17th ISRTPCON 2024

17th ANNUAL NATIONAL CONFERENCE AND INTERNATIONAL CME OF INDIAN SOCIETY OF RENAL AND TRANSPLANTATION PATHOLOGY

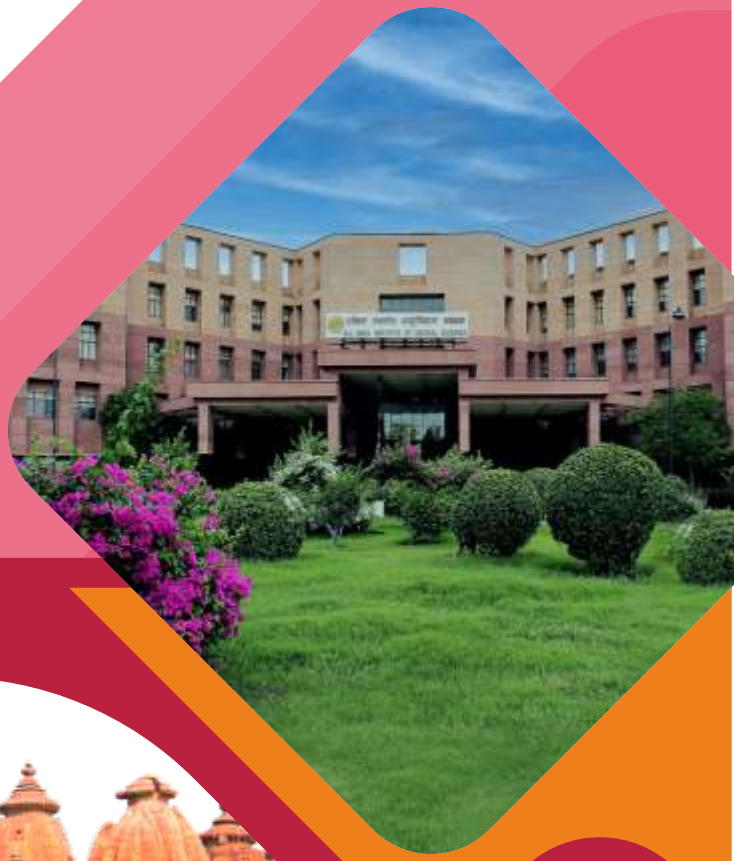
Renal Pathology: Concepts, Contemporary Approach & Updates

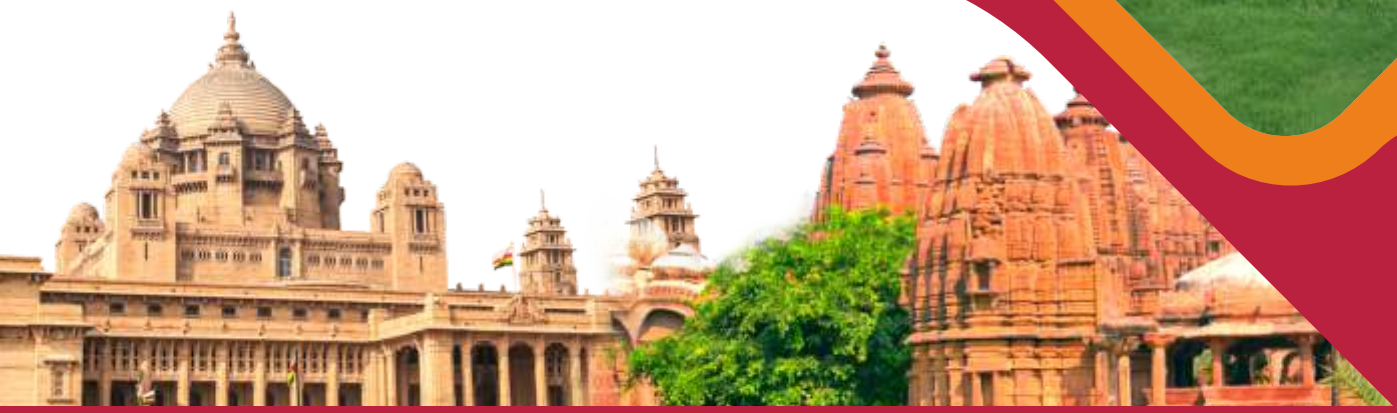
Auditorium, AIIMS Jodhpur

23rd - 25th Feb. 2024

SOUVENIR

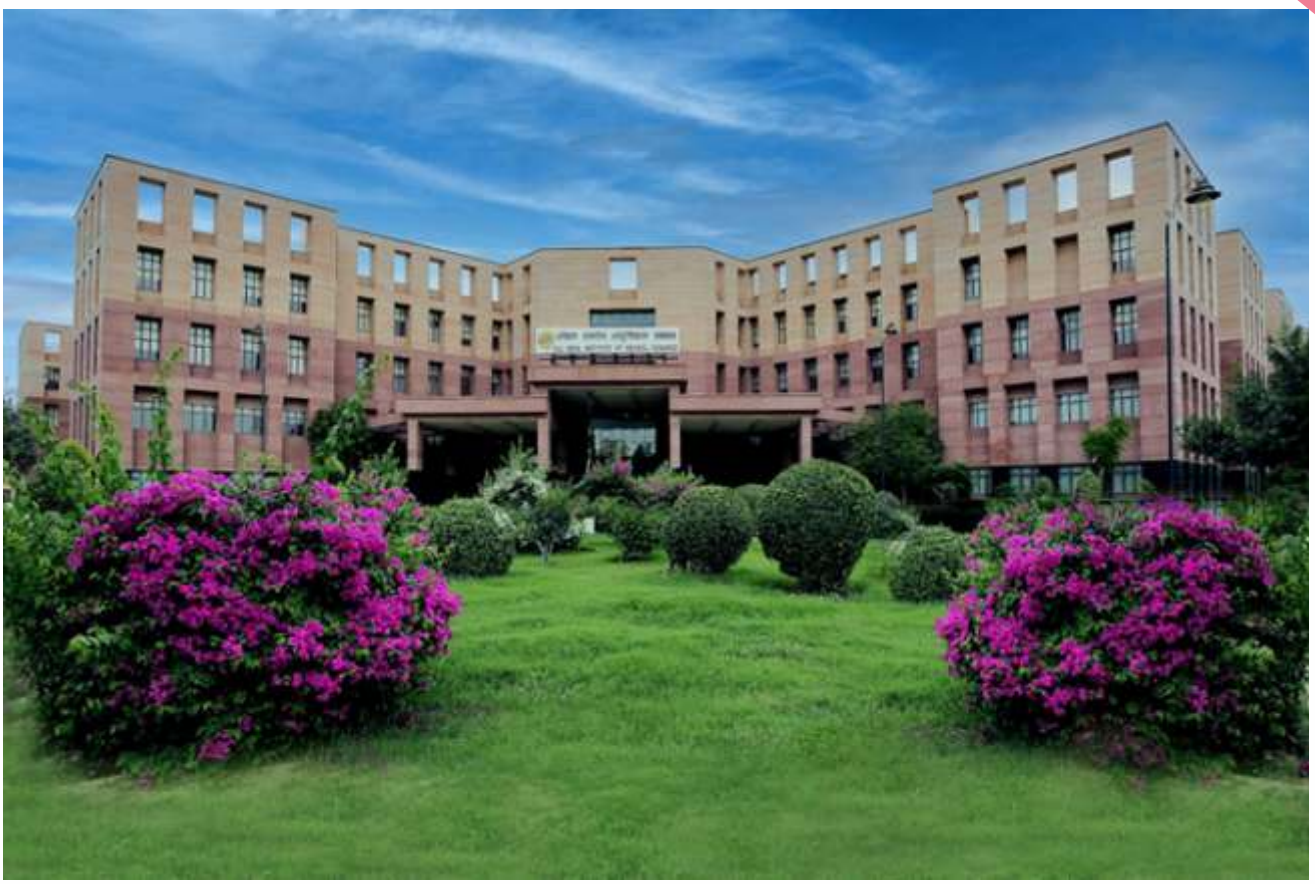
UNDER THE AEGIS OF
INDIAN SOCIETY OF RENAL AND
TRANSPLANTATION PATHOLOGY
(ISRTP)



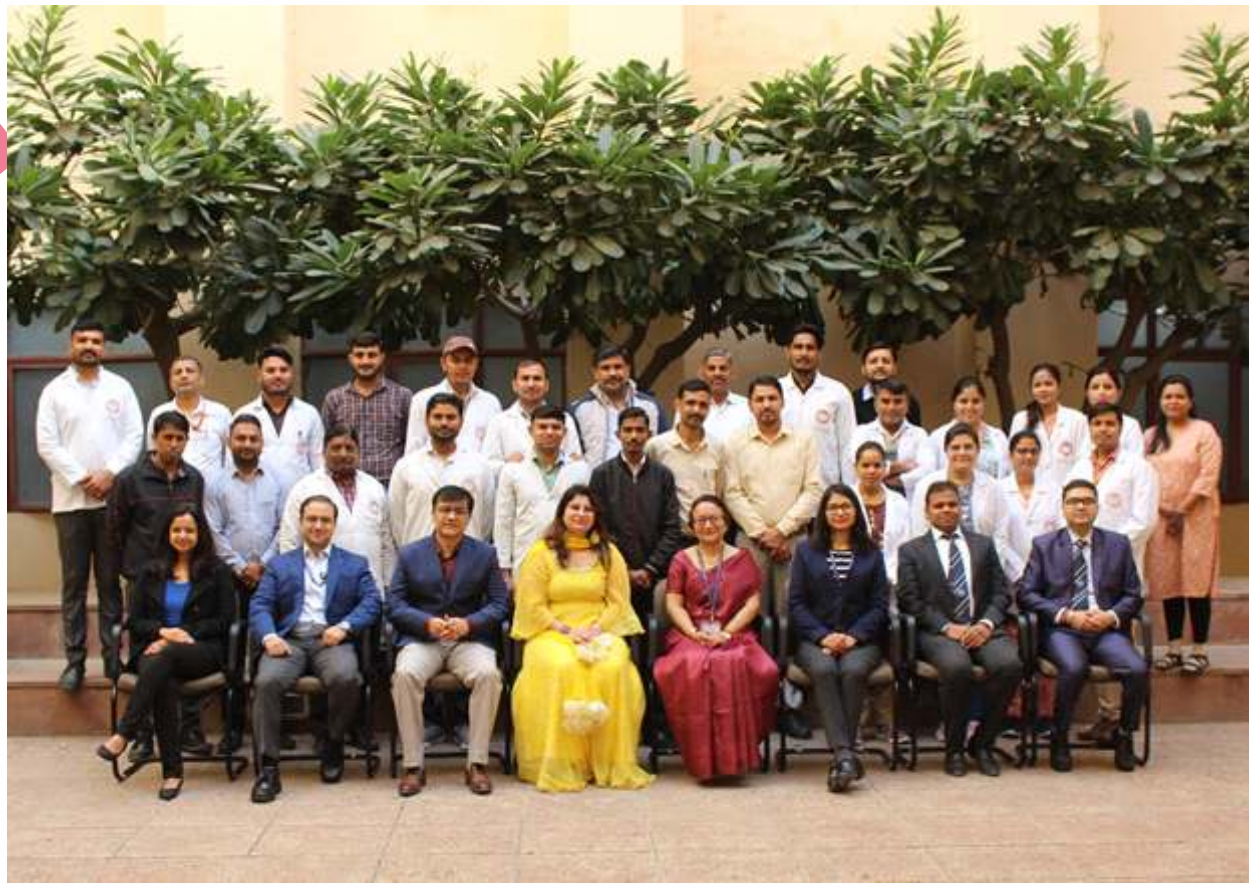


ORGANIZED BY
DEPARTMENT OF PATHOLOGY AND LAB MEDICINE, AIIMS JODHPUR

About AIIMS, Jodhpur

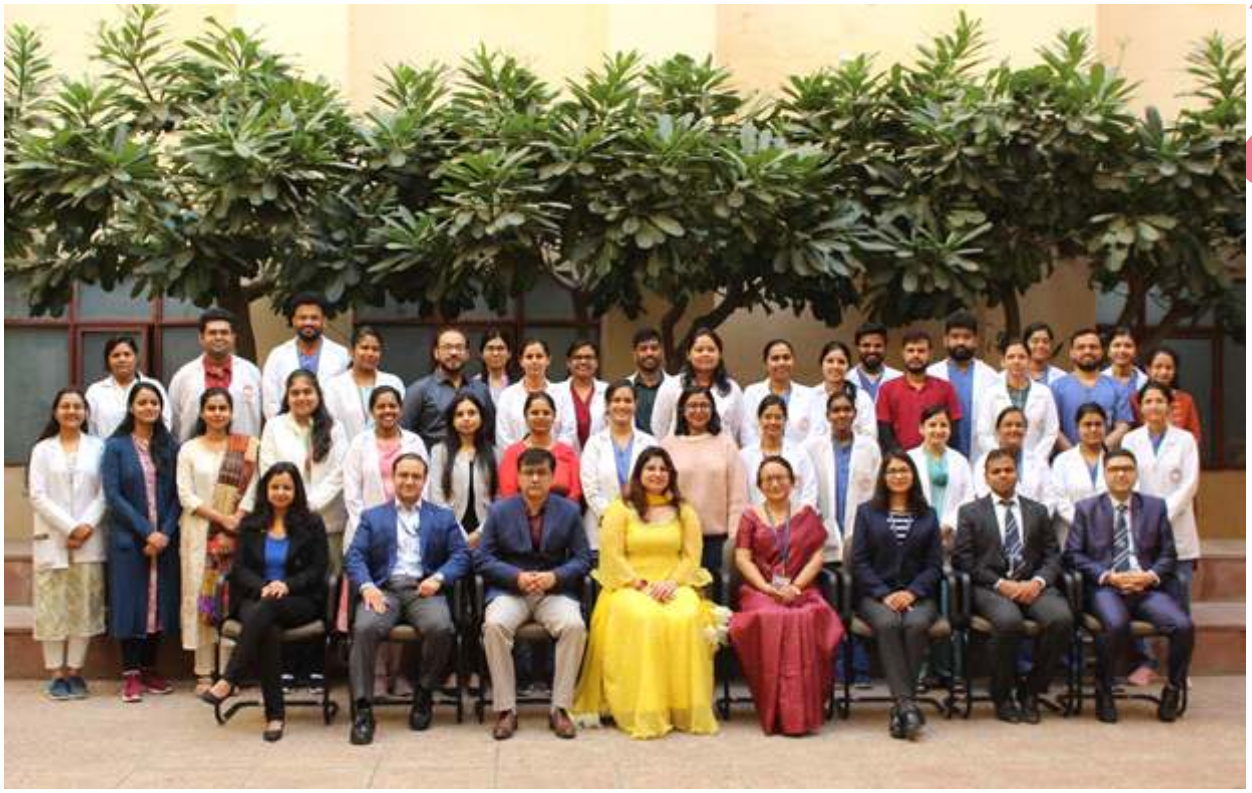


AIIMS Jodhpur is one of the six new AIIMS established by the Ministry of Health and Family Welfare, Government of India under the Pradhan Mantri Swasthya Suraksha Yojna (PMSSY). It was established to set a benchmark in medical education and research facilities in all branches of health care. Department of Pathology & lab medicine is equipped with modern diagnostic techniques involving Molecular, Immunofluorescence, Immunohistochemistry etc., focusing to provide quality diagnostic benefit to all.



