiring referral to Nephrology Department in patients with malignancy in a tertiary care hospital in Chennai.

Materials and methods: The data of patients referred to Nephrology Department of the hospital from the Oncology Departments (Medical, Surgical and Radiation), such as name, age, gender, co morbidities, primary malignancy and treatment given with purpose of referral were analyzed and the renal syndrome associated was observed.

Results: 74 patients were studied. There were 34 females among them. There was only one patient below 20 years of age, 5 patients between 20 to 40 years, 34 each in the age groups 40 to 60 years and more than 60 years. The primary malignancies were Gastrointestinal-8, Skin–5, Lymphoproliferative–2, Breast–7, Laryngopharyngeal–6, Pulmonary–4, Sarcoma-2, CNS–5, Ca Cervix–10, Ca Endometrium–4, ovarian Ca–4, Testicular–1, Phaeochromocytoma–1, Bladder–3, RCC–3, Maxilla–2, Myeloma-1, Hepatocellular Ca–2, CML-!, Thyroid–2, Prostate-1, TM Jt–1, Unknown primary–1. Two patients had more than one primary malignancy. The renal syndromes encountered were : Prerenal azotemia–23, AKI due to lymphomatous infiltration-1, Drug induced AKI–15, HT/AKI–1, Hypokalemia-2, Contrast induced Nephropathy–1, Sepsis / AKI–3, Obstructive nephropathy–9, Myeloma Kidney–1, RCC causing renal insufficiency–2 and 9 patients had Multifactorial AKI due to various combinations of Drugs, obstructive Nephropathy, contrast exposure and prerenal factors. Almost all the patients with mild renal failure recovered. Drug induced AKI was moderate in severity and recovered as the drugs were withheld. Only the very sick patients and those with sepsis had a poor prognosis.

Conclusion: Renal manifestations due to malignancy or treatment of malignancy carry a significant disease burden and underscores the need for the subspecialty OncoNephrology .

CNP30.Tuberculosis and glomerular disease. Are we underdiagnosing?

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Aim: To study the spectrum of glomerular involvement in patients diagnosed to have tuberculosis and glomerulonephritis

Material and method: Patients presenting to Nephrology department, Manipal Hospital, Bangalore during the 2 year period (January 2015 to December 2016) with symptoms of glomerular diseases and diagnosed to have tuberculosis were included. All underwent kidney biopsy.

Results:7 patients with tuberculosis and glomerular disease were included. All had tissue evidence of tuberculosis with sputum positive, granulomatous lesions in biopsy or gene Xpert positive. All were above 20 years. 3 were males and four were females.

3 biopsy showed IgA nephropathy, among which 2 had crescents with severe renal failure and 1 required renal replacement therapy. All 3 patients had negative ANA, ANCA, anti GBM and C3 normal.

2 patient had FSGS, one with collapsing type and other with NOS type with granulomatous interstitial nephritis. Patient with collapsing FSGS had lymph node tuberculosis, severe renal failure and was dialysis dependent. Her renal functions did not improve and she became ESRD.

6th patient was 21year old male with past history of treated tuberculosis with recurrent infective exacerbations and required renal replacement therapy. His renal biopsy showed AA amyloidosis with mild tubulointerstitial chronicity.

7th patient was 59 year old male who had lymph node tuberculosis. Biopsy revealed granulomatous interstial nephritis.

Conclusion: Tuberculosis can cause renal disease in a number of ways. The most common presentation is genitourinary tuberculosis via direct invasion by bacilli. However, glomerulonephritis associated with T.B. infection is very rare. We wanted to highlight the prevalence of glomerular lesions in patient with tuberculosis. Our case series consists of 7 patients with varied glomerular histology.

CNP31.Acute Kidney Injury Secondary to Acute Lymphoblastic Leukemia: A Case Report

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Introduction: Acute kidney injury associated with Acute lymphoblastic leukemia (ALL) has a variety of causes.Renal involvement in leukemia is not uncommon, the incidence of which is variable ranging from 4 to 47 %. This report describes a patient with ALL who developed acute kidney injury secondary to leukemic infiltrates and who has recovered partially after chemotherapy from renal perspective. This diagnosis should always be considered when a patient with ALL, regardless of the clinical stage, presents with renal insufficiency because it appears to respond reasonably well to a variety of therapies. We here describe a case of 5 years' boy who has presented to us with generalized weakness, fever, reduced urine output since 5 to 7 days. His Blood parameters were showing pancytopenia, high serum creatinine of 1.1 mg/dl (weight-20Kg) with features of Tumor lysis syndrome. His genetic analysis for Philadelphia chromosome negative. His USG was showing increased size of kidney and hepatosplenomegaly. He had receivedinduction chemotherapy with combinations of drugs including vincristine, prednisone, cyclophosphamide, doxorubicin, and L-asparaginase, which were given over 4 weeks.He has been started on alternative day of hemodialysis in view of worsening azotemia with hyperkalemia. After initial three weeks of regular alternativehemodialysis, renal biopsy was done with stabilization of blood parameters to rule out unexplained AKI. Renal biopsy histopathology which revealed diffuse infiltrates of lymphoid cells in interstitium which is characterized by round nuclei with coarse chromatin, indistinct nucleoli, & scanty cytoplasm. On Immunehistochemistry, these cells were positive for Tdt conforming the lymphoblastic nature of these cells. The glomeruli and blood vessels were unremarkable.In this paper, we describe the case of a patient with ALL who developed Acute Kidney Injury. Renal biopsy demonstrated a dense infiltration of leukemic cells.

CNP32.A Study Of Carcinomacervix Epidemiology And Its Contribution To The Ckd Burden In A Tertiary Care Center

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Introduction : Carcinoma Cervix is the second most common cancer affecting women and majority of these patients develop renal failure progressing to end stage renal disease .

Aim : To study the epidemiology , associated risk factors and its progression in patients who were diagnosed Carcinoma Cervix.