Faculty & Staff, Department of Pathology & lab Medicine
AIIMS, Jodhpur

Organizing Team
Indian Society of Renal & Transplantation Pathology
(ISRTPCON, 2024)



Faculty & Residents, Department of Pathology & Lab Mednince
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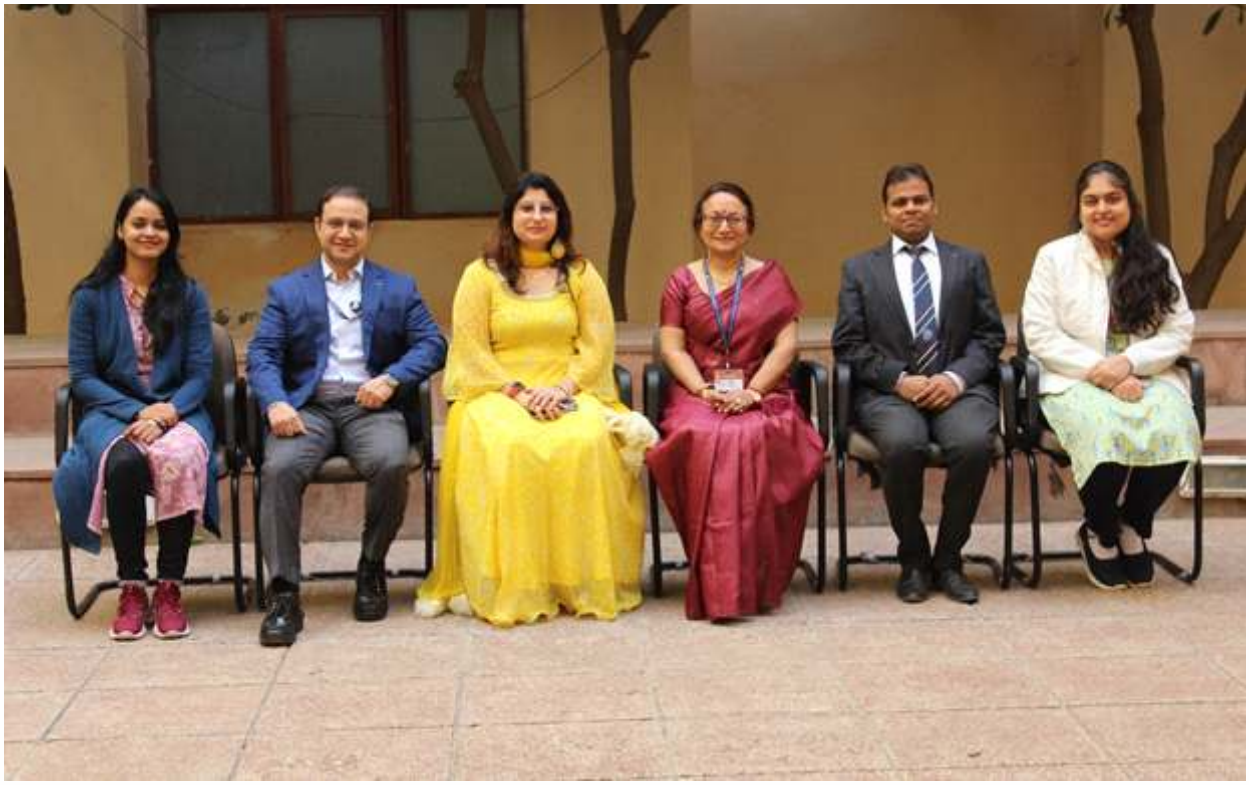

Renal and Transplant Pathology Team
AIIMS, Jodhpur

Message

Dr. Ritambhra Nada
President
Indian Society of Renal & Transplantation Pathology
(ISRTPCON,2024)

Greetings from Chandigarh!

It gives me immense pleasure to state that our Renal pathology community in India is growing and flourishing. Credit goes to our worthy, committed leaders and teachers, Prof. RK Gupta and Prof. Kusum Joshi, who conceptualized and initiated this drive of focused group and meetings. It is our responsibility to carry forward the spirit of fraternity and keep improving. We can see that renal pathology is now available in most parts of our country. Impact of our annual meetings is palpable in form of change in routine practices like use of light chains in immunofluorescence panels adopted universally by all practicing renal pathology in India. Electron microscopes are available in many centers.

Our endeavour is to penetrate medical colleges so that our young minds get interested and join us. We attempted an online basic series for them, which was very well conceived and executed by Dr. Swaranlatha and Dr. Geetika. We shall organize an online slide course for them. On the other hand, we organize to have masters from across the globe to learn from the experts to update our practicing renal pathologists.

My personal view is that we need to increase our international representation, improve national data collection to publish them and Pan-India mapping of nephropathologists to organize availability in deficient areas. We need to make Special Interest groups depending on the availability of expertise in those institutes /centers, which become referral centers for that disease. International referrals are not accessible to most of our patients. Our solutions have to be local.

I see a very bright future for our society with young, enthusiastic pathologists and valuable guidance from our seniors.

I best wishes for this meeting and hope it accomplishes the intended purpose.

RNada

Dr. Ritambhra Nada

Message

Dr. Alok Sharma
Secretary,
Indian Society of Renal & Transplantation Pathology
(ISRTPCON, 2024)

Dear colleagues, respected seniors, and dear students,

It gives me immense pleasure to welcome you to the 17th annual conference and International CME of Indian Society of Renal & Transplantation Pathology (ISRTP).

These are exciting times for our speciality, as we are witnessing unprecedented rapid strides in diagnosis and management of renal diseases. The critical role of Nephropathologist as a key member of team that cares for patients with native renal diseases as well as renal allografts is now well recognized, and it is of paramount importance that we keep ourselves abreast with recent advances and updates in this field.

The robust scientific program of ISRTPCON 2024 features lectures from national & International stalwarts in Renal & Transplantation pathology and promises to be a thorough academic feast for one and all. The trainees under mentorship of senior faculty and practitioners will participate in case & poster presentations and showcase the remarkable spectrum of Nephropathology being practised in India.

I urge everyone to use this opportunity to draw inspiration from leaders in this field, reflect on accomplishments and extend your collaborations & networks. I am sure that you will have a fruitful and enjoyable experience in the beautiful city of Jodhpur. I convey my warm greetings to the organising committee and extend best wishes for the success of ISRTPCON2024.

Warm Regards,

Dr. Alok Sharma

Message

Dr.Poonam Elhence
Professor & Head,
Department of Pathology & Lab Medicine,
AIIMS, Jodhpur

I welcome each one of you to the 17th Annual National Conference and International CME of Indian Society of Renal and Transplant Pathology (ISRTP)- ISRTPCON2024 from 22nd-25th February, at AIIMS, Jodhpur.

All my Faculty colleagues, Residents and Department staff, ably lead by Dr. Aasma, the Organizing Secretary, have worked day & night to make this event a success.

I wish to extend my heartfelt gratitude to Professor GD Puri, Executive Director, AIIMS, Jodhpur, the Administration and all the ancillary staff for their constant support.

I am sure that it will be a wonderful academic event with excellent talks, lectures, discussions and presentations. After the 4 days of academic deliberations by esteemed National and International Faculty and forging of personal and professional bonds, I hope we can go back much richer, both academically and culturally.

I hope each one of you enjoys this academic feast and the hospitality for which Jodhpur is known.

I wish this conference a huge success.
Jai Hind.

Dr.Poonam Elhence
Organizing Chairperson
Indian Society of Renal & Transplantation Pathology (ISRTPCON,2024)

Message

Dr. Aasma Nalwa
Organizing Secretary
Indian Society of Renal & Transplantation Pathology
(ISRTPCON, 2024)

Dear Esteemed Colleagues and Honoured Guests,

It is with great pleasure and a profound sense of gratitude that I extend my warmest welcome to each of you through these pages of our conference souvenir. As the Organizing Secretary of 17th Annual National Conference and International CME of Indian Society of Renal and Transplantation Pathology, it has been an honour to play an instrumental role in bringing together such a distinguished gathering of experts and enthusiasts in the field of Renal Pathology. From the inception of the idea to the meticulous planning and execution, every step has been guided by our shared commitment to excellence.

The Indian Society of Renal and Transplantation Pathology (ISRTP) was conceived during the "International CME on Renal Pathology," organised at the Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS) in Lucknow, India in March 2005. This society was brought to life with the primary aim to foster progress, research, educational initiatives, and training in the realms of native renal and transplantation pathology. Additionally, ISRTP seeks to engage in collaborative efforts with like-minded organizations, both domestically and internationally, to enhance healthcare delivery for individuals afflicted with ailments affecting native kidneys and renal transplants.

The Department of Pathology and Lab Medicine at AIIMS Jodhpur is committed to the cause of research and development in the domain of renal pathology. In addition to modern diagnostic techniques like immunohistochemistry, immunofluorescence, the institute also offers annual post-doctoral fellowship in renal pathology. The lab receives above 200 native

kidney biopsies yearly and is in the process of procuring an electron microscope to further elevate and enhance its standards of patient care. The institute also boasts of a one of its kind, renal transplant unit, that has been active since 2016 and contributes upto 35 transplant biopsies across a year.

Renal Pathology is evolving at a fast pace and it is of paramount importance that we keep ourselves abreast with recent advances and updates. Taking this into account, the theme for this year: Renal Pathology: Concepts, Contemporary Approach and Updates, was chosen after ample deliberation.

As I reflect on the journey that led us to this moment, I am humbled by the depth of knowledge, the spirit of collaboration, and the unwavering passion that each participant brings to the table. It is this collective energy that has made our conference a resounding success.

Thank you to each and every one of you for your contributions, your enthusiasm, and your commitment to excellence. It has been an honour and a privilege to serve as your Organizing Secretary, and I look forward to our paths crossing again in the pursuit of scientific advancement.

Dr. Aasma Nalwa

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PRE-CONFERENCE WORKSHOP

Pre-Conference CME 22nd February 2024- Thursday		**Venue: LT -1**
Time	**Topic**	**Speakers**
8:00 - 8:35 am	**Registration**	
8:35- 8:45 am	Welcome Address with Introduction of Speakers	Dr Deepika Gupta/ Dr Ayushi Saxena
8:45-9:30 am	Interpretation of native kidney biopsies	Dr. Aasma Nalwa
9:30-10:15 am	Non-proliferative lesions in the kidney	Dr. Vikrant Verma
10:15- 10:30 am	Tea Break	
10:30 am-11:15 am	Proliferative lesions in the kidney	Dr. Vikarn Vishwajeet
11:15-11:45 am	Video Demonstration of IF	Dr Debasis Gochhait
11:45 am-12:15 pm	Electron microscopy	Dr Vikrant Verma
12:15-12:45 pm	Basics of Tubular and Vascular lesions	Dr. Aravind Sekar
12:45-1:15 pm	Quiz	Dr Deepika Gupta/ Dr Ayushi Saxena
1:15- 2:00 pm	Lunch	

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SCIENTIFIC PROGRAM

Day 1- 23rd Feb 2024			
Time	**Topic**	**Speakers**	**Chairpersons**
8:15-9:00 am	Registration		
SESSION 1 (9:00 am- 10:20 am)			
9:00-9:30 am	Nephrotic syndrome - clinical perspective	Dr. Manish Chaturvedy	Dr. Kuldeep Singh, Dr Surendra Rathore, Dr Manoj Jain
9:30-10:00 am	FSGS- revisited	Dr.Seethalekshmy N V	
10:00-10:20 am	Genetic Storage & Lipid Disorders of the Kidneys	Dr Joris Roelofs	
10:20-10:55 am	Tea Break & Poster Viewing		
SESSION 2 (10:55 am – 12:35 pm)			
10:55- 11:15 am	Clinician's perspective of monoclonal renal disorders	Dr Rajesh Jhorawat	Dr. Alok Sharma, Dr Kusum Joshi, Dr. Rajesh NG
11:15 - 11:40 am	Diagnosing renal amyloidosis- from IHC to mass spectrometry	Dr Ritambhra Nada	
11:40am- 12:10 pm	Role of electron microscopy in glomerular diseases with organized deposits	Dr Manoj Jain	
12:10-12:35 pm	Case-based approach- Monoclonal Renal disorders	Dr Mahesha V	
12:35- 1:30 pm	Lunch Break & Poster Viewing		

SESSION 3 (1:30 -3:20 pm)			
1:30-2:00 pm	Basement membrane disorders- Alport and beyond	Dr Joris Roelofs	Dr R K Gupta Dr. Manish Chaturvedy, Dr Seethalekshmy N V
2:00- 2:35 pm	Endemic Nephropathy	Dr Swarnalatha Gowrishankar	
2:35- 2:50 pm	Renal side effects of immune checkpoint inhibitors	Dr Joris Roelofs	
2:50- 3:20 pm	Drug-induced renal injury	Dr Srinivas BH	
3:20-3:50 pm	Tea break & Poster viewing		
3:50- 4:05 pm	Inauguration		
SESSION 4 (4:05- 5:45 pm)			
4:05- 4:30 pm	Lupus Nephritis in 2023	Dr Vinita Agrawal	Dr Harsharan K Singh, Dr Joris Roelofs, Dr Chitra Madiwale
4:30- 4:55 pm	ANCAs and Atypical Anti-GBM	Dr Anila Kurien	
4:55- 5:45 pm	Case Discussion: Native kidney disease	Dr Tushar Toprani Dr Usha Rani Dr Sanjeet Roy Dr. Vikrant Verma Dr Khushbu Agarwal	Moderator- Dr Chitra Madiwale Chairpersons: Dr Seethalekshmy N V, Dr. Alok Sharma, Dr. Arvind Kalla
5:45 pm onwards	ISRTP executive committee meeting		

Day 2- 24th Feb 2024			
Time	Topic	Speakers	Chairpersons
Session 5 (9:00-11:00 am)			
9:00- 9:30 am	MPGN and C3 glomerulopathy	Dr Anthony Chang	Dr. Mayank Jain Dr Ritambhra Nada, Dr Swarnalatha Gowrishankar
9:30-10:00 am	Thrombotic microangiopathy	Dr Geetika Singh	
10:00- 10:20 am	Company Talk	Everlife CPC Diagnostics	
10:20- 10:40 am	Bacterial (Non-streptococcal) Infection-related GNs	Dr Kamal Kanodia	
10:40- 11:00 am	QUIZ	Dr Rajan Duggal	
11:00 - 11: 25 am	Tea break & Poster Viewing		
Session 6 (11:25 am -1:00 pm)			
11:25- 11: 45 am	IgA nephropathy: what's new?	Dr Rajesh N G	Dr Manoj Jain, Dr Anila Kurien, Dr. Geetika Singh
11:45 am- 12:05 pm	Membranous Nephropathy	Dr Alok Sharma	
12:05- 12:30 pm	Tubulointerstitial disorders	Dr Vineeta Batra	
12:30- 1:00 pm	Artificial intelligence in renal pathology	Dr Pallav Gupta	
1:00- 2:00 pm	Lunch Break & Poster Viewing		