Materials and methods: Patients with Carcinoma Cervix(44 % biopsy proven) diagnosed to have renal failure and admitted in the institute of nephrology, Madras Medical College between August 2014 to October 2016 were prospectively studied. Course and severity of renal failure were studied. GFR was estimated using CKD EPI formula. Blood biochemistry ,hemogram and ultrasonogram were done .Details of treatment for renal failure – type and duration of dialysis, ureteric stenting , percutaneous nephrostomy were studied.

Results: Eighty four patients were included in the study and the mean period of follow-up was 21.16 months(1 – 144 months). Mean age of the study group was 52.46 years (35 – 85 years).Fourteen patients had Diabetes Mellitus at presentation, 4 were hypertensives and 4 had both. Two patients had established CKD at presentation.All womenbelonged to low socio economic strata and 62 patients (73.80 %) were multiparous. Thirty one patients had squamous cell carcinoma , 4 adenocarcinoma and 2 CIN . Thirty eight percent presented with Ca Cervix stage III, 16% with stage II and IV each and 3.5% with stage I disease.Thirty eight patients (45.23%) received radiotherapy , 10 patients (11.09%) received a combination of radio and chemotherapy and 12 (14.28%) underwent total abdominal hysterectomy with bilateral salpingo oophorectomy especially those who had stage II disease. Sixty six patients(78.57%) had renal failure dueto obstructive nephropathy while the cause of CKD was uncertain in other patients. Nineteen patients required percutaneous nephrostomy, 2 were unwilling for the procedure, DJ stenting was done in 6 and in 2patients attempted DJ stent placement failed. Thirty three patients(39.28%) required dialysis of whom 10(11.90%)underwent hemodialysis and 23(27.38%)underwent peritoneal dialysis.Sixty one (80.26%) patients were in CKD stage V, 14 (18.42 %) had CKD stage IV and 1 had CKD stage III. The mean time interval between the development of renal failure and the development of carcinoma cervix was 25.65 months (1month–154 months).Twenty six patients expired,46 patients lost follow up and 1 patient is on regular hemodialysis.Eight patients in CKD stage IV / V are on conservative management.

Conclusions: Eighty percent patients were in stageVCKD.Obstructive nephropathy was the cause of renal failure in 78.57 % of patients.Twenty six patients (30.95%) expired during the study period.Forty six patients(54.76%) were lost for follow up during the study period implying poor social support.

CNP33.Acute Kidney Injury following Consumption of Averrhoa Carambola

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Introduction:Averrhoa carambola , is popularly known as 'star fruit' in South India. It has been used in various forms like pickles, health drink, and curries. We have encountered a few cases which presented as AKI, in which there was history of use of this fruit. Aim To study the clinical profile and outcome of patients with AKI following consumption of star fruit.

Materials and Methods:It is a prospective observational study including 5 patients who presented between May 2015 to November 2016. Informed consent was obtained. Patients reporting with AKI as per RIFLE criteria were included. Detailed history regarding aetiological factors of AKI was obtained. Clinical profile and outcome were analysed.

Results:We had 5 patients who presented with AKI in whom there was a history of consumption of star fruit. The mode of consumption was as juice in 4 and as fresh fruit and pickle in 1 patient. The reason for use was for reducing cholesterol and weight. The initial complaints were vomiting and oliguria, during evaluation showed renal dysfunction. The lowest creatinine 3.5 and the highest was 7.2 mg. Immunological tests and ultrasound were normal. Since there was a clear history pointing to the aetiology and temporal association of events, renal biopsy was not done. Patients were treated with IV fluids, alkalinisation of urine and supportive measures. Average days for renal recovery was 10 to 14. None of our patients required dialytic support and creatinine improved to baseline at the time of discharge.

Discussion: Star fruit is a documented neuro and nephrotoxin in CKD. But it's a significant contributor for AKI in Southern states of India. Many a times ,this is overlooked as it has been used as a popular food since ages. Hyperoxaluria and resultant tubular obstruction has been depicted as the reason for AKI. Prompt diagnosis and treatment may aid the red the renal recovery.

CNP34.Experience of PRES in renal disease patients

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Introduction: The posterior reversible encephalopathy syndrome (PRES) is chracterized by headache, seizures, altered mental status and visual disturbances and typically causes the reversible changes in the posterior circulation system of the brain . The most common causes of PRES are hypertensive encephalopathy, eclampsia-preeclampsia, drug intoxications (immunosuppressive and cytotoxic drugs) and chronic renal failure (CRF) with hypertension, collagen vascular disease

Aim:we report 3 cases of renal failure who developed PRES and improved with treatment during their hospital course

Case presentations:case 1: A 16 yr old boy with a 2 yr history of SLE on treatment for extra renal lupus with oral steroids and on antihypertensivespresented with rapid progression of renal failure and treated with pulses of methylprednisolone for disease activity and Hemodialysis for renal failure.During the course patient had sudden onset gtcs with accelerated hypertension imaging showed characteristic pattern of PRES. Case 2 & 3 :2 patients diagnosed as CKD ESRD with Hypertension initiated on antihypertensives and hemodialysis developed seizures with accelerated hypertension and treated with antiepileptics and antihypertensives.symptomsresolved.MRI imaging showed characteristic features of PRES.

Discussion:PRES mostly is a benign and reversible condition, especially when the causative factor likehypertension can be eliminated. Although PRES can be diagnosed with MR-images, suspicion must be raised by the clinician. Both should be familiar with this underdiagnosed, clinically frightening syndrome to avoid persistent deficits.Because of multisystem involvement in SLE several associated causes may lead to PRES in SLE.PRES in the patients with CRF was known as uncommon condition,but needs high clinical suspicion.

CNP35. Takayasu Arteritis presenting as Nephrotic syndrome

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Introduction : TAKAYASU ARTERITIS is a non specificaortoarteritis predominantly affecting females in the age group of 10 to 40 yrs.The most common renl manifestation of takayasu arteritis is HYPERTENSION followed by glomerular involvement with mesangioproloferative glomerulonephritis being the most common followed by FSGS and AMYLOIDOSIS.