Session 7 (2:00 pm- 3:40 pm)			
2:00- 2:20 pm	Procurement Biopsies & Implantation Biopsies	Dr Harsharan K Singh	Dr Ritambhra Nada, Dr Joris Roelofs, Dr. Vinita Agrawal
2:20- 2:40 pm	Life of tubulointerstitial lesions in kidney transplants	Dr Volker Nickeleit	
2:40- 3:00 pm	Post-transplant viral infections	Dr Harsharan K Singh	
3:00- 3:20 pm	BK virus associated urothelial tumors	Dr Volker Nickeleit	
3:20 - 3:40 pm	Practical application of electron microscopy in renal transplants	Dr Harsharan K Singh	
3:40 – 4: 00 pm	**Tea Break & Poster Viewing**		
Session 8 (4:00-5:20pm)			
4:00- 4:20 pm	Glomerular injury in Kidney Transplants: navigation tools for pathologists	Dr Volker Nickeleit	Dr Ritambhra Nada, Dr. Vinita Agrawal
4:20- 5:20 pm	Case discussion- renal allograft	Dr A W Kashif Dr Kamlesh Suthar Dr Shruti Sabnis Dr Bhavana Mehta Dr Debasis Gochhait Dr Shailesh Soni	Moderator: Dr. Ranjana Ranade Chairpersons- Dr. Vineeta Batra Dr Mahendra Jangid Dr Pallav Gupta
5:20- 5:40 pm	ISRTP general body meeting		

Day 3- 25th Feb 2024			
Session 9 (8:45- 12:15 pm)			
8:30-8:45 am	Tea		
8:45- 9:15 am	Novel Modalities to explore the Immune landscape of Kidney Biopsies: A deep dive into kidney allograft	Dr. Mariam Priya Alexander	Dr Volker Nickeleit Dr. Vineeta Batra Dr. Vinita Agrawal
9:15- 9:35 am	Role of biopsy in renal allograft- clinician's perspective	Dr Sishir Gang	
9:35- 10: 00 am	Newer techniques in nephropathology	Dr Aravind Sekar	
10: 00- 10: 30 am	Tea Break		
10:30-10:55 am	Microvascular inflammation in kidney transplant	Dr Adarsh Barwad	Dr Rajesh Jhorawat Dr Shailesh Soni, Dr Anila Kurien
10:55- 11:25 am	HLA testing	Dr Biman Saikia	
11:25 am-11:45 am	Oral paper presentation		
11:45 am - 12:15 pm	Valedictory function		

Evaluation of Target Antigens in Membranous nephropathy and Clinico-pathological correlation.

Dr.Chitra J, Dr.Seethalekshmy N V, Dr.Preethi S, Dr.A Navya Sri

Background:
Membranous nephropathy (MN) is the most common cause of idiopathic nephrotic syndrome in adults. The subepithelial immune complexes in MN are primarily composed of a target antigen and IgG directed towards it. Emerging evidence suggests that the different target antigens are associated with distinct patterns of associated diseases.

Aim:
To study the expression of target antigens in MN.

Method:
PLA2R, THSD7A, NELL1, EXT1 and SEMA3B were studied in 28 MN cases reported at our institute during the period of 6 months from July 2023 to December 2023. Membranous Lupus Nephritis were excluded from our study.

Result:
Out of the 28 cases studied, 20 were females and 8 were males. Age range :10-71 years.

Target Antigen	Positive cases (number & %)
PLA2R	6 (21%)
NELL1	13(46%)
EXT-1	1(3.5%)
THSD7A	0(0%)
Semaphorin 3B	0(0%)

Out of 13 NELL1 positive cases, 4 had alternate medications and 5 had use of fairness creams. EXT-1 positive case was later diagnosed to have SLE. None of the patients had history of malignancies. No dual positivity was noted. All 5 antigens were negative in 8 cases (28%).

Conclusion:
MN associated with specific antigens likely represent distinct diseases. Evaluation of secondary causes of MN need to be more exhaustive in NELL1 associated MN as many cases had use of complementary and alternate medicine and none of them had any malignancy. Further studies are required to understand the pathophysiology, response to treatment, and outcomes of MNs.

Study of NELL1 in PLA2R/THSD7A negative Membranous Nephropathy: Pilot study

Komal 1 , Trishla Jadon 1 , Sheeba Kalam 1
Adarsh Barwad 1 , Soumita Bagchi 2, Deeksha Mittal 2 , Aditi Sinha 3, Arvind Bagga 3 , S K Aggarwal 2 , Amit K Dinda 1 , Geetika Singh 1
Departments of 1 Pathology, 2 Nephrology and 3 Pediatrics, All India Institute of Medical Sciences, New Delhi – 110029

Background: Two target podocytic antigens PLA2R and THSD7A, account for approximately 76.2% of primary MN (PMN) in the Indian population. In the remaining, target antigens remain unknown. Recently, many novel antigens have been identified with distinct clinicopathological features.

Aim: We aimed to study the novel antigen: Neural epidermal growth factor like protein 1 (NELL1) in PLA2R and THSD7A negative primary membranous nephropathy and correlate with clinical profile.

Methods: Between Jan 2020 and Oct 2023 cases with a diagnosis of primary membranous nephropathy, negative for PLA2R and THSD7A with adequate tissue for further staining were included. Paraffin immunofluorescence was performed for NELL1. Western blot assay in non-reducing conditions was done for NELL1 positive patients with available sera.

Results: 81 out of 125 primary MN cases were PLA2R/THSD7A negative and had sufficient tissue for further staining. 12% (15/125) of all PMN cases were positive for NELL1. 23.4% (15/64) of the PLA2R/THSD7A negative PMN cases were positive for NELL1 with mean age of 41 years, classic MN IF and G1 and G4 as dominant IgG subclasses without significant history of malignancy or traditional indigenous medicine intake. Three patients showed sera reactivity for anti-NELL1 antibody by Western blotting.

Conclusion: It is important to stain with an extended antigen panel in cases of MN to determine target antigens with possible future targeted therapy options.

ORIGINAL STUDY

A study on expression of newer antigens in membranous nephropathy

Dr. Anuradha Deole, Dr. Swarnalata Gowrishankar
Department of Histopathology, Apollo Hospitals, Jubilee Hills, Hyderabad

Background: Membranous nephropathy (MN) is the most common cause of nephrotic syndrome in adults. Due to availability of newer techniques for antigenic detection, many novel antigens have been identified which are implicated in pathogenesis of MN, such as M type phospholipase A2 receptor (PLA2R), neural epidermal growth factor-like 1 (NELL1), etc. Majority of the cases of MN are PLA2R mediated. NELL1 has many clinical associations such as history of exposure to toxins, heavy metals, native medicine intake and malignancies.
Aim: To analyse distribution of newer antigens PLA2R and NELL1 in our cohort versus published literature, to assess demographic profile and association of each antigen and to study histologic and immunofluorescence profile of each.
Methods: A retrospective and prospective study was done on 300 histologically and immunofluorescence (IF) proven cases of MN. Immunohistochemistry (IHC) for PLA2R and NELL1 was done, and findings were assessed with regards to clinical details, light microscopy and IF.
Results: 54.3% of cases were positive for PLA2R, 25.3% for NELL and 20.3% were negative for both antigens. PLA2R positive cases were associated with higher levels of serum creatinine, haematuria and chronicity, while NELL1 positive cases had more levels of proteinuria. A significant association was noted between NELL1 positivity and exposure to exogenous agents (6 cases).
Conclusion: Our study supports the findings in literature as regards to assessment of antigens in MN and their clinical associations. This can guide the nephrologists to evaluate for underlying triggers of disease and assist in serological monitoring of disease progression.

ORIGINAL STUDY

Clinical and pathological characteristics of NELL-1-associated Membranous Nephropathy

Suruthy N1, Aravind Sekar1, Ashwani Kumar1, Sahil Bagai3, Manish Rathi2, Harbir Singh Kohli2, Raja Ramachandaran2, Ritambhra Nada1

1 Department of Histopathology, 2 Department of Nephrology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

3 Max Super specialty Hospital, Saket, New Delhi

Introduction: Membranous Nephropathy (MN) is the most common cause of Nephrotic Syndrome in adults. It is characterized by formation of immune complexes in the subepithelial region of glomerular basement membrane. PLA2R is the most common target antigen against which antibody is formed (50 to 70%). NELL1 is the second most common target antigen after PLA2R. Recent studies suggest that complementary and alternative medicine (CAM) and malignancies may play a role in the development of NELL1-associated MN. In this study, we evaluated disease association and clinical characteristics of NELL1-associated MN.

Materials and Methods : We retrospectively evaluated consecutive cases of PLA2R and THSD7A-negative MN reported from 2018 to 2022. The clinical details, renal biopsy findings, NELL1 status, and follow-up details in available cases were retrieved and evaluated.

Results : NELL1-associated MN comprised of 30% (n = 24) of PLA2R and THSD7A-negative MN (n = 80). The mean age at presentation was 43.88±15.2 years and was almost equally prevalent in males and females (M: F - 1:1.2). It was not observed in pediatric and adolescent patients (< 20 years). Proteinuria at presentation was 7.4±5.3g/24 hours and serum creatinine was1.1±0.74mg/dL. Heavy proteinuria (>10 gm/24 hours) was present in 4 patients and 3 patients had proteinuria with low serum albumin levels. Hypertension was present in 3 patients. History of CAM intake with temporal association with the disease was present in 9 patients (37.5%). Other associations were type 2 diabetes mellitus (n-3, 12.5%), autoimmune diseases (n-4,16.6%), malignancy (n-1, CML) and hypothyroidism (n-3, 12.5%). None of the patients had solid organ malignancies. Kidney biopsies showed thickened basement membrane in most except 3(12.5%). None of the patients with heavy proteinuria had interstitial foam cells. On immunohistochemistry, segmental granular capillary wall staining for NELL-1 was present in 7 (29.2%) cases; All the cases showed diffuse bright (2-3+/3) granular staining along the capillary wall for IgG, kappa and lambda light chains. C1q was positive in 3 biopsies (12.5%). Treatment status was available in 15 patients. Out of which 7 (46.7%) patients did not require treatment and 8 (53.3%) patients required treatment. On follow-up, proteinuria progressed in 3 patients and complete remission was achieved in 3. The rest of the patients had a partial response. Three cases had renal dysfunction which persisted after 6 months of follow-up.

Conclusion: NELL-1-associated MN was present in 30% of PLA2R AND TSHD7A negative MN. Associations observed in our cohort were mainly CAM intake. None had solid organ malignancy despite extensive workup as reported in the Western literature. NELL1-associated MN is different in North Indian patients when compared with the West.

ORIGINAL STUDY

Glomerulopathies with Podocyte Infolding: A comprehensive clinico-pathological and ultrastructural analysis of 35 cases

Dr. Garima Garg, Dr. Alok Sharma
Department of Renal Pathology & Electron microscopy,
Dr Lal Path Labs, National Reference Lab, Rohini, Sector-18, Delhi

BACKGROUND: Glomerulopathies with podocyte infolding (PI) are a rare group of glomerular diseases characterized by presence of spherular/ tubular/ oblong microparticles in isolation and/or in aggregates in the GBM

AIM: The study aimed to analyze the clinical, morphological & ultrastructural spectrum of these glomerulopathies

METHODS: Cases showing spherular/ tubular/ oblong microparticles (50-120nm in diameter) present in isolation and/or in aggregates and involving significant proportion of glomerular capillaries; reported between 2016 & 2023; were retrieved from the archives. Their clinical, morphological & ultrastructural features were analyzed.

RESULTS: A total of 35 cases fulfilling the ultrastructural criteria were examined. The mean age of the patients was 40.1 years (15-78 years). The Female: Male ratio was 1:0.75. The most common clinical presentation was nephrotic range proteinuria (n=27; 77.1%). An underlying autoimmune disease was found in 8 cases (22.8%). Membranous pattern Glomerulopathy was the most common LM appearance (n=31; 88.5%). The DIF findings were variable and were negative/ unusual in 19 (54.2%) cases (negative=7, low intensity IgG= 10, monotypic=2). On the basis of ultrastructural appearance, the cases were divided into 2 groups: 1) cases (n=7) showing thickened GBM with presence spherular/ tubular microparticles dispersed throughout the GBM. 2) Cases (n=28) showing predominantly discrete spherular microparticle aggregates in the subepithelial &/or intramembranous location

CONCLUSION: This study identifies a subset of glomerulopathies diagnosed exclusively on ultrastructural examination which show Membranous pattern Glomerulopathy on LM, variable including negative or unusual staining intensity/ pattern on DIF studies and presence of predominantly spherular/ tubular microparticles in the GBM on EM.

Collagenofibrotic Glomerulopathy: Comprehensive Clinicopathologic Analysis of 52 Cases of a Rare Glomerular Disease

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Background: Collagenofibrotic glomerulopathy is a rare renal disease of unknown etiology resulting from the deposition of abnormal type III collagen within the glomerulus. Case reports and only rare small case series exist in the literature.

Methods: Cases of collagenofibrotic glomerulopathy (n=52) diagnosed at our institute from 2016 to 2023 were included in the study. Detailed clinical data, laboratory workup, and morphological details were reviewed, and results were compared with other similar studies.

Results: 52 cases were found among 104,832, yielding an incidence rate of 0.05. All patients presented with proteinuria; nephrotic-range proteinuria was present in 43 (83%) patients, and 32 (61%) patients had hypertension. The average age was 42 years, with 29 (56%) males and 23 (44%) females. Most of the patients had sporadic disease occurrence; only two (4%) patients had a family history of renal disease. The average serum creatinine was 1.9±1 mg/dl. All cases showed diffuse mesangial expansion, nodule formation, and double contour and were weakly PAS positive and negative for silver stain. All DIF studies were negative. By electron microscopy, all cases showed electron-dense, banded to curvilinear collagen bundles within the mesangium and subendothelial aspect of capillary walls.

Conclusion: Collagenofibrotic glomerulopathy, a rare disease, is more frequently observed among adult Indian population. This study presents largest case series of collagenofibrotic glomerulopathy globally. When comparing across East Asian countries, a higher prevalence was noted in males in the Indian population. In Japan, an equal distribution between sexes was observed, while in China, a higher prevalence was found in females (57%). In contrast, in Western Europe, particularly in France, most cases are noted in pediatric age groups with a higher prevalence in males.

ORIGINAL STUDY

Clinico-Pathologic study of Fabry's disease- A series of cases

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Background: Fabry's disease is an X –linked lysosomal storage disorder due to excessive deposition of glycosphingolipids in vascular endothelial cells, epithelial cells and smooth muscle cells caused by genetic deficiency of alpha-galactosidase A. The hallmark of the disease is formation of myelin like inclusions in the affected cells.

Aim: The aim of this study is to highlight the clinocopathological characteristics of Fabry's in the Indian scenario.

Method: We present a series of cases between 2018-2023 retrieved from our records. The light microscopy and Electron microscopy was studied. A complete clinical history with enzymes studies were also recorded wherever available.

Results: A total of 18 out of total 89231 (0.02%) cases were retrieved of which 10 were males and 8 were females. The males presented a decade later (30-70years) than females (20-60years). Majority of the patients presented with symptoms of proteinuria (1+to 4+) with deranged renal functions. One male patient had family history of undiagnosed renal disease and hearing loss. LM examination showed segmental tuft sclerosis with fine vacuolization in podocytes, mild tubulo-interstitial chronicity and vascular hyalinosis in all patients which was further confirmed on EM examination which reveal characteristic myelin figures seen in podocyte cytoplasm.

Conclusion: Fabry's disease is a very rare disease which requires a high degree of suspicion in patients presenting with proteinuria and FSGS lesions. Careful examination of podocytes with foam cell change is the key to further evaluate for Electron microscopy requiring further confirmation by enzyme studies.

Clinicopathological spectrum and outcomes of non-lupus full house nephropathy compared to lupus Nephritis; single centre experience

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Background: Non-lupus full-house nephropathy (FHN) is "full-house" immunofluorescence pattern in patients with various histopathologic lesions in renal biopsies who do not fulfil 2019 EULAR/ACR classification criteria for diagnosis of systemic lupus erythematosus. It represents diagnostic & therapeutic challenge.

Aim: To study clinicopathological spectrum & outcomes of non-lupus FHN, in addition comparing them with lupus nephritis (LN) patients.

Methods: We retrospectively reviewed all non-lupus FHN and LN renal biopsies between January 2022 to June 2023. Clinical & laboratory data at time of renal biopsy & at end of follow-up were retrieved from our base records & compared with patients diagnosed with LN.

Results: Total 97 renal biopsies met criteria for FHN, out of them, 77 (79.38%) were diagnosed with LN & 20 (20.62%) as non-lupus FHN. Main features of patients with non-lupus FHN were male preponderance 11 (55%), mean age of 27.30 (±14.5) years, serum creatinine of 2.73 (±4.46) mg/dl, proteinuria of 3.61 (±3.83) g/day. Hypocomplementemia occurred in 12 patients (66.67%), haematuria occurred in 19 (95%) patients & Hypertension occurred in 6 patients (30%). Out of 20 non-lupus FHN, 8 (40%) presented with a nephrotic syndrome, 5 (25%) with rapidly progressive glomerulonephritis, 3 (15%) with nephritic syndrome, 2 (10%) with nephritic nephrotic syndrome & 2 (10%) with acute renal failure. Most common histopathology pattern observed was Membranoproliferative glomerulonephritis in 10 cases (50%) followed by Membranous nephropathy in 7 cases (35%), IgA nephropathy 2 cases (10%) & C1q nephropathy 1 case (5%). Compared with LN patients, non-lupus FHN patients were more often in male ($p<0.001$), significantly lesser number of patients showing hypocomplementemia ($p=0.024$) and haematuria ($p=0.01$), serum creatinine higher at presentation ($p=0.731$) & lower at follow up ($p=0.488$). Proteinuria was found to be slightly higher at presentation ($P=0.493$) in cases of non-lupus FHN compared to LN & resolved ($p=0.3999$) during follow up in both of cases.

Conclusion: Non-lupus FHN is an idiopathic form, affecting mainly males, with broad spectrum of glomerular histological findings & this showing good outcome. Clinical outcomes of non-lupus FHN & LN are not statistically significantly different.

ORIGINAL STUDY

HISTOLOGICAL SPECTRUM OF RENAL LESIONS NOTED IN CONNECTIVE TISSUE DISORDERS EXCLUDING LUPUS"

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BACKGROUND: CTDs are a heterogeneous group of disorders with systemic involvement due to dysregulated and disturbed immunoregulation, leading to autoantibody production and tissue injury, affecting lung and kidney as the disease progresses. Varied clinical manifestations are seen depending on severity and duration, and are a major cause of long-term morbidities requiring specific therapeutic interventions.

AIM: To study the histopathological spectrum of renal lesions in CTDs excluding lupus.

METHODS: Renal biopsy cases were reviewed retrospectively between 2018 to 2023, with patient demographic data, lab investigations, light microscopy and IF findings. Patients with SLE were excluded from the study.

RESULTS: 15 cases of CTDs were retrieved from the archives, 9 cases were included in the study with a mean age of 40 years and female predominance, and 6 cases were excluded due to an overlap association of lupus. All cases had raised serum creatinine levels, ANA positivity seen in 6 cases and 1 case with anti-U1RNP positivity. The cases included Sjogren's syndrome (n=4), SS (n=2), RA (n=2) and dermatomyositis (n=1). Light microscopy showed varied morphological spectrum with MPGN pattern of injury (n=4), tubulointerstitial nephritis (n=3), membranous nephropathy (n=1) and FSGS pattern of injury (n=1). IFTA was severe (n=2), mild (n=6) and moderate (n=1) respectively. Majority of the cases showed atherosclerotic changes in the vessel wall, independent of the age. No full house pattern was noted on IF. Codominant expression of IgG and C3 was seen in 1 case (n=1) and IgA and C3 was seen in 1 case (n=1). No extraglomerular deposits in tubules and blood vessels was seen in any of the cases, unlike lupus.

Table: Spectrum of renal lesions in CTDs

Sl. No.	CTD	Morphology	Degree of arteriosclerosis
1	Sjogren's syndrome	Acute tubulointerstitial nephritis	Moderate
2	RA	Membranous nephropathy	Mild
3	RA	MPGN	Mild
4	SS	MPGN	Mild
5	Sjogren's syndrome	MPGN	Mild
6	Sjogren's syndrome	Chronic tubulointerstitial nephritis	Mild
7	SS	FSGS	Moderate
8	Dermatomyositis	MPGN	Mild
9	Sjogren's syndrome + RA	Acute tubulointerstitial nephritis	Moderate to severe

CONCLUSIONS: Renal manifestations in CTDs are a frequent presentation with varied morphological spectrum and IF findings in renal biopsies. Such lesions aid the clinician in decision-making for therapy options and in predicting prognosis. A renal biopsy is essential to ensure proper diagnosis with an interdisciplinary approach to optimize treatment in patients with CTDs.

ABBREVIATIONS: CTDs: Connective tissue disorders; RA: Rheumatoid arthritis; SLE: Systemic lupus erythematosus; SS: Systemic sclerosis; MPGN: Membranoproliferative glomerulonephritis; FSCG: Focal segmental glomerulosclerosis; IFTA: Interstitial fibrosis and tubular atrophy; IF: Immunofluorescence

ORIGINAL STUDY

Petite IgA nephropathy versus hefty C3 glomerulopathy glomeruli: A case series on glomerular size difference between IgA nephropathy, C3 glomerulopathy and membranous nephropathy in native renal biopsies.

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Background: Glomerular diseases have divergent morphologic spectrum with variable glomerular basement changes or matrix expansion or proliferation. Since, due to delayed referrals for biopsies or patients trying alternate therapies before reaching referral centres, biopsies of our patients show extensive glomerular sclerosis. Can such glomeruli give us some diagnostic clue to diagnoses as Immunofluorescence in sclerosed glomeruli can be difficult to interpret. A significant parameter in the non-sclerosed glomeruli gives a diagnostic clue in IgAN (IgA nephropathy), C3G (C3 glomerulopathy) and MN (membranous nephropathy).
Aim: Morphometric analyses of non-sclerosed glomeruli of IgAN, C3G and MN to compare MGD (Maximum glomerular diameter), Mean GA (Glomerular area) and MGV (Maximum glomerular volume).
Method: A retrospective analysis over a period of year was performed. Biopsies of IgAN, C3G and MN were recruited, with age matched controls (more than 18 years). Native renal biopsies with more than 5 glomeruli and IFTA less than 25% were included.
Results: A total of 48 cases were recruited in this study, of which 27 were IgAN, 13 were C3G and 8 were MN. In C3G, MN and IgAN the MGV was 11±7, 9±3 and 6±2 x 106 um3 respectively with the p-value of 0.001. Similarly, the Mean GA among the non-sclerosed glomeruli were 2.4, 2.3 and 1.9 x 104 um2 respectively with the p-value of 0.001. The MGD among C3GN and IgAN was 196.32± 5.44 and 175.50±39.47 um with the p-value of 0.001.
Conclusion: The non-sclerotic glomeruli of C3G were significantly larger than glomeruli of IgA and MN. C3G is another cause of enlarged failed kidneys like diabetes and amyloidosis.

Clinico-histopathological Spectrum of Renal Biopsy in pediatric patients: A Single Centre Study

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BACKGROUND: Renal Biopsy is a well-known diagnostic tool for determining kidney diseases in paediatric patients and helps in selection of precise therapeutic approaches for several renal diseases.

AIM: To study the clinical indications and histopathological spectrum of renal diseases in children in a tertiary care centre.

METHOD: This retrospective study includes patients (age ≤ 18 years) undergoing native renal biopsies from January 2022 to December 2023. The clinical profile, laboratory parameters & renal biopsy findings were recorded from the Departmental Data.

RESULTS: Out of 1039 renal biopsies, 217 (20.1%) were performed on the paediatric patients. Mean age was 10.8 ± 4.9 years, Male: Female Ratio was 1.1:1. The commonest indication for biopsy was Nephrotic Syndrome (NS) (58.06%) followed by Rapidly Progressive Renal Failure/Acute Kidney Injury (RPRF/AKI) (14%), Urinary abnormality (11.05%).and Lupus nephritis (LN) (11.5%).

On histopathology, 81.1% patients had primary glomerulonephritis, 12.4% had secondary GN, 4 % had tubulo-interstitial and 3 % had vascular disease. Minimal Change Disease (29.5%) was the commonest findings in primary GN followed by Focal Segmental Glomerulosclerosis (20.4%) and Crescentic Glomerulonephritis (9.09%). Lupus nephritis (11.9%) followed by Amyloidosis (0.5%), were the commonest secondary GN. MCD and Lupus nephritis were most common findings of NS and RPRF/AKI respectively.

CONCLUSION: The commonest indication for renal biopsy in paediatric was nephrotic syndrome. Minimal Change disease was the commonest histopathological diagnosis in glomerular diseases.

KEYWORDS: Paediatric, Nephrotic Syndrome, Renal Biopsy.

ORIGINAL STUDY

Spectrum of renal disease in elderly population above 65 years of age - 1 year study of 425 cases

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Introduction: Renal biopsy is considered as the gold standard for a diagnosis of renal parenchymal disease. Paucity of information on renal disease in elderly exist in Indian literature.

Aims: To study spectrum of renal disease in elderly population.

Methods: prospective study conducted for a period of 1 year 01/01/2023 to 01/01/2024.Clinical data and lab parameters were collected from records.

Results: Out of 5843 renal biopsies during 1 year of study period, 425 (7.3%) were elderly. Males were predominantly affected 294 (69.17%), females 131 (30.8%), M: F 2.24:1. Majority of patients were in 7th decade 246 (57.8%) followed by 165 (38.8%), 12 (2.8%), 2(0.4%) in 8th, 9th and 10th decade respectively. Clinical indication for biopsy were nephrotic syndrome in 162 (38.11%), chronic kidney disease 77 (18.11%), AKI 62 (14.58%), rapidly progressive renal failure 54 (12.7%), nephritic syndrome 35 (8.2%) and sub nephrotic range proteinuria 21 (4.94). Most of the patient had renal dysfunction 259 (60.9%). Associated comorbidities were diabetes mellitus 194 (45.6%), hypertension 243(57.17), both 148 (34.8%) , Glomerular diseases seen in 206 (48.4%) cases: podocytopathies 51 (12%) was common followed by , membranous glomerular nephritis 42(9.8%) , IgA 20(4.7%) , C3 GN 18(4.23 %), amyloidosis 17 (4%) , immune complex mediated glomerulonephritis 16 (3.7%) , pauci immune 15 (3.5%), CGN 7 (1.64%), LCDD 5(1.17%), MIDD 3(0.7%) ,), anti GBM 3(0.7%) , PGNMID 2 (0.4%) , diffuse mesangioproliferative proliferative glomerulonephritis 2(0.4%),MPGN 2 (0.4%), lupus nephritis 1(0.23%) , glomerular organoid deposit disease 1 (0.23%).Tubulointerstitial disease were seen in 121(28.47%) cases :majority were chronic interstitial nephritis 45(10.58%) ,followed by ATN 20 cases (4.7 88%), LCCN 15 (3.52%) , pigment nephropathy 14 (3.29%), tubulointerstitial nephritis 14 (3.29%), pyelonephritis 7(1.64 %), papillary necrosis 2(0.4%), cortical necrosis 2(04%), scar 2(0.4%). Vascular disease were 7, TMA 5 (1.17%), vasculitis 2(0.4%), Diabetic patients 194 (45.64 %) underwent biopsy ,74(17.44%) had diabetic nephropathy, 77(39.69 %) non-diabetic renal disease and 43 (27.3%) both Transplant biopsies 14 cases, majority had ATN 8(1.88%). bACR 3(0.7%), BKV 1(0.23), acute T cell mediated rejection 1(0.23%), pyelonephritis 1(0.23%)

Conclusion: Renal disease studied in our population formed 7.3%; with varied morphological spectrum. Patients presenting with glomerular symptomatology have either podocytopathy or MGN in majority of them. Patients with plasma cell dyscrasia 15 were clinically suspected and diagnosed ,37 cases were clinically unsuspected and diagnosed work up was done later.

Unusual Electron microscopy findings in kidney biopsies during COVID pandemic period

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BACKGROUND- Tubuloreticular inclusions (TRIs) are phospholipids containing subcellular structures found in the cytoplasm of endothelial and lymphoreticular cells. They have a strong association with systemic lupus erythematosus (SLE) and human immunodeficiency virus-associated nephropathy (HIVAN).

METHODS- We received total of 2531 kidney biopsy samples for electron microscopy (EM) in 2021- 23. 78 cases with kidney biopsies with TRIs were included in our study after cases of SLE and HIV were excluded and examined further. Serological positivity for Hepatitis B & C were negative. Serum samples were available in 29 cases for which parvovirus and COVID antibodies were performed.

RESULTS- On light microscopy, a variable diagnoses of minimal change disease, IgA nephropathy, focal sclerosing glomerulonephritis were rendered on the kidney biopsy. Parvovirus antibodies analysed showed 49% IgM positivity and 55% IgG positivity. 3% and 17% cases showed IgM and IgG positivity for COVID antibodies respectively. Number of biopsies with TRIs showed a bell-shaped curve peaking in the last half of 2022 and showing recession of number as the Covid epidemic abated.

CONCLUSION- Our cases highlight the incidental finding of TRIs during the Covid epidemic. Covid antibodies were negative in serum samples in these biopsies. A high incidence of parvovirus infections was noted suggesting a possible association of viral disorders in the setting of decreased immune status. In conclusion, we note that the presence of TRIs is an indicator of systemic interferon activity with the possible association of parvovirus infections and SARS-CoV-2 infection during the COVID pandemic.

ORIGINAL STUDY

Diaspora Of Glomerular Diseases With Organized Deposits In A Tertiary Diagnostic Centre In Northern India

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Background
Glomerulopathies with organized deposits is an all-encompassing expression for entities like amyloidosis, cryoglobulinaemic glomerulonephritis, fibrillary glomerulonephritis, immunotactoid glomerulopathy, collageno-fibrotic glomerulopathy, fibronectin glomerulopathy, monoclonal Immunoglobulin deposition disease, cast nephropathy, and diabetic fibrillosis. Increased use of electron microscopy has led to increased frequency of diagnosis of glomerular deposition diseases.