Case Presentation: 21/m presented with features of facial puffiness and swelling of legs and on clinical examination found to have edema with absent radial and brachial pulses with decreased intensity of carotid on left side. BP on RT upper limb was 100/60 mmhg. left side BP not recordable.no abdominal bruit.on evaluation found to have nephrotic syndrome,urine albumin 4 +,24hour urine protein was 4.5g/day with dyslipidemia.renal biopsy was done, it showed features of renal amyloidosis.

2D echo was done which showed dilated root of aorta with AR,neck vessel Doppler was done which was suggestive of takayasuarteritis.CT Angiogram of arch of aorta and abdominal aorta was done.other secondary causes were ruled out by doing bone marrow examination,serum protein electrophoresis,ANA,IgM RF,TUBERCULOSIS was ruled out with chest x-ray and mantoux test.A diagnosis of RENAL AMYLOIDOSIS secondary to TAKAYASU ARTERITIS is made.Initiated on empirical ATT and steroids 1mg/kg.Two months followup of patient showed no response.

Conclusion: Secondary renal amyloidosis is often due to chronic infective and inflammatory conditions,mostly tuberculosis in india,Though takayasu arteritis manifests mostly as hypertension .nephrotic syndrome is not uncommon though it is rare as presenting manifestation.hence we would like to emphasize that takayasu arteritis should be considered in the differential diagnosis of renal AMYLOIDOSIS and careful clinical examination of the patients can give clues to underlying etiology before proceeding to invasive procedure.

To be considered for FREE POSTER.

CNP36.Clinical profile and outcome of acutepyelonephritis - evaluation study in a Tertiary health care hospital

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Introduction: Pyelonephritis may be defined as an infection of renal parenchyma and collecting system. Acute non-obstructive pyelonephritis was previously thought to cause an Acute Kidney Injury occasionally. To our disbelief, several studies recently have demonstrated the presentation of acute pyelonephritis as acute kidney injury especially in diabetic patients

Aim: Study was intended to evaluate the clinical spectrum of acute pyelonephritis and its impact on renal function/ survival.

Material and methods: It is an analytical study enrolling patient admitted with pyelonephritis. The period of study was from January 2015 to October 2016. Various data including the demographic profile, the risk behaviour, clinical and laboratory profile, medical therapy, surgical intervention and renal replacement therapy were analysed.

Results: 121 patients with pyelonephritis were enrolled over a period of 22 months. Of them, sixty one (50%) were males. Mean age of presentation was 53.5yrs. Eighty two patients (67.7%) were diabetics. Twenty two (18%) had evidence of urinary tract obstruction. Fever was present in 85 patients (70.8%), dysuria in 69 patients (57.9%), loin pain in 60 patients (50%), and Frank pyuria in 13 patients (10%). Fifty two patients (42.9%) were in AKIN 3 at presentation. CT evidence of acute pyelonephritis was present in all the patients. Bilateral involvement was present in 55 patients (45%) and emphysematous pyelonephritis was seen in 11 patients (9%). Culture positivity in urine was present in 62 cases (51%). 18 patients underwent ureteric stenting (Bilateral DJ stenting in 7 patients).Fungal culture was positive in 6 patients. Escherichia coli (27%) was the commonest organism grown, followed by Klebsiella. 60 patients were supported with renal replacement therapy (56 with Hemodialysis and 4 with Peritoneal dialysis). Mortality was alarmingly high at around 11% (14patients expired during the followup).

Conclusion:

1) 82% had Non obstructive pyelonephritis
2) Need for renal replacement arose in almost 50% of the study population
3) 18 patients underwent ureteric stenting

CNP37.Collapsing Glomerulopathy and IgANephropathy: A Rare Association

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Aim : Collapsing glomerulopathy(CG) is a rare form of podocytopathycharacterized by segmental or global collapse of the glomerular tuft and marked hypertrophy and hyperplasia of podocytes. Prognosis is usually poor as, in spite of treatment, most cases develop end-stage renal disease(ESRD).

IgA nephropathy (IgAN), considered the most common cause of glomerulonephritis in the world, is characterized by predominant IgA deposition in the glomerular mesangium.It has ahighly variable presentation, both clinically and pathologically with some patients progressing to ESRD CG is associated with viral infections, autoimmune disease, and drugs. Theassociation of CG and IgAN is extremely rare with only one case series reported in literature. In this report, we describe two cases of CG superimposed on IgAN.

Material and methods : Clinicopathological features were retrospectively studied in 2 patients with biopsy proven IgAN and CG. Routine light microscopic stains, immunofluorescence studies for immunoglobulins, complement and light chains were evaluated. MEST score was assigned. Electron microscopic studies were performed on paraffin embedded tissue.

Results

Features	**Patient 1**	**Patient 2**
Age (years)	**42**	30
Gender	**Male**	**male**
Presentation	**nephrotic syndrome**	**nephrotic syndrome**
Hypertension	**No**	**yes**
Serum creatinine	**2.9**	**7.7**
Proteinuria	**3+**	**3+**
IF	**Ig A (+3), C3(+2)**	**Ig A (+3), IgM(+1),C3(+1)**
Light Microscopy		
Global glomerulosclerosis	**7/11**	**10/15**
IFTA	**50-60%**	**75-80%**
Arteriosclerosis	**Moderate**	**Moderate**

The patients presented with acute kidney injury and nephrotic syndrome

Conclusion : Massive proteinuria and rapidly progressive renal failure in IgAN may have renal injury not related only to the classic immune complex-mediated disease but as a result of a podocytopathy, such as CG.

The association between CG and IgAN should be recognized as it may have prognostic significance.

Kidney biopsy is useful for the definitive diagnosis and findings of prognostic value in these patients.