Aim
To observe the spectrum of glomerular diseases with organized deposits in renal biopsy specimens using light microscopy, Immunofluorescence and electron microscopy

Methods
Kidney biopsies diagnosed as glomerular diseases with organized deposits on electron microscopy were pulled out from the records of GIPMER, New Delhi over a period of 8 years. They were further classified and analysed according to the morphological nature of these deposits on light, immunofluorescence and electron microscopy into various categories.

Results
A total of 10,800 renal biopsies were received in our department over a period of 7 years. Of these, 237 biopsies were diagnosed as glomerular diseases with organized deposits on electron microscopy (2.2%). Cases of Amyloidosis (55.3%), MGRS (22%), Cast nephropathy (9.3%), Cryoglobulinaemia (3.3%), Fibrillary glomerulonephritis (2.5%), Diabetic Fibrillosis (1.7%), Immunotactoid and Fibronectin glomerulonephritis and Dense deposit diseases (5%) were also diagnosed. Cases of Collagenofibrotic glomerulopathy were not identified.
Deposition diseases presented most commonly in the 4th and 5th decade of life. Most patients presented with nephrotic syndrome or acute renal dysfunction.

Conclusions
The spectrum of renal deposition disease as presented in this study highlights the spectrum of deposition diseases in our country. It emphasises the use of electron microscopy as a routine diagnostic tool.

Morphological spectrum of plasma cell dyscrasia -1 year study of 60 cases.

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Introduction:
Para proteins are the monoclonal immunoglobulins produced by malignant clones of plasma cells or B cells. These proteins are associated with spectrum of kidney disorders by direct and indirect effect. In recent decade there is increase in prevalence of renal disease associated with Para proteins. MGRS is well defined syndrome entity describe in last decade.

Aim: To study spectrum of plasma cell dyscrasia

Methods: It is prospective study conducted for a period of 1 year 1/1/2023 to 1/1/ /2024. Clinical data and laboratory parameter of diagnosed cases were collected from the files

Results: Out of 5843 renal biopsies during 1 year of study period 60 (1.02%) cases were plasma cell dyscrasia. Males were most affected - 42 (70.0%) female 18 (30%), male to female ratio 2.3:1. Age group ranged from 31-40 years 2cases (3.33%), 41-50 years 01case (1.6%), 51-60 years 17 cases (28.3%), 61-70 years 26 cases (43.3%), 70-80 years 14cases (23.33%). The clinical presentations were nephrotic syndrome -27 (45.0%) rapidly progressive renal failure - 14 (23.33%), acute kidney injury 9 (15%), chronic kidney disease 6(10 %), acute on chronic kidney disease 4 (6.66%), Renal dysfunction was seen in 29 cases (48.33%). History of non-plasma cell dyscrasia neoplasm was seen in 5 cases (8.33%) (2 cases of mantel cell lymphoma, follicular cell lymphoma, renal cell carcinoma, chronic lymphoid leukaemia). clinical manifestations suggestive of plasma cell dyscrasia (PCD) were seen in 17 cases (28.33%). M band was seen 9 cases (14.06%). The most common disease was amyloidosis 22 (36.66%), followed by light chain cast nephropathy 17(14.06%), light chain deposition disease 9 (14.06%), monoclonal immunoglobulin deposition disease 6 (10 %),proliferative glomerulonephritis with monoclonal immune deposit disease 6(10 %).

Conclusion: Plasma cell dyscrasia (PCD) represents 1.02% in our population, with male predominance.
Direct immunofluorescence is pivotal in diagnosis of PCD. Renal biopsy is one of the gold standards in assessment of PCD with 17 cases had renal clinical manifestation and were diagnosed, 43 cases had no clinical suspicious.

ORIGINAL STUDY

Histopathological spectrum of monoclonal renal disorders in a Tertiary center in South West Rajasthan.

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Background: A diverse spectrum of kidney diseases is associated with monoclonal immunoglobulin deposits; caused by abnormal paraprotein secreted by clonal B cells. The disease manifestations range from tubulopathies to glomerular diseases with varying degrees of proteinuria and renal dysfunction. Herein, we present clinico-pathological features of monoclonal renal disorders diagnosed in our institution.

Aim: To study the spectrum of lesions with monoclonal immunoglobulin deposits in the South West region of Rajasthan.

Methods: All cases of monoclonal immunoglobulin deposits from 2019-2023 were included.

Results: A total of 16 cases of monoclonal renal disorders were identified. Mean age of patients was 48.1 years (age range: 30-70 years) with M:F ratio of 1:3. Renal insufficiency was present in 14 (87.5%) cases with mean creatinine (5.52 mg/dl) and nephrotic syndrome was present in 10 (62.5%) with mean proteinuria (8.34g/24 hour). Acute kidney injury was commonest presentation with 7 (43.75%) and progressive renal failure was noted in 4 (25.0%). On microscopy, MIDD and LCCN with 37.5% (6/16) cases each were commonest, followed by PGNMID (18.75%, 3/16). Type 1 cryoglobulinemia and LCPT comprised of 12.5% (2/16) cases each. Direct infiltration by neoplastic plasma cells was observed in 2 (12.5%) cases. Two or more than two monoclonal lesions were noted in 3 cases (18.75%) and one case of LCCN had coexistent lanthanic IgA deposits. Monoclonal protein was detected in 5 (31.25%) cases on SPEP, UPEP and/or serum immunofixation or SFLC. Bone marrow was performed in 13 cases and showed clonal plasma cells in 5 (31.25%) cases. 100% of cases with PGNMID and type 1 cryoglobulinemia had normocellular marrow. On immunofluorescence, kappa light chain predominance was noted in 9 (56.25%) cases, followed by lambda light chain in 5 (31.25%), and one case had heavy chain deposits. One case had possibility of masked deposit which could not be characterized on immunofluorescence, however electron microscopy revealed powdery electron dense deposits on the subendothelial areas.

Conclusions: Paraprotein related kidney diseases include a wide spectrum of pathologic lesion. Around one fourth of the patients who underwent bone marrow biopsy after initial kidney biopsy had plasma cell dyscrasia. Kidney biopsy is essential to establish the specific diagnosis, therapeutic, and important prognostic information before cytotoxic treatment.

***Abbreviation:** MIDD: Monoclonal Immune Deposition Disease, LCCN: Light Chain Cast Nephropathy, PGNMID: Proliferative Glomerulonephritis with Monoclonal Immune Deposits, LCPT: Light Chain Proximal Tubulopathy, SPEP: Serum Protein Electrophoresis, UPEP: Urine Protein Electrophoresis, SFLC: Serum Free Light Chain Assay.

Conventional immunohistochemical panel of serum amyloid A, kappa and lambda is not a very sensitive tool for typing amyloid deposits in renal amyloidosis.

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Background- Amyloidosis is a group of protein misfolding disorder characterized by the deposition of extracellular eosinophilic substance in various organs, causing progressive organ dysfunction. AL, AA, and several of the hereditary amyloidosis such as AFib, ATTR, etc. are known to involve kidney.

Aim: We aimed to characterize the type of amyloid deposits in renal biopsies using conventional immunohistochemical panel of serum amyloid A, kappa and lambda.

Materials and methods: All cases of renal amyloidosis in past 3 years (Jan2019-Dec2023) were retrieved. Relevant clinical details were collected from the histopathology requisition form. All cases were stained for primary antibodies against serum amyloid A, kappa and lambda. Direct immunofluorescence study was performed as a routine diagnostic procedure.

Results: A total of 14 cases were diagnosed with renal amyloidosis. The mean age of patients was 56 years (age range 25-78 years) with male predominance. Renal insufficiency and nephrotic syndrome were present in 71% cases. Typing of amyloid deposits was possible in 8 cases, and all cases had AA amyloidosis. No case of AL amyloidosis was detected. Predisposing factor for AA amyloidosis was noted in all cases (5 tuberculosis, 1 opioid addict & 2 COPD). Associated pathologic lesions include moderate to severe acute tubular necrosis in 5 cases.

Conclusion: Conventional panel of IHC comprising of SAA, kappa and lambda correctly typed amyloid deposits in only 57% cases. Most common form of amyloid deposits is AA amyloidosis. Tuberculosis is the most common predisposing factor. Proteomic studies are needed for further characterization.

ORIGINAL STUDY

Histomorphology of kidney in thrombotic microangiopathy-a case series study

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Back ground:
Thrombotic microangiopathy (TMA) refers to a condition characterized by microvascular endothelial injury and thrombosis. It can present as generalized form with multisystem involvement, or can be renal limited (rTMA). Both carry a poor prognosis. Primary causes of TMA include inherited conditions like mutation in ADAMTS13 (TTP) and deletion of CFHR3/CHFR1. Secondary causes include Infections like Hemolytic uremic syndrome and covid-19, Preeclampsia/ HELLP syndrome, malignancy, post bone marrow transplantation, certain drugs, malignant hypertension, SLE, HIV etc.

Case Report:
Study sample was collected from January 2020 to December 2023, and includes 23 native kidney biopsies and 6 graft biopsies. Male predominance was noted in our study. The most common etiology was malignant hypertension followed by infections, especially Covid 19. A single case of atypical HUS identified characterized by deletion of CFHR3/CFHR1 gene. The features in renal biopsy were glomerular changes like cortical necrosis, fibrin thrombi and mesangioproliferative pattern. Only one case showed crescent. Arteriolar changes included fibrinoid necrosis, mucoid change of intima and onion-skin like lesions, which were mostly seen in cases of accelerated HTN. Among the graft biopsies, two cases were due to antibody mediated rejection, two due to CNI toxicity and rest because of donor kidney disease.

Conclusion:
Even though renal limited TMA is a pathologic diagnosis, etiology cannot be distinguished based on pathological findings alone. It is important to identify TMA even in the absence of features of hemolysis and thrombocytopenia, as it is associated with significant morbidity and mortality.

Renal manifestations of Thrombotic Microangiopathy across diverse disease spectra

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Background: Thrombotic microangiopathy (TMA) is a condition wherein small blood vessels develop clots, leading to decreased platelets and red blood cell destruction, causing organ damage. These include HUS, HTN, pregnancy, lupus, pancreatitis, and post-transplant complications like rejection or drug toxicity.

Aim: To study the frequency and manifestations of TMA across diverse disease spectrum

Method: We assessed (n=43) TMA cases between 2019 and 2023 with diverse clinical presentations, categorizing them based on causes or related clinical features, laboratory parameters, and histopathology. Relevant data were assessed and evaluated.

Result: The study includes patients aged 7 to 54 years, etiology identified in all 43 cases which included, 19 (45%) cases of preeclampsia/postpartum females, 10 (24%) of hypertension, 7 (17%) cases had malaria-associated TMA, 2 (4%) cases had history of drug abuse, 2 (4%) cases of transplant-associated TMA, 1 (2%) case of HUS, 1 (2%) case had of snake bite, and 1 (2%) case with Alport syndrome associated TMA.

On analysing laboratory parameters, (43, 97%) cases presented with renal insufficiency with raised serum creatinine (mean 4.9). Haematuria was present in 13 (30%) cases. Moderate increase in proteinuria was noted in 29 (67%) cases and severe in 10 cases (23%). Blood urea nitrogen was raised in 35 (81%) of cases. LDH was raised in 39 (90%) cases. ANA positive in 3 cases (6%). Low C3 complement level was observed in 7 (16%) cases.

On microscopy, both acute and chronic changes of TMA in glomeruli, in the form of cortical necrosis, micro-thrombi, mesangiolysis, GBM splitting and sclerosis was observed. Vascular changes in the form of fibrinoid necrosis, thrombi, medial hypertrophy, and hyaline arteriosclerosis were seen. Two postpartum females had diffuse cortical necrosis (10.5%). Patchy cortical necrosis was present in 10 cases of postpartum TMA (52.6%). Fibrinoid necrosis was seen in 10 cases. Malaria-associated TMA displayed the presence of malarial schizonts.

Conclusion: Kidney is commonly affected in TMA. Knowledge of various histopathological features in diverse clinical spectrum is necessary. This study underscores the necessity of renal biopsies in cases where clinical indicators and low levels of biochemical parameters like creatinine, LDH, and BUN do not prompt clinical suspicion, highlighting the importance of histology in confirming TMA.

Abbreviations: Thrombotic microangiopathy- TMA, Haemolytic uremic syndrome – HUS, hypertension- HTN, LDH- Lactate dehydrogenase

ORIGINAL STUDY

CLINICOPATHOLOGICAL SPECTRUM OF ANCA ASSOCIATED GLOMERULONEPHRITIS: EVALUATING THE EFFECT OF ASSOCIATED VASCULITIS

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Background: ANCA associated Glomerulonephritis (AAGN) is an uncommon GN with characteristic necrotizing, crescentic morphology with few to no immune deposits on IF. It most commonly occurs in elderly patients, with vasculitis in 5-35% cases.

Aim: To study frequency of vasculitis in AAGN and its clinicopathological spectrum.

Methods: We performed a retrospective analysis of medical renal biopsies diagnosed between January 2018- January 2024 at our institute. All the cases showing positive serology for ANCA on ELISA were included (total 27 cases). Relevant data was collected and clinical, histopathological and IF findings were evaluated.

Results: Mean age of patients was 46.6±21.3 years with M: F ratio 1.1:1. Most patients presented with RPRF (mean serum creatinine: 4.83±3.74 mg/dl) and sub-nephrotic proteinuria (mean 24 hr proteinuria:2.29±1.44 g/dl). MPO-ANCA was positive in 44% (n=12), PR3-ANCA in 49% (n=13) and both in 7 % (n=2) of the cases. 17 (63%) were associated with hypertension, two (7.4%) with type 2 DM and one (3.7%) showed associated malignancy. Seven patients (27%) had features of vasculitis. No immune deposits were noted for 26 cases, one case with positive serology for anti-GBM antibodies showed linear deposits for IgG on IF. In patients with vasculitis, the mean creatinine levels (7.67±4.76 mg/dL vs 3.77±2.48 mg/dL) ($p<0.0005$), and average percentage of crescents and fibrinoid necrosis of the tuft relative to total glomeruli were higher (65.6% ±23.69 and 32.67± 23.04 vs 32.67% ± 23.03 and 27.78 ± 24.33% respectively) ($p=0.008$ and 0.005) and a higher BVAS score while total percentage of normal glomeruli were lower as compared to other group (2.77% vs 35.3%) ($p= 0.04$). Most cases presented with cellular to fibrocellular crescents (93%), and fibrous crescents seen in 4 (18.5%) cases.

Conclusion: Patients with vasculitis have higher mean creatinine value at the time of presentation, lower percentage of normal glomeruli and higher percentage of crescents and fibrinoid necrosis of tuft. Thus, AAGN with vasculitis is associated with severe renal impairment and disease activity affecting the treatment and prognosis in such patients.

Abbreviations: ANCA- Anti neutrophilic cytoplasmic antibody, AAGN -ANCA associated glomerulonephritis; IF- Immunofluorescence; RPRF; MPO- Myeloperoxidase; PR3- Proteinase 3; DM- Type 2 Diabetes Mellitus; BVAS- Birmingham Vasculitis score.