CNP38.Spectrum Of Renal Pathologies – A single center experience from South India

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Aims: To study the spectrum of renal pathologies among patients who underwent biopsy in a tertiary care center in south India.

Materials and methods: Renal pathological findings in patients who underwent biopsy from September 2013 to October 2016 were analysed.

Results:A total of 1570 renal biopsies performed during the study period(September 2013 to October 2016) were reviewed of which 164 allograft biopsies and 29 biopsies with insufficient tissue were excluded. Out of 1377 biopsies included, there were 747 males(54.24%) and 630 females(45.75%) with an age range of 10 to 85 years (mean 37.93 ± 15.15 years). This biopsy series comprised of 1030 glomerular(74.8%), 274 tubulointerstitial (19.89%), 67 vascular (4.88%) and 6combined pathologies(0.43%). Glomerular diseases included 73.58% primary (PGD) and 26.42% secondary glomerular diseases (SGD). Membranous nephropathy (MN - 17.95%) was the most common type of PGD, followed by minimal change disease (MCD - 17.16%), IgA nephropathy (IgAN – 15.19%) and focal segmental glomerulosclerosis (FSGS – 12.43%). Lupus nephritis (LN – 73.08%) was the most common SGD, followed by diabetic nephropathy (DN – 20.33%). In males, the most common PGD and SGD were MN and DN respectively. In females, the most common PGD and SGD were FSGS and LN respectively. MCD and LN were the most common PGD and SGD respectively in patients < 20 years of age. In the 20-39 years age group, IGAN (PGD) and DN (SGD) were the most common pathologies. In the 40-59 years age group, MN (PGD) and LN (SGD) were the most common pathologies. MN (PGD) and DN (SGD) were the most common pathologies among individuals ≥ 60 years.

Conclusion:MN was the most common PGD in this part of the country. The most common glomerular pathologies wereMCD (< 20 years), IgAN (20-39 years), MN (>40 years). The most common PGD was MN in males and FSGS in females while DN in males and LN in females were the most common SGD.

CNP39.Clinico Pathological Study of Nephrotic Syndrome in Adults

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Aim: Etiology of Nephrotic syndrome (NS) in adults and it's clinical outcome varies depending on age, sex, race, socioeconomic status and geographic location. The aim of present study is to analyze the histopathological spectrumin adults with Nephrotic Syndrome

Methods And Material: Patients in age group 18-60years with Nephrotic syndrome who were presented to Osmania general hospital and underwent a kidney biopsy from 2011 to 2016 were included in this study. All biopsies were subjected to light microscopy and Immunofluorescence.

Results: This study includes renal biopsy specimens from 250 cases of adult Nephrotic syndrome in years. Out of these 140(62%) patients were male and 110(38%) patients were female. The average age at presentation was 33+/-3 years. Among the patients, 140 cases(60%) were diagnosed with Primary Glomerular Disease (PGD) and 110(40%) with Secondary Glomerular Disease(SGD). The most common histological lesions were Membranous Nephropathy(20%) followed by Minimal Change Disease (MCD)(18%) . The most common form of SGD was Lupus Nephritis (LN) (19%), followed by DM. Among primary glomerulonephritis, Membranous nephropathy was the common lesion in males and FSGS in females. In age group less than 40 years , Minimal change disease was the common lesion and Membranous nephropathy was common among age group >40yrs. Lupus Nephritis was more prevalent among young adults with Nephrotic Syndrome with mild renal failure. Among 30 Diabetic patients, 9 were presented with non-diabetic kidney disease. Other glomerulonephritis included IgA Nephropathy , C3glomerulopathy , mesangioproleferative glomerulonephritis . One case each of C1Q nephropathy , Collagen Fibrotic Nephropathy , Amyloidosis were seen .

Conclusion : Overall Membranous nephropathy is the most common cause of Nephrotic syndrome in adults in South India whereas FSGS is common lesion in north India. Lupus nephritis is common Secondary Glomerular Disease among adults with Nephrotic Syndrome and mild Renal dysfunction, its prevalence is high in south india compared to other parts of our country.

CNP40.Clinico Pathological study of rapidly progressive renal failure (RPRF) in Adults.

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Aim : The aim of present study is to analyse the various clinical parameters and histopathological spectrum of rapidly progressive renal failure in adults.

Materials and Methods : Patients (>18 years old) with RPRF presented to Osmania general hospital and underwent a kidney biopsy from 2011 to 2016 were included in this study. All biopsies were subjected to light microscopy and immunofluorescence.

Results: This study includes renal biopsy specimens from 135 cases of adult RPRF. Out of which 87 (64%) patients were male and 48 (36%) were female. The average age at presentation was 38 +/- 3 years. The clinical features among patients were rash (8%), anemia (16%), Diabetes (2%), Hypertension (34%), oliguria (12%), proteinuria (24%). Among the patients 89 (66%) were presented with renal failure and 46(34%) with systemic disease. The most common histological lesions among isolated renal failure were Acute tubulointerstitial nephritis (25%) followed by Ig A nephropathy (19%). Among patients with systemic disease the most common histological lesions were post infectious glomerulonephritis (24%) and benign nephrosclerosis (17%). The other causes of RPRF includes thrombotic microangiopathy (10%), vasculitis (9%), lupus nephritis (8%) and one case each of granulomatous nephritis, anti GBM disease and myeloma cast nephropathy.

Conclusion : Early definitive diagnosis of RPRF by renal biopsy, serology is essential to reverse the otherwise relentless progression to ESRRD.

Discussion : Rapidly progressive renal failure is an initial clinical diagnosis in patients who present with progressive renal impairment of short duration from few days to three months. The etiology may be a primary renal or systemic disorders. Good history taking, clinical examination and investigations like serology and kidney biopsy are helpful in clinching the diagnosis. The important differential diagnosis includes Vasculitis, SLE, Myeloma, TMA and Acute interstitial nephritis.

CNP41.A case report of IgG4 related renal disease

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A 64-year-old male presented to us with history of vague generalized compliants of weakness, loss of appetite for past 3 months. He also had history of fever on and off for last 2 weeks. Past histroy he had abdominal pain on & off since last 6 months. He had past history of recurrent pain abdoment in the past for which he had been evaluated outside and found to have Fibrocalcific pancreatitis. On investigation he was found to have to have deranged renal functions(urea-94mg, creatinine-4mg) and bilateral normal sized kidneys. Urine analysis showed bland urine sediment and spot urine protien/creatinine ratio-0.6. Complement 3(C3) 56.6mg/dl&Complement(C4) 7.12mg/dl were low. His ANA was weakly positive. In view of normal sized kidneys and unexplained renal failure in an elderly male he underwent kidney biopsy. Kidney biopsy showed IgG4 related chronic tubulointerstitial nephritis. Immunohistochemistry of renal biopsy showed IgG4 -plasma cells positive staining and more than 10% of plasma cells were noted. His serum IgG level were 4280mg/dl and IgG4 sub class level were 19.8g/L which was quite high. CT plain abdomen was done which showed atropic pancreas. He was started on prednisalone 60mg per day and he came for follow-up after 3 months his renal functions had improved marginally and his serum creatinine was 2.9mg/dl.

Here we present a unusal case of IgG4 related renal disease with fibrocalcific pancreatitis with no other system involment. High degree is suspesion this entity to be kept in mind in evaluating renal failure of unknow etilogy with multisystem inlovement.
IgG4-related disease (IgG4-RD) is relatively a new growing entity of immune-mediated origin, characterized by a mass-forming lesion, the infiltration of IgG4-positive plasma cells and occasionally elevated serum IgG4. It is considered to be both a systemic inflammation and sclerosing disease. The most common manifestations are parotid and lacrimal swelling, lymphadenopathy and autoimmune pancreatitis. Sclerosing cholangitis and retroperitoneal fibrosis are among the other mentioned frequent manifestations. The diagnosis should be approved histo-pathologically but other conditions such as lymphoma should be carefully excluded. Patients with IgG4-RD respond beneficially to glucocorticoid therapy especially when given at early onset stages.

CNP42. Spectrum of biopsy proven renal disease in children

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Introduction: Renal biopsy has adefinite role in confirmation of diagnosis in various renal diseases.Histopathological diagnosis is not only helpful in diagnosis but also usefull in treatment and prognostication.

Aims: To study the spectrum of clinical profile of BPRD in children
Below 18 yrs.presented to the department of nephrology

Materials and Methods: Retrospective analysis of the histopathlogical reports of all renal biopsies
Performed from 2011-2016 in children below 18 yrs of age.All renal biopsies were studied by Light microscopy and IF microscopy.

Results: A total of 340 cases were analysed in the study age ranged from as early as 28 days to children less than 18 yrs.Male to female ratio was 1.3. the most common clinical indication for renal biopsy was nephrotic syndrome.Commnest BPRD was primary glomerulonephritis. MCD was commnest accounting for 42.3% followed by FSGS accounting for 12.35% Among the SGN PIGN was seen in 12.6% followed by Lupus nephritis 2.9%.
RPRF presentation was seen in 10% of cases among them among them ATIN was commonest 20%.Indication for renal biopsy in primary glomerulonephritis were FRNS(29%)
Age below1&10 yr (36%) SRNS(25)% ,SDNS .8%. In 2 cases of HIV positive children MCD was seen.2 cases of oxalosis were reported in infants.congential nephrotic syndrome finnish type 3 cases were reported below 1yr of age

Conclusion :our study represents an important contribution to understanding renal diseases in children.

CNP43. Histopathological spectrum of pediatric lupus nephritis

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Aim: We present the histopathological characteristics of lupus nephritis (LN) in children from eastern and southern parts of India.
Materials and Methods:A total of 10,953 native kidney biopsies received from August 2013 to November 2016 were reviewed and125 cases of lupus nephritis in children (16 years and below) were included in this study. Routine panel of light and immunofluorescence microscopic stains were performed on all biopsies. Lupus nephritis was classified according to ISN/RPS 2004 classification.
Results: The mean age of patients was14 years. Female to male ratio was 6:1.Hypertension was present in 9.6% of these patients.Class IV was the most common class. Two patients of class IV and one patient with class II also showed thrombotic microangiopathy. Two class IV patients had necrotizinglupus vasculitis.

Histopathologic features:

Class	I	II	III	IV	V	Mixed (III/V or IV/V)
Number of cases (%)	3.2	8.8	12.8	41.6	19.2	14.4
Crescents (%)	0	0	6.3	44.2	0	11.1
Karryorhexis(%)	0	0	0	21.2	0	16.7
Fibrinoid necrosis(%)	0	0	6.3	7.7	0	0
Interstitial inflammation(%)	0	0	6.3	23.1	0	0
IFTA(%)	0	0	6.3	11.5	4.2	11.1

Age and class distribution:

	Group 1(%)	Group 2(%)
Female: Male	4.5:1	6:1
Class I	0	4(3.50)
Class II	1(9.09)	10(8.77)
Class III	3(27.27)	13(11.40)
Class IV	3(27.27)	49(42.98)
Class V	1(9.09)	23(20.18)
Mixed	3(27.27)	15(13.17)
Total	11(100)	114(100)

Youngest patient in our study was 6 years old.We further classified the patients into two groups: children between 6 to 10 years were grouped into one group (group 1) and the other included children between 11 to 16 years of age(group 2).Class IV was the most common class. Class IV constitutes about 43% in group2 whereas in group 1it is only about 27%. In group 1,class III, class IV and mixed groups were equally distributed

Among the total 125 children, 11 were in group 1 and the remaining 114 were in group 2.Five patients (4%) were below 10 years of age.