Antiphospholipid syndrome nephropathy in patients with antiphospholipid antibodies and the renal outcome. A series of cases.

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Background: Presence of anti-phospholipid antibodies results in arterial venous thrombosis in various organs. They are mostly present in auto-immune diseases like SLE. The renal manifestation of APLA are rare event but have potential to impact the renal outcomes.

Aim: To study the histological findings of renal injuries in APLA positive patients and to correlate with renal outcome.

Methods: A total of 13 cases were studied which were serologically proven to be APLA positive between 2017 to 2023. Biopsies were reviewed and clinical records from nephrology and rheumatology division were obtained.

Results: There were 13 APLA positive patients in 5 years. 12 of the 13 cases were associated with systemic lupus erythematosis except for one who had an association with hepatitis A infection. Of the 13 cases, 12 were female and the mean age of the patients was 35 years. The renal biopsies were reviewed and an attempt was made to diagnose APSN and correlate the morphological findings with the outcome after 6 months. 8 patients were diagnosed with Lupus nephritis (class II-1, class III-1, class III+V-1, class IV+V-1, Class V-4). The morphological findings was a spectrum which included those without significant changes in the glomerular or vascular compartment despite APLA positivity, to those with acute and chronic glomerular and vascular changes of thrombotic microangiopathy resulting in double contours of glomerular basement membrane termed as antiphospholipid syndrome nephropathy. Follow up data was available for 8 patients, from a minimum of 4 months to a maximum of 11 months. The cases with no significant glomerular and vascular changes showed a significant response, where as one of the cases which showed acute changes of thrombotic microangiopathy showed only a partial response after 11 months.

Conclusion: The importance of this study was to highlight the significance of APSN in APLA positive cases, and their impact on renal outcomes.

ORIGINAL STUDY

Kidneys not always small and contracted in CKD??- Reasons explored

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Introduction: Chronic kidney disease and contracted kidney size are almost synonymous except in cases of renal amyloidosis and diabetic nephropathy. This study aims to explore the causes of non-contracted kidneys in patients with chronic kidney disease.

Materials and methods: We prospectively recruited forty-nine adult patients (n=49) over a period of 1.2 years who are known cases of chronic kidney disease. Renal volume was assessed by imaging including ultrasonogram and computer tomography. Renal biopsy was performed in all the cases for presumed co-existing ATI. Sections were stained with hematoxylin & eosin, Periodic acid Schiff and Jones methenamine silver stains for histopathological diagnosis. Further, severity of ATI and chronicity scores were assessed and graded. Results were correlated with renal volume and size.

Results: On histology, glomerular pathology was diagnosed in 30 patients (including 2 cases of DN), tubulo-interstitial pathology in 15 patients and 4 patients had glomerular and tubulo-interstitial pathology. Chronicity score was also evaluated in 41 cases by assessing percentage of sclerosed glomeruli, interstitial fibrosis, tubular atrophy and arterial changes. Interstitial fibrosis and tubular atrophy (nephron loss pattern) associated with sclerosed glomeruli was present in 7 cases (17%). Superimposed moderate to severe ATI was seen in most cases; severe ATI in 30 cases (61.2%), moderate ATI in 14 cases (28.5%), and the rest had mild ATI. Acute tubular injury was associated with interstitial oedema in 35 cases (71%). Out of all the patients of chronic kidney disease (irrespective of chronicity score) with acute tubular injury, none had small contracted kidney size.

Conclusion: Co-existing acute tubular injury in chronic kidney disease patients results in enlarged kidneys defying the general rule of CKD & small contracted kidneys

A study of mast cells in T-cell mediated rejection: Their correlation with histology and clinical outcomes

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BACKGROUND:
Kidney allograft cellular rejection remains a significant challenge in renal transplantation pathology, necessitating a deeper understanding of immune cell dynamics within the allograft microenvironment. Mast cells (MCs), known for their role in inflammation and immune modulation, through interaction with regulatory T cells, effector T cells, B cells, by degranulation of cytokines and other mediators and have recently emerged as potential contributors to rejection processes. Data on correlation between MCs counts with other criteria of rejection and treatment response is scanty.

AIM:
This study aimed to investigate the presence and significance of MCs in cellular rejection, with a focus on their correlation with other parameters such as creatinine levels, interstitial inflammation, tubulitis, tubular atrophy, interstitial fibrosis and their potential role as prognostic markers and contributors in graft pathology.

METHODS:
Renal biopsy samples from patients with confirmed cellular rejection were analysed for MCs infiltration using immunohistochemically mast-cell specific marker CD117 and semi quantitating by whole slide digital scans.
Clinical data, including patient outcomes and response to treatment, pathological parameters, were correlated with mast cell quantification.

RESULTS
A preliminary analysis of the results shows a varying correlation of MCs infiltrate with the histologic features of rejection and the outcome.

CONCLUSION
This study provides evidence of mast cell involvement in renal cellular rejection, suggesting a potential role in the pathogenesis of rejection episodes. Mast cell quantitation may serve as a valuable prognostic marker. Targeting mast cell pathways could represent a novel therapeutic approach to improve outcomes in renal transplant recipients experiencing cellular rejection.

ORIGINAL STUDY

PSMP EXPRESSION AND SPATIAL DISTRIBUTION OF IMMUNE CELL INFILTRATES IN THE ALLOGRAFT BIOPSIES WITH TRANSPLANT GLOMERULOPATHY

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Background: Transplant glomerulopathy is the most common lesion observed in chronic allograft injury due to repetitive endothelial cell damage. Antibody-mediated rejection, thrombotic microangiopathy, hepatitis C virus infection and T-cell-mediated rejection are some of the causes of Transplant glomerulopathy. Type and spatial distribution of immune infiltrates in chronic rejection was lacking though described in acute rejection cases. Further, there is a need for an immunomarker to distinguish Chronic active antibody-mediated rejection from chronic inactive antibody-mediated rejection. PSMP is a cytokine and plays a crucial role in the pathogenesis of chronic active antibody-mediated rejection.

Aims and objectives: Determine the distribution of immune infiltrates and PSMP/MSMP expression in the transplant glomerulopathy cases of different aetiologies.

Materials and methods: A retrospective and prospective study with all cases of allograft biopsies with findings of TG. Immunohistochemistry for CD3, CD20, CD4, CD8, CD68, CD163 and PSMP was done.

Results: 80 cases with findings of transplant glomerulopathy in the allograft renal biopsy from 2016-2022 were selected. CD163+ macrophages (58.6%) were the predominant inflammatory cells in the glomerulus in Chronic active antibody-mediated rejection cases, followed by T lymphocytes (20.69%) and B lymphocytes (10.3%). CD8 subset of T-lymphocytes was dominant in the glomerulus. The admixture of B, T-lymphocytes, and type II macrophages are dominant in the fibrosed tubulointerstitial compartment. CAAMR with C4d positivityCAAMR with C4d negativityChronic inactive ABMRPSMP expression64.4%41.3%12.5%Number of cases showing positivity8 out of 175 out of 123 out of 10p value0.010.57750.53

There was a significant association between CD163+ macrophage infiltration and PSMP expression in cases with CAAMR.

Conclusion: PSMP is an immunomarker that can determine the presence of ongoing activity in transplant glomerulopathy cases for deciding additional immunosuppressive drug administration.

HLA allele diversity and their representation in commercially available SAB (Single Antigen Bead) assay panel- A single centre study from Western India

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Background-
HLA gene is the most polymorphic gene in the human genome. There is wide allele variation across different genetic backgrounds and ethnicities. It's most common clinical application is for organ matching before transplantation. Preformed Donor Specific Anti HLA Antibodies may be present in sensitized individuals and can result in ABMR. SAB assay helps in detection and quantification of Anti HLA Antibodies.

Aim-
To study the allele diversity of the study population and to look for their representation in commercially SAB assay panel and understand its impact on PRA with purpose of using virtual crossmatch for renal transplantation.

Methods-
This is a retrospective study done in a tertiary care hospital in Gujarat, India. 1000 individuals (500 donor-recipient pairs for renal transplantation) underwent HLA typing for 6 loci- A, B, C, DRB1, DQB1, DQA1 as per institutional pre-transplant work-up protocol. As per the same protocol, every recipient underwent SAB testing to look for presence of Anti HLA Antibodies. HLA typing of this study population was studied for allele diversity and compared with the SAB panel to look for representation.

Results-
Among 1000 individuals, 344 unique alleles were identified. Of them, 34% (n=117) we represented in the SAB panel while 66% (n=227) were not. A total of 14.1% (n=141) had all of their HLA loci represented in the SAB panel while 85.9% (n=859) had at least one allele not represented in the SAB panel. Among the unrepresented alleles, 6 alleles namely A*02:11 (n=145), B*35:03 (n=125), B*40:06 (n=220), C*12:03 (n=110),C*03:02 (n=90),and DQA1*05:05 (n=139) were present in significant number of individuals in the study population.

Conclusion-
Non-representation of the alleles prevalent in our local population is a important drawback of using SAB testing alone for DSA identification. It also has significant impact on PRA estimated from the SAB panel. Owing to these limitations, virtual crossmatch solely cannot be utilized for organ allocation for renal transplantation, CDC or flow cytometry crossmatch is still a standard practice.

ORIGINAL STUDY

An Ultrastructural evaluation of glomerular and peritubular capillary endothelial injury in Acute T Cell Mediated Rejection

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Background: Microvascular endothelial cells (Glomerular and Peritubular capillary) respond to humoral alloimmune injury by recruiting inflammatory cells. Light microscopically this is recognized as Glomerulitis(g) and Peritubular Capillaritis (ptc). Banff criteria includes tubulointerstitial (t, i) and vascular (v) inflammation for the diagnosis of cellular rejection. However, Microvascular injury (g and ptc) is assumed to be cell mediated, in the absence of C4d and circulating donor specific antibody. Ultrastructurally endothelial cell swelling, subendothelial electron lucent widening and early GBM duplication can be seen as early as 3 months post-transplantation in ABMR but not much data is available for ATCMR.

Aims: To study ultrastructure of Glomerular and Peritubular capillary endothelium in Cellular Rejection and other causes of Graft dysfunction.

Methods: Ambispective study taking 18 cases of ATCMR, 08 cases of Active ABMR and 20 cases of other non-immunological causes of rejection presenting within 1 year of transplant (February 2016 to October 2023). For each case, ≥1 glomeruli (≥8 capillary loops in each glomerulus) and ≥10 PTCs were evaluated for ultrastructural endothelial injury.

Results: Significant Microvascular Injury (MVI) was noted in 75% of ABMR cases, 66.67% of ATCMR cases and 05% of other non-immunological causes of rejection. Lesions of microvascular injury on TEM were loss of fenestrations (GLEF, PLEF), endothelial cell enlargement (GE), subendothelial electron lucent widening (GSLW), glomerular vacuolation (GV), glomerular serration (GS), subendothelial neo-densa glomerular basement membrane multilayering (GBMML), peritubular capillary basement membrane multilayering (PTCML), and adhesion of inflammatory cells to endothelium (GADH, PADH). Lesions were noted in ATCMR, ABMR and other non-immunological causes of dysfunction.

Conclusions: Microvascular injury is a significant component of ATCMR. Acute endothelial injury, early GBM reduplication and PTCML are noted in ATCMR, ABMR and other non-immunological causes of rejection with no histological correlation.

Histopathological spectrum of CNI toxicity in renal allograft biopsies

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Background: Calcineurin inhibitors (Cyclosporin and tacrolimus) are mainstay of management in renal allograft patients to prevent graft rejection. However, calcineurin inhibitor toxicity (CNIT) is also a significant cause of renal allograft deterioration. Histopathological features of CNIT have been topic of study and consensus is still improving.

Case series: We received diagnostic biopsies from 33 patients of renal transplant, of which 10 cases showed features of CNIT. Patients' records were evaluated for demographic details, baseline and peak creatinine levels, drug levels, and histopathological features. Cases include 7 males with age ranging from 21 to 55 years and 3 females of 21 to 43 years. Mean age at time of biopsy was 32.80±11.68 years. Eight patients were on tacrolimus, of which 2 had normal drug levels and 6 had high levels of drug. One patient each were on cyclosporin and everolimus and had low and normal levels of drugs respectively. Mean peak creatinine levels were 1.94+0.58 mg/dL in patients with CNIT. Interval between allograft and biopsy ranged from 1 to 32 months with mean of 10.8±10.2 months. On histopathology, 40% patients had mixed features of both acute and chronic toxicity, while features of isolated acute and chronic toxicity were present in 30% patients each. Histopathological features included arteriolar hyalinosis (50% cases), striped fibrosis, isometric vacuolations of the proximal tubules (40% cases), vacuolization of smooth cells of arterioles (10% cases) and glomerulosclerosis. Interstitial fibrosis ranged from mild (50 % cases) to moderate (20% cases).

Conclusion: CNIT was present in 30% of renal allograft biopsies in our study, forming a major factor in renal allograft deterioration. Although the histological features of CNIT are not entirely specific, allograft biopsy is mainstay for the diagnosis of CNIT and to exclude rejection in renal transplantation cases. Therefore, extensive analysis of renal biopsy for these features is essential in view of specific management protocol of these cases with proper monitoring of the drugs.

ORIGINAL STUDY

THE INITIAL STRIDES IN AI-BASED ASSESSMENT OF RENAL PATHOLOGY

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BACKGROUND: Artificial Intelligence's (AI) medical ascendency marks a groundbreaking shift, offering unprecedented advancements in renal pathology. The synergy of AI and medicine heralds a new era of enhanced diagnostic precision and improved patient care. Assessing glomerulosclerosis is vital in renal pathology for accurate diagnosis and treatment planning. Pathologists may err in underestimating the same due to subjective interpretation, emphasizing the need for objective tools, like AI, to enhance diagnostic precision.

AIM: Using advanced AI algorithms to differentiate between normal and sclerosed glomeruli in renal biopsy specimens.

METHODS: One hundred ten images of normal and sclerosed glomeruli from H&E-stained images of renal biopsy were split into training and testing sets. A convolutional neural network was trained on the training dataset for 20 epochs. The trained model was then evaluated on the testing dataset.

RESULTS: Differentiating the two entities was achieved with an accuracy of 88.89%, validated independently by two nephropathologists.

CONCLUSION: Renal pathology stands on the brink of a transformative era with the integration of AI, where its true potential will lie in tackling the intricacies of rare and challenging cases. Our study demonstrates promising strides in accuracy, and as we strive for an accuracy of 99% with a larger sample size, we envision the application of this AI model in diagnosing challenging cases becoming imminent for a future of enhanced precision and diagnostic capabilities.

A study of Spectrum of Histopathological Changes in Nephrectomy Specimens - One year study from a tertiary care center.

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Introduction: Kidneys are major organ of the human body serving various function like electrolyte balance in blood, maintaining PH homeostasis, urea excretion and hormone secretion. Nephrectomy is standard surgical procedure indicated in patients with organ confined renal malignancies and irreversible kidney damage resulting from chronic infections, trauma, obstructive causes including strictures and calculus disease, vesicouretric reflex, congenital malformation.