Gender and class distribution:

Among the 125 patients, 107 were females and 18 were males. In both males and females, class IV was the most common class. 72% of males has class IV LN whereas in females class IV LN was diagnosed in 36.5%.

	Female (%)	Male (%)
Class I	3.74	0
Class II	9.34	5.56
Class III	14.95	0
Class IV	36.45	72.22
Class V	20.56	11.11
Mixed	14.95	11.11

Discussion: In our study we attempt to delineate the spectrum of lupus nephritis in the pediatric population.4% of children with LN were below 10 years of age.

The incidence of the disease was six times more common in girls than in boys.

ISN/RPS class IV was the most common class.

72% of boys had class IV LN.

Thrombotic microangiopathy and necrotising lupus vasculitis, though rare, were seen in ourpediatric population.

CNP44. Acute interstitial nephritis : An unusual histopathological finding in snake bite associated acute kidney injury

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Aim: Acute kidney injury(AKI) is one of the most important complications following snake bite, particularly viperine snake bites.This study presents the spectrum of changes seen in the kidney following snake bite

Materials and Methods: Native kidney biopsies reported from August 2013 to November 2016were reviewed and 62cases of snake bite associated acute kidney injury were identified. Renal biopsy was performed on patients with AKI who did not improve after 3 weeks of supportive care or if at any point in time showed deterioration of renal function.We compared our data with previously published studies.

Results: Among the 62 patients,27 patients (43.5%) had acute tubular injury, 19 (30.6%) had acute interstitial nephritis(AIN), thrombotic microangiopathy with patchy cortical necrosis was present in 5 patients (8.1%), cortical necrosis in 5 patients(8.1%), rhabdomyolysis induced acute tubular injury in 5 patients and (8.1%) papillary necrosis was present in 1 patient (1.6%).

Discussion:AIN was diagnosed in nearly one third of the cases in our series. Most of the published studies reported acute tubular injury and cortical necrosis as the most common histopathological finding. Only occasional single case reports and a case series of 5 patients with AIN following snake envenomation have been reported. To our knowledge, this is the largest series of this unusual histopathological finding in snake bite associated AKI. Many factors may contribute to the development of AIN. Direct nephrotoxicity of snake venom or hypersensitivity to anti snake venom may be postulated as possible causative factors.

CNP45. Low Fetuin A Levels Is An Independent Predictor Of Vascular Calcification In CKD

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Background And Objectives: Cardiovascular disease contributes to significant morbidity and mortality in patients with CKD. Fetuin A is a circulating calcium regulatory glycoprotein that inhibits vascular calcification. The present study aims to assess the Fetuin A levels in CKD against a normal control population and its relationship to vascular calcification.

Methods: 30 CKD patients on conservative management, 30 patients on hemodialysis were compared against 30 controls who included age matched normal individuals. Vascular calcification was assessed by abdominal aortic calcification score (AAC). Fetuin A levels in the patients were assessed against their calcification scores to look for any correlation

Results: The mean serum Fetuin-A levels in patients with CKD were significantly lower than controls. AAC scores were significantly higher in patients with CKD and showed considerable correlation to low Fetuin A levels. No correlation could be demonstrated between calcium phosphorus product and vascular calcification.No significant difference was noted between patients on conservative management and those on hemodialysis.

Conclusions: Increased vascular calcification in CKD depended on vasculoprotective factors like Fetuin A levels. Fetuin levels in CKD was significantly reduced independent of the metabolic profile

CNP46.Microalbuminuria and serum cystatin C correlation as an early predictor of renal impairment in patients with type 1 diabetes

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Introduction : Early detection of renal impairment is important in type 1 diabetes to retard the progression to ESRD. Even though the microalbuminuria is an important indicator, some patients have renal impairment without microalbuminuria. Hence use of other earlier markers like Cystatin C levels which increases in serum earlier than creatinine.

Aim &Objectives :To study the correlation of micro-albuminuria with serum cystatin C levels as an early predictor of renal impairment in patients with type 1 diabetes in our tertiary care hospital.

1. To estimate the ACR ratio in urine and serum creatinine, urea, cystatin C, plasma HbA1c
2. To correlate ACR with serum cystatin C levels in type 1 diabetic patients.

Materials and Methods :the study was conducted in type 1 diabetic patients from diabetology clinic. patients who had hypertension, uti, heart failure and thyroid dysfunction were excluded. in this study 72 subjects with type 1 diabetes were evaluated. they were divided into two groups depends on the duration of diabetes. group i had 17 subjects (<5 yrs of diabetes), group ii had 55 subjects (>5yrs diabetes). in both groups the patients are sub-categorised into cases (30 to 300 mg/g) and controls (<30mg/g) based on the urine albumin creatinine ratio.

Micro-albuminuria tested with dipstick and latex turbidometry, modified Jaffe's method used for creatinine measurements. Colloidal gold enhanced turbidometry used for cystatin C measurement. In both cases and controls, serum cystatin C, creatinine levels were estimated and correlated with urine albumin levels. .The results were compared by using excel software. Student unpaired "t" test, and Pearson coefficient of correlation were used for statistical analysis.

Results : Significant difference noted only in ACR levels between cases and controls of group I (96.99 vs 19.79). But significant difference noted in ACR levels, serum creatinine, cystatin C, and HbA1c levels between cases and controls of group II.

Pearson correlation showed significant positive correlation between duration of diabetes and ACR (0.1), cystatin C (0.4), HbA1c (0.4). Significant positive Pearson correlation of ACR with serum creatinine, cystatin C, and HbA1c levels, onlyin group II patients. Unpaired "t" tests between ACR and serum creatinine, cystatin C, and HbA1c levels in group II patients showed statically significant difference (7.65, 7.98, and 7.29)

Discussion : This study showed that elevation of serum creatinine, cystatin C, ACR, and HbA1c levels among cases of >5yrs duration, denotes that prevalence of nephropathy is related to duration of diabetes. Statistically significant ACR levels between cases and controls of group I signifies that screening for microalbuminuria may be done one year after diagnosis. The cystatin

C is increased in microalbuminuric group of diabetic patients with >5yrs duration. Serum creatinine in these patients was not significantly increased. This refers that serum cystatin C is an early marker of diabetic nephropathy in comparison to serum creatinine.