Aim and Objective: To study spectrum of histopathological changes in radical nephrectomy specimen and to analyse age and sex distribution of these neoplastic and non-neoplastic lesions.

Material and methods: This is retrospective study done for a period of one year from Sep 2022 to August 2023 which included 61 nephrectomy specimens received in our department. Paraffin blocks and slides along with case records were retrieved and studied for age and sex distribution, neoplastic and non-neoplastic causes.

Observation and results: There were 32 male and 29 females. Majority of patients were in 4th to 5th decade. Non-neoplastic cases were 41 (67%) and neoplastic cases were 20 (33%). Most common benign cause was chronic pyelonephritis 28 cases (68%) while most common neoplastic cause was clear cell carcinoma 14 cases (70%).

Conclusion: Radical Nephrectomy is an accepted surgical procedure for non-functioning kidneys due to various pathological disease processes. Detailed histopathological examination is necessary for diagnosis and the prognostication of the lesions. This study gives a fair insight of the current state of incidence of neoplastic and non-neoplastic lesions of kidney requiring surgical intervention.

ORIGINAL STUDY

Reclassifying Membranous Nephropathy in India: Evaluation of Novel Antigens, Etiological Correlations, and Implications for Diagnosis

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Background: Membranous nephropathy (MN) is prevalent in adult nephrotic syndrome, impacting 36%, with one-third progressing to end-stage renal disease (ESRD). Historically categorized into primary and secondary forms, MN, an autoimmune disease, involves thickening of the glomerular basement membrane. Identification of PLA2R as the primary antigen in 70% of "primary" MN cases prompted research into additional antigens. A potential association between mercury exposure and NELL-1-associated MN was explored, particularly among users of complementary alternative medicine or brightening creams.

Aim: To evaluate novel target antigens (NELL1, EXT1/2, Semaphorin-3B, PCDH7, NCAM1, HTRA1, TGFBR3, etc.) in MN cases, determining their clinical significance in the Indian population. Additionally, to investigate heavy metal exposure, particularly mercury, in NELL-1- positive MN patients.

Methods: In our ongoing ICMR prevalence study, advanced techniques like immunohistochemistry, immunofluorescence, Western blotting, electron microscopy, and ICP MS are employed.

Results: In our cohort, 76% exhibited PLA2R and THSD7A antigens, while 24% of Indian patients were negative for both. Preliminary findings revealed 23.4% PLA2R/THSD7A-negative primary MN cases, of which 12% are attributed to NELL-1, with 80% confirmation through Western blot analysis. We observed positive treatment responses in patients post-therapy through Western blot monitoring. Findings of other antigens and their etiological association are in process.

Conclusions: Western blotting emerges as a crucial confirmatory test, guiding tailored treatment plans. Integrating findings into routine clinical practice holds promise for advancing MN understanding and management, significantly contributing to diagnostic refinement. Currently, NELL-1 stands as a pivotal player, propelling nephrology into new realms, benefiting patients, and advancing the field.

THE GENETIC LANDSCAPE OF ALPORT SYNDROME IN INDIAN PATIENTS WITH PHENOTYPIC CORRELATION

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BACKGROUND: Alport Syndrome is a rare genetic disorder caused by mutations in Col4-A3/A4/A5 genes, largely underdiagnosed in India, and remains limited to clinical and pathological features (Anterior lenticonus, SNHL, disrupted GBM).

AIM: Majorly, presenting Col4-A3/A4/A5 genes, and 17 potential modifiers/mimics of Alport Syndrome analysed via whole exome sequencing on a cohort of suspected Alport syndrome Indian patients.

METHOD: Next generation whole exome sequencing of eight suspected Alport families in trio (proband, parents, and/or sibling) was performed via NovaSeq6000 comprising detailed pedigree analysis and comprehensive phenotypic evaluation (eye and ear) including evaluation of kidney biopsy with light microscopy, immunofluorescence and electron microscopy. To execute sequenced data, downstream analysis was performed, and based on ACMG criteria variants annotated.

RESULTS: Of 8 probands, five presented proteinuria and haematuria while 3 had proteinuria only (2- 3+ on dipstick). 4 had Anterior Lenticonus and SNHL while 1 had only SNHL and 3 were normal. Kidney biopsy was examined in 5 patients, 4 showed the classical lamellation and basket weaving of thickened GBM while one only displayed long thin segments, 3 found with kidney family history. In sequencing, five showed mutation in COL4A5 including missense mutation in 2, duplication with frameshift in 1, deletion with frameshift in 1 and a large exonic deletion in 1. These were segregated with mother in 2 cases consistent with X linked inheritance, and 1 case with asymptomatic father/sibling, while one appeared de-novo. COL4A4 homozygous mutations (1 frameshift and 1 stop gain) and COL4A3 heterozygous mutation were noted in 2 cases and 1 case (with thin basement membranes), and found segregating with parent /sibling consistent with Autosomal recessive and dominant inheritance respectively. Single modifier LAMA5 with uncertain significance missense variant was observed in one X-linked case segregating with mother, remaining modifiers appeared benign in all families.

Collagen type IV genes (Col4-A3, Col4-A4, Col4-A5) variants and modifiers presentation in tabular form		
	Collagen type IV gene, HGVSC, Variant, Interpretation	Modifiers/Mimics, HGVSC, variant, Interpretation
Family-1	Col4-A5: c.2297G>A, missense variant, pathogenic	Nil
Family-2	Col4-A5: c. 2605G>A, missense variant, pathogenic	LamB2: c.5026_5027delinsAT, missense, uncertain significance
Family-3	Col4A-4: c.3622del, frameshift, likely pathogenic	Nil
Family-4	Col4-A4: c.3933C>G, stop-gained, pathogenic	Nil
Family-5	Col4-A5: c.3584dup, frameshift	Nil
Family-6	Col4A5: c. (141+1_142-1) (2509+1_2510-1), exonic deletion, pathogenic	Nil
Family-7	Col4-A3: c.2702G>A, missense, likely pathogenic	Nil
Family-8	Col4-A5: c.907del, frameshift, likely pathogenic	Nil

CONCLUSION: This is the first Indian cohort with comprehensive phenotypic and genetic evaluation. Spectrum of mutations were noted with variable phenotypic presentation, underscoring the need for a low index of suspicion and availability of genetic testing for this patient group in India.

Spectrum of Pathological changes in C3 glomerulopathy: A tertiary institute study

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Background:C3 glomerulopathy(C3G) includes a group of renal diseases caused by uncontrolled activation of the alternative complement pathway in the circulation and at the surface of the GBM in the glomerulus with dominant C3 deposition in glomeruli on immunofluorescence (IF). Very little data is available on patients of C3G from India.

Aim and Methods: All patients diagnosed as C3G on EM were included in the study. Clinical data and kidney biopsy findings were analysed. All serological data and serum samples were collected for complement work up. Urine was examined for degree of active disease by predefined criteria. Patients were followed up to a period of one year. Activity of disease was evaluated with serum creatinine levels, and repeat urine examination to assess efficacy of treatment.

Results and Conclusions:

Kidney biopsies of 96 patients of C3G received in the Department of Pathology, GIPMER, New Delhi over a period of 5 years (2018-2023) were analysed. 74% of patients were less than 18 years of age with M: F ratio of 1.3:1. Most patients presented with haematuria & proteinuria.

Patterns of injury in kidney biopsies on light microscopy, includes mesangioproliferative glomerulonephritis, DPGN, MPGN, crescentic glomerulonephritis, and CKD. On immunofluorescence, glomeruli showed a predominant deposition of C3. Diagnosis was confirmed on EM, according to which patients were classified into dense deposit disease (DDD) and C3 glomerulonephritis (C3GN).

Our patients predominantly included C3GN [n=72(75.8 %)] whereas cases of DDD included 23 patients (24.2 %). Most patients showed low levels of C3 with normal C4. Classical and alternative pathway activity was also analysed in these patients.

CASE REPORT

Podocyte infolding glomerulopathy: Still a debatable entity?

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Background: Podocyte infolding glomerulopathy (PIG) is an extremely rare manifestation of renal disease that was initially documented in Japan in 1992. The Japanese Society of Nephrology formally proposed PIG as a new disease entity in 2008. However, it is not included as a separate category in the current classification of glomerular diseases. Due to a shortage of reports, its underlying pathogenic mechanism and clinical manifestations are yet unknown.

Case report: We present two cases of PIG. A 40-year-old male presented with pedal edema, facial puffiness and a subnephrotic range proteinuria. On light microscopy, most glomeruli were globally sclerosed. The viable glomeruli revealed basement membrane thickening. Immunofluorescence showed coarse granular deposits of IgG (3+) along the peripheral capillary walls. A diagnosis of membranous glomerulonephritis was suggested.

The second case was of a 34-year-old female who presented with similar complaints and significant proteinuria. In addition, she had a history of facial rashes associated with photophobia, however her autoimmunity profile was non-specific. Light microscopy showed features of chronic kidney disease with acute tubular necrosis. On immunofluorescence, no significant immune deposits were identified. Interestingly, on electron microscopy both these cases revealed thickening of basement membrane, diffuse effacement of foot processes and an unusual presence of microspheres in the glomerular basement membrane. Therefore, a final diagnosis of PIG was rendered.

Conclusion: PIG is an uncommon renal manifestation which can occur independently or concurrently with an underlying immune process. Without an ultrastructure study, it is often confused with and misdiagnosed as membranous nephropathy. There is an ongoing debate on whether PIG represents a distinct disease entity or merely an unusual renal pathological finding of co-existing disease.

INTERESTING CASE OF ALPORT SYNDROME

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Introduction:

Alport syndrome is a genetic condition characterized by kidney disease, loss of hearing and eye abnormalities. It's also called hereditary nephritis. It occurs due to an abnormality of a gene that codes for type 4 collagen. Therefore, it usually presents in patients with hematuria, oedema, and hypertension. In 80% of cases, Alport syndrome is inherited in an X-linked pattern and caused by COL4A5 gene mutations, although other inheritance patterns exist. It can be inherited as an AR or AD pattern by COL4A3 or COL4A4 gene mutations.

Case Report:

A 30-year-old male presented to OPD with decreased vision and hypertension. He had a family history of Alport Syndrome in his mother and brother. On investigations, S. Creatinine level: 3.7 mg/dl, B Urea level: 90 mg/dl and BP:160/90 mm of Hg. On Urine analysis, Glucose: negative, albumin and Granular casts: ++, eGFR: 21.3 ml/minute. The clinical diagnosis of Alport Syndrome was made.

Renal biopsy showed 12 glomeruli of which 10 were globally sclerosed. Remaining 2 glomeruli showed mesangial matrix expansion and hypercellularity. Chronic tubulointerstitial activity (60-70%) was noted along with changes of hypertension. Based on family history, clinical history and histological findings, a diagnosis of Alport syndrome was considered. Electron microscopy was advised for confirmation.

Conclusion:

Lipid-laden interstitial foam cells are considered a marker of Alport's syndrome. During the course of the disease, the glomerular basement membrane gradually thickens, leading to global glomerulosclerosis, interstitial fibrosis, and tubular atrophy. As in our case, Diffuse Glomerulosclerosis with moderate chronic tubulointerstitial activity with hypertensive changes is indicative of end-stage kidney disease. Family and clinical history helped us to consider a diagnosis of Alport's syndrome.

CASE REPORT

Role of Genetic study in Steroid-Resistant Nephrotic Syndrome

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Background

Children presenting with idiopathic nephrotic syndrome (NS), mostly have minimal change disease, which is responsive to steroid therapy. In such cases, initial treatment is empirical steroid therapy and most patients respond it.

Steroid-resistant NS (SRNS) are cases that do not respond to initial steroid therapy. Approximately one-third of SRNS are due to single gene variants that affect glomerular podocyte structure and/or function, and are poor responders to immunosuppressive therapy with faster rate of disease progression to kidney failure. Thus, therapeutic decisions in children with SRNS depend in part on the underlying etiology.

Case report

2 yr old female girl, with nephrotic syndrome, haematuria and hypertension. Clinically as Steroid resistant Nephrotic Syndrome after 4weeks of Inj. Methylprednisolone.

Result

Renal biopsy was done, Light Microscopy suggested Minimal Change Disease (MCD); DIF – All negative, Electron Microscope – Alport's syndrome. As EM findings not correlating clinically, genetic workup done, revealed Nephrotic Syndrome type2 - NPHS2 gene mutation.

Conclusion

In cases of SRNS, it is important to rule out the underlying etiology for betterment of the child, and it can be best achieved if complete evaluation include genetic workup. Genetic workup gave exact underlying etiology of the condition, and so accordingly further management can be done.

Pathological insights into Diffuse Mesangial Sclerosis: A case report

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Introduction: Diffuse mesangial sclerosis (DMS) is a rare cause of nephrotic syndrome in the infantile and childhood period. DMS is a phenotypic expression of syndromic entities such as WAGR syndrome, Denys Drash syndrome, Pierson syndrome and Frasier syndrome.

Case Report: One-month full term male neonate presented with proteinuria (3+) and abnormal kidney function tests (S. Creatinine- 3.4 mg/dl and BUN-75 mg/dl). No obvious perinatal insult or increased placental weight was noted. MRI did not show any cysts. The biopsy revealed small glomeruli, variable degrees of mesangial sclerosis and obliteration of glomerular capillary loops, with few showing globally solidified tuft and obliterated capillaries. Few of the glomeruli showed shrunken tufts and markedly dilated Bowman space, consistent with glomerulocystic change. Tubular compartment shows many dilated tubules with PAS positive hyaline casts.

DMS is characterized by progressive sclerosis of the mesangial matrix with minimal or absent mesangial cell proliferation, hypertrophy of podocytes, thickened basement membranes, diminished patency of capillary lumen, pseudocrescent formation and crown-like appearance of vacuolated podocytes. Differential diagnosis of DMS includes collapsing variant of focal segmental glomerulosclerosis. DMS is associated with genotypic disorders such as WAGR syndrome, Denys Drash syndrome, Frasier syndrome, Pierson syndrome and Galloway–Mowat syndrome, and hence multiorgan abnormalities associated with these conditions. Management includes antiproteinuric measures, including nephrectomy in non-responders to medical management.

Conclusion: The case report highlights the key pathological features of DMS. Correct histopathological diagnosis of this disease is essential for proper and early management as it may lead early ESRD and may also be associated other organ involvement as a part of syndromic association.

CASE REPORT

Collapsing Glomerulopathy in case of Lupus Nephritis- An uncommon pathologic finding in SLE.

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Background:

Systemic lupus erythematosus (SLE) is a chronic inflammatory autoimmune disease affecting multiple organ systems characterized by the production of autoantibodies against nuclear and cytoplasmic antigens. (1).

Kidney involvement by SLE (Lupus Nephritis) affects more than 50% of patients with SLE and is a major cause of morbidity and mortality (2).

Collapsing glomerulopathy is characterized by glomerular collapse and epithelial hypercellularity and presents with severe nephrotic syndrome and rapid progression to ESRD (3).