Conclusion: Microalbuminuria is correlated with cystatin C in patients with more than 5yrs of type 1 diabetes mellitus. Hence cystatin C can be used for early detection of renal impairment in patients with >5yrs of type 1 diabetes.

CNP47.IgG lambda myeloma in a patient with IgA nephropathy

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IgA nephropathy is a relatively common disorder, with a prevalence of 1–2 in 1000.Monoclonal gammopathy accounts for 1% of population in people greater than 50 years. IgA nephropathy with IgA lambda myeloma with mesangial proliferative glomerulonephritis has been reported . Droz *et al.* have reported a case of iga nephropathy associated with an increase in polyclonal IgA and a monoclonal IgAλ in the serum, with only monoclonal IgAλ deposition in the kidney. We report a rare case of 65 year male with IgA nephropathy who developed IgG myeloma which is not reported so far as to our knowledge

A 65 year gentleman, diabetic and hypertensive presented with pedal edema facial puffiness and genial swelling with nephrotic proteinuria with urine PCR 3.1,creatinine 1.04,total cholesterol 242 which on renal biopsy revealed IGA nephropathy Lee class III. He was started on oral steroid prednisolone 60mg/day.1 month later patient developed ascites and fever diagnosed as abdominal tuberculosis and was started on ATT. He stopped ATT after 6 months.5 months later patient had GTCS, found to have parietal tuberculoma hence restarted on 5 drug ATT.2 months later patient presented with loss of appetite and weight loss.He was found to have mild renal dysfuncion with subnephrotic proteinuria. Bone marrow aspiration revealed plasmacytosis with marrow plasma cells 24% with IGG lambda predominance on immunofixation, b2 microglobulin 7842 and free light chain ratio 0.47,serum albumin 3.1. He was started on treatment for myeloma following which he recovered

CNP48.Hypernatremia in an acute kidney injury patient

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Hypernatremia is less common than hyponatremia in a patient of acute kidney injury requiring haemodialysis. Literature on the management of hypernatremia with acute kidney injury provided two diametrically opposite approaches. The first approach is a cautious one and entails to first dialyse the patient with dialysis solution sodium concentration close to that of plasma and then correct the hypernatremia by slow administration of slightly hyponatremic fluids.The divergent approach in the literature is from the reports of acute hypernatremia with hypotonic haemodialysis. The serum sodium decreased by19 to20 mEq/L within four hours of dialysis with 110 mEq/L of dialysate sodium. We were caught on the horns of dilemma in treating a 22-year-old lady with hypernatremia and acute kidney injury.

CNP49. A maintenance haemodialysis with pericardial effusion: an unusual cause

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The central venous catheters were complicated by the brachiocephalic and the superior vena cava stenosis and thrombosis. The standard textbooks of the internal medicine have not cited the thrombosis of these veins as a cause of pericardial tamponade.We report a patient of thrombosis of the brachiocephalic and the superior vena cava with cardiac tamponade. This resulted in insufficient drainage of pericardiophrenic veins which serve the pericardium.

CNP50.A report of lupus vasculitis with cANCA positivity

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Background: Systemic lupus erythematosus (SLE) and small sized vessel vasculitis are two well defined clinical entities. There are very few case reports of systemic lupus erythematosus overlapping with c-anca posotive vasculitis decribed till now int he literature. **Aim of the study:** TO elucidate the severity of presentation and its worse prognosis despite the best possible treatment in a patient of systemic lupus erythematosus with canca posotive vasculitis. **Methods:** A 55-year-old woman was admitted for complaints of both for 3 months; easy fatigue with exertional breathlessness and reduced urine output for 3weeks. She had repeated episodes of hemoptysis during this period. Investigations were suggestive of serum creatinine: of 7.4 mg/ dl withserial fall in haemoglobin from 9.8 mg/dl to 7.7 mg/dl ; PO2 70% on arterial blood gas analysis . HRCT thorax was suggested bilateral lower lobe consolidation with pulmonary haemorrhage. On bronchoscopy there was bleed from right lower lobe. She had anti dsDNA-positive with more than ANA 2+ positive ;5 fold rise in anti ds dna antibody titres; speckled pattern; CANCA positive 57(< 6.5 mg/dl(R.R – 10-40); direct coomb's was 2+ positive; lupus anticoagulant positive **. Results:** Renal biopsy was suggestive of diffuse proliferative sclerosing glomerulonephritis with cellular to fibrocellular crescents in 4 out of 6 glomeruli and a concomitant focus of acute necrotisng vasculitis of a medium sized interlobular artery; IF-granular deposits of IgG on the capillary walls 1+ to 2+ . Focal trapping of IgM in a glomerular tuft; focal granular mesangial c3 deposits 1+. classIV; with a final impression of crescentic glomerulonephritis; activity score being 21out of 24.She was treated with 3 doses; 500 mg of iv pulse methylprednisolone; 1 dose 500mg of iv cyclophosphamide; 6 sessions of plasmapheresis and intravenous imminuoglobulin 30 grams in 5 divided doses . Despite these efforts the patient continued to have recurrent episodes of hemoptysis with respiratory failure requiring artificial ventitlatory efforts. ; but finally could not be revived .**Conclusions:** We stress here that a patient of systemic lupus erytemtosus; lupus nephritis; with vasculitis; positive for c-ANCA had severemanifestations; dismal prognosis despite extensive treatment.

CNP51.Collagenofibrotic glomerulopathy

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A 54-year-old lady, had been a non-diabetic and hypertensive for the last one year. About two years ago, she had swelling of feet, facial puffiness and abdominal distension over a period of one month. She was evaluated outside. The investigations performed then were: urine albumin 3+, urine protein creatinine ratio: 9.5, serum albumin: 1.9 g/dL. She underwent a renal biopsy then. There were 13 glomeruli, all were enlarged in size. There were a few pale eosinophilic acellular, weakly PAS positive material in mesangium. Similar material was found along the capillary loops. There were negative with silver stain. The Congo red stain does not show apple green birefringence. The negative Congo red confirmed even on thick section. There was no hypercellularity, necrosting lesions, or crescents. Tubules and interstitum showed atrophy, fibrosis or interstitial deposits. There was hyalinosis of small arterioles, tunica media hyperplasia and no vasculitis. Immunofluorescence microscopy: showed focal and segmental trapping of IgM, 1+ and C3, trace positive in the areas of hyalinosis. C1q, IgA, IgG, C3, fibrin were negative. It was reported as membranoproliferative glomerulonephritis. Prednisolone and angiotensin converting enzyme inhibitors (ACEi) were given as the treatment.

She presented to our Institute with history of worsening anasarca. The blood pressure was 150/100 mm Hg. The investigations were, serum creatinine: 2.3 mg/dL, blood urea: 54 mg/dL, haemoglobin: 6.3 g/dL, total serum proteins: 4.8 g/dL, serum albumin: 2.0 g/dL, 24 hour urine protein: 9.3 g. The peripheral smear revealed normocytic, normochromic anaemia. She showed negative serologies for ANA, hepatitis C, hepatitis B and HIV and had normal serum C3 and C4 levels. ANCA serologies were negative. There was no previous family history of renal disease or signs/symptoms of nail–patella syndrome. A second renal biopsy was performed. The findings were, there were ten glomeruli. One glomerulus was globally sclerotic. The remaining were enlarged with deposition of a pale PAS and silver negative material in the mesangium in a diffuse and nodular pattern. This material is also focally deposited over the capillary loops It was blue on trichrome stain. The Congo red stain for amyloid was negative. The capillary loops showed double contours. There was no endocapillary proliferation, necrotising lesions or crescent formation. The interstitial fibrosis and tubular atrophy involved 25% of the core. Prominent arteriolar hyalinosis was present. The electron microscopy displayed the expansion of the mesangium by stacked frayed collagen bundles. The glomerular basement membrane was globally thickened. Visceral epithelial cells show severe foot process effacement. No immune type electron dense deposit was visible. The immunoperoxidase stain for collagen III was positive. She was started on Prednisolone 1 mg/kg/d, ACEi and angiotensin receptor blockers. During next two months the serial serum creatinine values showed a declining trend.

CNP52.An unusual case of retained DJ stent for 10 years presenting with associated complications and AKI

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A 45 year aged male patient presented with complaints of left loin pain,dyuria x 15 days,Oligoanuria x 3 days,Azotemia x 3 days. Patient had H/s/o LUTS,H/o Gravelluria,H/o Perurethral surgery 10 yrs back at Karimnagar (details not available),lost for follow up thereafter.

At admission,General and systemic examination is unremarkable

On evaluation his Hb-10.3gm/L,TLC-14800,PLT-2.3L,LFT-WNL,S.Creat18mg/dl,CUE Alb-nil, RBC-nil, Pus cells plentyL and his Urine C/S is sterile.

His x-ray showed migration,encrustation,fragmentation o f the stent and vescical calculus

His USG abdomen**:** Right kidney normal.Left gross HDUN, hyperechoic content noted in the bladder with posterior shadow extending upto ureteral opening : suggestive of calcified DJ stent his CT KUB showed 30mm calculus in urinary bladder with calcified DJ stent traceable upto distal ureter.

Diagnosis:Forgotten DJ stent with complications,Left gross hydroureteronephrosis with Vesical calculus with urosepsis AKI(AKIN3)recoveredManagement:Initiated on HD through Right IJV. Left PCN doneUrsl +Left DJS removal followed by Open cystolithotomy.Serum creatinine was 2mg/dl and is dialysis independent. Take home message:Adequate counseling before putting DJ stent. Stringent and regular follow up of those with stent.

CNP53.Association of Bone Morphogenic Protein 4 Gene Polymorphism and Left Ventricle Hypertrophy in Diabetic Chronic Kidney Disease Patients: A Pilot Study

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Background: Bone Morphogenetic Protein 4 (BMP4) is identified to play a significant role in cardiac remodelling. Gene polymorphism of BMP4 and its resulting associations with Left Ventricular Hypertrophy (LVH) in diabetic Chronic Kidney Disease (CKD) patients are yet to be well established.

Aim: Toanalysethe association between BMP4 gene polymorphism and LVH in diabetic CKD patients.

Materials and Methods: Isolation ofDNA from whole blood samples of ten patients each; patients diagnosed with LVH and diabetic CKD (group 1), patients with LVH without diabetic CKD (group 2), patients with diabetic CKD without LVH (group 3),
normal patients as control (group 4) were extracted.The gene of interest (BMP4 gene) purified from various samples digested using zero-cutter restriction endonucleases (*Hind III* and *Bam HI)* by employing the Restriction Fragment Length Polymorphism (RFLP) technique. The restriction has been analysed using 1 % agarose gel Electrophoresis.

Results: The gene from group 2; patient having LVH without diabetic CKD when digested with *Hind III* showed fragmentation, more specifically, it presented threefragments which were at a comparable distance corresponding with the following size reference markers at 1500bp, between 700bp to 600bp and the last one near 100bp. This fragmentation pattern was repeated identically for the gene from blood sample of patient having LVH with diabetic CKD which was also digested with *Hind III*. A similar fragmentation was not visualized for sample from group 3 patient having diabetic CKD without LVH and group 4 normal individual when digested with *Hind III*. But no such fragments were noted for the samples from the same patients when digested with *Bam HI*.

Conclusion: BMP4 genepolymorphism has been confirmed in patients having LVH regardless of the presence or absence of diabetic CKD along with it.