Collapsing glomerulopathy in cases of SLE with or without Lupus Nephritis is an uncommon pathologic finding and has been reported in small case series and case reports. (4,5,6, 7)

Case Report:

Clinical History: Patient is 22 years old female/ FTNVD/Uneventful birth and childhood history.

c/o: bilateral pedal edema insidious onset and progressive since last 2.5 months. At the same time found to have her 2nd pregnancy. She had uneventful 1st pregnancy was 2 yrs back

Past history of hypothyroidism.

Underwent D&C due to progressive oedema and uncontrolled hypertension. She has nephrotic range proteinuria (3.9 gm/day), renal dysfunction and low C3, ANA and ds DNA Positive.

Renal Biopsy showed features of Class III Lupus Nephritis along with co-existing Collapsing glomerulopathy.

Conclusion: Lupus Nephritis with co-existing Collapsing Glomerulopathy is an uncommon pathologic entity. The exact pathogenesis of Collapsing glomerulopathy in Lupus Nephritis and its relationship with Lupus podocytopathy is still not clear. However, it may represent a severe form of Lupus Podocytopathy. (8). In our case we propose that 2nd pregnancy could be the precipitating factor for Collapsing Glomerulopathy.

Single atrophic kidney with Persistent Hematuria and uncontrolled hypertension a precursor to focal segmental glomerulosclerosis with Tubular Necrosis

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Background

Focal segmental glomerulosclerosis is a progressive glomerular disease exhibited by microscopic hematuria with hypertension. Impairment of renal function is detected at onset. Patients with persistent proteinuria show histological lesions indistinguishable from FSGS. Idiopathic FSGS should be distinguished from secondary FSGS that occur in reflux nephropathy, sickle cell disease, hyperfiltration injury. Familial forms of FSGS, inherited in an autosomal dominant or recessive manner, occur in mutations in genes encoding important podocyte proteins.

Case report

The case is of a 16/ female presented with complaints of foamy cola urine and burning micturition, fever with fatigue and shortness of breath on mild activity along with headache. Her blood pressure turned out to be greater than 95th centile. On further evaluation proteinuria was present with deranged renal functions and presence of pus cells were also seen. NCCT KUB exhibited left renal atrophy. Her C3 levels within limits. ASO- positive and ANA-negative. Amlodipine with envas were prescribed.Her urine microalbumin was raised and A:C ratio was increased. Diagnosis of acute glomerulonephritis with atrophic left kidney with UTI was made on first visit but persistent hypertension and microscopic haematuria with proteinuria raised the suspicion, then amlodipine, was given along with spironolactone. Renal Biopsy showed focal segmental glomerulosclerosis with tubular necrosis.

Conclusion

Aggressive Protocol with high dose methylprednisolone. Discharged on antihypertensive. Her visual acquity is regularly evaluated but irregular use of antihypertensive makes the prognosis grave.

CASE REPORT

Crescentic presentation of diabetic nephropathy in absence of non-diabetic kidney disease.

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Background:

Glomerular crescents are formed by the extraglomerular accumulation of cells in the bowman's capsule. They are generally associated with rapidly progressive crescentic glomerulonephritis. Herein, we present a case of crescentic glomerulonephritis superimposed on type III diabetic nephropathy in the absence of another disease.

Case report: A 49-year-old male, who is a known case of DM type 2, presented to our hospital with bilateral lower limb swelling, shortness of breath, decrease in urine output, and blurring of vision over the last one month. Investigations revealed nephrotic range proteinuria, microscopic hematuria and renal insufficiency. His HbA1c was 6.8 %. Autoimmune work-up and monoclonal work-up was negative. Viral markers were also negative. Complements were normal. Renal biopsy was performed to rule out non-diabetic kidney disease (NDKD). Kidney biopsy included 21 glomeruli, and most of them show mild glomerulomegaly and classical Kimmelstiel- Wilson nodule. Eleven glomeruli show crescents formation (10 fibrocellular and 1 fibrous). Moderate interstitial fibrosis and tubular atrophy with accompanying inflammation and vascular hyalinosis was present. On direct immunofluorescence, pseudo linear positivity along the glomerular capillary loop and tubular basement membrane was noted against IgG, kappa and lambda. A diagnosis of diabetic nephropathy class III with crescents was offered. Further investigation did not support any systemic disorders or NDKDs. After two months of biopsy, the patient became dialysis-dependent.

Conclusion:

Crescentic GN superimposed on diabetic nephropathy, in the absence of NDKD, are rare. There is limited data in the literature on the management and prognosis of these cases. Our case adds to the existing literature.

Keywords: diabetic nephropathy; crescent; diagnosis; histopathology; immunofluorescence

HIV Nephropathy- A Deep Dive into the Spectrum of Renal Complications

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BACKGROUND: HIV (Human Immunodeficiency Virus) affects multiple organs including kidney. Term "HIV associated renal disease" includes HIV-associated nephropathy (HIVAN), with hallmark being collapsing glomerulopathy, thrombotic microangiopathy, and less commonly described HIV associated immune-complex kidney disease (HIVICK), which mostly presents as lupus-like lesion. Herein, we discuss two such interesting cases of HIVICK.

CASE REPORT: 41-year- seropositive male, presented with haematuria, proteinuria and pruritic skin lesions. Kidney biopsy showed mesangial matrix expansion & hypercellularity, segmental scarring with synechiae formation and prominent tubular microcystic dilatation. Immunofluorescence (IF) showed granular glomerular staining against anti IgA (3+ along mesangium). Diagnosis of HIV associated immune-complex deposition disease (IgA nephropathy with FSGS-NOS) was rendered.

21-year-old male on anti-retroviral therapy (ART) presented with hypertension, proteinuria, haematuria, raised creatinine & reduced complement levels for 10 days. Kidney biopsy showed membranoproliferative pattern of injury with fibrinoid necrosis. On IF, 3+ capillary wall granular deposits against antisera for IgG, IgM, C3, light chains kappa and lambda were seen and diagnosis of Immune- complex mediated membranoproliferative glomerulonephritis was made.

CONCLUSION: Renal disease is an important cause of morbidity and mortality, affecting approximately 30% of retro-positives. Before the era of ART, HIVAN was the leading cause of end-stage kidney disease. After its introduction, prevalence of HIVAN has reduced with corresponding rise of HIVICK, which responds to corticosteroids. No consensus for the management of HIVICK has been described due to limited existing data. These two cases highlight the important role of renal biopsy in retro-positives for differentiating HIVICK from other glomerular diseases.

Abbreviations: - HIV: Human Immunodeficiency Virus; HIVAN: HIV-associated nephropathy; HIVICK: HIV associated immune-complex kidney disease; IF: Immunofluorescence; FSGS-NOS: Focal segmental glomerulosclerosis- Not otherwise specified; ART: Anti-retroviral therapy

CASE REPORT

Dengue Related Glomerulonephritis in a 21-year male - a case report

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Background: Dengue is a systemic acute febrile disease caused by an RNA arbovirus and transmitted by mosquitoes of the genus Aedes. Various forms of kidney involvement are reported in patients with dengue, including acute tubular necrosis, haemolytic uremic syndrome, glomerulonephritis. Herein, a case of infection related glomerulonephritis related to dengue infection is described.

Case Report: A 21-year male presented with bilateral lower limb swelling, facial puffiness and haematuria for last 15 days. He had fever for three weeks and was tested positive for dengue NS1 IgM ELISA. Investigations showed serum creatinine of 1.1mg/dl and blood urea nitrogen of 31 mg/dl. Urine microscopy revealed 100-120RBCs/hpf and 30-40 WBCs/hpf. 24 hr urine protein was 5.5gm/dl. Complement work-up revealed low C3 and normal C4 value. Autoimmune work-up and viral markers tested negative. The renal biopsy showed diffuse endocapillary and mesangial hypercellularity of the glomeruli accompanied by variable neutrophilic exudation and segmental cellular crescent formation (38%). The direct immunofluorescence showed granular mesangial and capillary wall deposits against the antisera specific for IgG (2+), complement C3 (2-3+) and light chains. A diagnosis of infection related glomerulonephritis related to dengue virus infection was rendered. Following treatment, there is complete resolution of proteinuria with normal renal function.

Conclusion: Infection related glomerulonephritis is a rare and emerging cause for significant renal morbidity in adults. The main mechanism of dengue glomerulonephritis is still unknown. To avoid otherwise preventable mortality and morbidity, we emphasise the need for a proper renal biopsy diagnosis in cases of dengue fever presenting with proteinuria and renal dysfunction.

Key Words: Glomerulonephritis, Dengue, kidney biopsy

NEITHER INFECTION NOR ANTIBODY MEDIATED! - A CASE OF NON-CRYSTALLINE LAMBDA LIGHT CHAIN PROXIMAL TUBULOPATHY WITH LIGHT CHAIN CRYSTALLINE CAST NEPHROPATHY

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Background: Proximal light chain tubulopathy (LCT) is a rare renal manifestation in multiple myeloma Combination of light chain cast nephropathy and LCT is uncommon and a very few cases are reported in literature.

Case Report: 37-year-old, non-diabetic, hypertensive male patient presented with flank pain, breathlessness, generalised oedema and reduced urine output for the past one week. Laboratory investigations were significant for anaemia (Hb-9 g%), mild thrombocytopenia (Platelet count- 1.31 lakh/mm3). Serum creatinine was 7.43 mg/dl (ref. range 0.7-1.4 mg/dl). Urine examination revealed presence of pus cells (10-15/HPF) and proteinuria (urine albumin 2+). Serum anti-GBM antibody titres slightly higher than the reference range (9.6 IU/ml, <5 -negative, >9- positive). Clinical diagnosis of Dialysis dependent renal failure possibly due Acute Kidney Injury (AKI) with? pyelonephritis or anti-GBM disease was made and kidney biopsy was performed.

Renal biopsy sent for light microscopy revealed thirteen viable normal glomeruli. Tubules showed severe degree of acute injury. A few vermiform and crystalline casts with neutrophilic inflammatory reaction noted mainly in distal tubules. Clear to vacuolated inclusions were present with the cytoplasm of proximal convoluted tubules. Luminal Casts and vacuolations within the tubular epithelium showed lambda light chain restriction on immunofluorescence. A diagnosis of lambda light chain non-crystalline proximal tubulopathy with crystalline cast nephropathy was rendered. Serum protein electrophoresis performed later revealed presence of 'M' spike.

Conclusion: This case is presented not only due its rarity but also to emphasise the utility of renal biopsy and immunofluorescence to establish accurate diagnosis.

Key words: Plasma cell dyscrasia, Fanconi anaemia, light chain restriction

CASE REPORT

Atypical presentation of light chain deposition disease secondary to multiple myeloma in central India: 2 case reports

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Background: Light chain deposition disease is the deposition of monoclonal light chains in multiple organs. Approximately 50-60% of patients with LCDD have associated multiple myeloma and can primarily present with acute renal failure of unknown etiology. We report two such cases where cues from renal biopsy helped in making a diagnosis of multiple myeloma.

Case report: Case 1: A 65 male, known Hypertensive on medication presented with generalized weakness and loss of appetite since 3 months, along with loose stools (3-4 episodes/day). His CBC revealed Hb of 6.6 and Serum Creatinine of 8.89 mg/dL, total protein of 6.79 g/dL with albumin globulin ratio of 1.30. Urinalysis revealed 1+ protein, with nil white blood cells and nil RBCs per high power field. Case 2: A 49-year female presented with loss of appetite, burning sensation in chest, associated with bloating, since 3 months. Outside investigations revealed deranged renal parameters (serum creatinine – 6.1 mg/dl, serum urea – 106.7 mg/dl) for which she was admitted to the Nephrology department. General examination did not show any significant abnormalities. Urinalysis revealed trace proteinuria with 2-3 RBCs per high power field.

Renal Biopsy in both the cases showed Tubular Atrophy and Interstitial fibrosis with scattered atypical casts within tubules. Diffuse linear staining for kappa light chain was noted along the glomerular and tubular basement membranes along with atypical intratubular casts. Bone marrow biopsy followed, which revealed cellular bone marrow with increased plasma cell population and thus diagnosis of multiple myeloma was made.

Conclusion: Multiple myeloma can rarely present as acute renal failure of unknown etiology. Careful light microscopic examination of renal biopsy along with immunofluorescence examination can help picking up LCDD which can provide lead to the final diagnosis.

Light chain cast nephropathy with unusual morphology– A case report

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Background:
Multiple myeloma is a malignancy of plasma cells resulting in overproduction of monoclonal Immunoglobulins. Light chain cast nephropathy (LCCN) is the most frequent form of renal disease in plasma cell neoplasm showing precipitation of monoclonal immunoglobulin light chains in the lumen of the distal tubules.

Case Report:
A 57 yr old male presented with fever, generalized weakness and low backache for the past 6 months. On investigation, he was anaemic with a blood urea of 237 mg/dl and serum creatinine of 16.3 mg/dl. Urine examination showed traces of albumin, 15-20 pus cells per hpf and no active sediment. Skeletal survey showed multiple punched out lytic lesions. A diagnosis of Acute renal failure secondary to? multiple myeloma was made. Bone marrow aspiration showed 80% plasma cells. Renal biopsy showed pink eosinophilic fractured and lamellated casts with peripheral spikes, PAS positive, silver negative and congophilic casts. Immunofluorescence showed lambda light chain restricted casts. A diagnosis of Light chain cast nephropathy was made. There was an M spike on serum electrophoresis and Serum free light chain assay showed lambda restriction.

Conclusion:
LCCN is a leading cause of acute kidney injury in patients with multiple myeloma and is now defined as a myeloma defining event. Herein we report a case of LCCN exhibiting peculiar morphological feature, identification of which is crucial as this may indicate the presence of an underlying plasma cell myeloma associated with systemic light chain amyloidosis, which has grim prognosis.

CASE REPORT

Crystal Storing Histiocytosis with concurrent light chain associated proximal tubulopathy.

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Introduction- Crystal-storing histiocytosis (CSH) is a rare lesion which is characterized by the presence of crystal laden histiocytes. These crystals are abnormal accumulation of immunoglobulins within histiocyte lysosymes. This condition is seen associated with plasmacytic or B-cell lymphoproliferative neoplasms and rarely inflammatory disorders. Localized and generalized forms of these lesions have been reported. Here, we intend to report this rare lesion with concurrent light chain associated with proximal tubulopathy.

Case report- A 65-year-old male presented history of hypertension for 15 years and history of dysuria and abdominal pain for 2 years. Lab investigations- Creatinine-0.95mg/ dL, urea-17.9mg/ dL, total protein- 6.94 g/ dL, serum albumin- 2.64 gm/ dL, serum calcium- 7.78 mg/ dL, hemoglobin- 9.2 gm/dL, TLC- 9.4x109/L, Platelet count- 332X4x109/L , Bone marrow aspirate- 12% plasma cells, urine electrophoresis- nil, serum protein electrophoresis- M-band in far gamma region and immunofixation elextrophoresis shows monoclonal gammopathy IgG kappa. Kidney biopsy performed showed lymphoplasmacytic infiltration in the interstitium along with severe acute tubular injury. Direct immunofluorescence showed 3+ intense positivity for kappa in the proximal tubules. A biopsy from the peri-renal fascia was also performed and showed many dispersed and focal clusters of histiocytes containing eosinophilic refractile crystals. Direct immunofluorescence performed on the paraffin tissue of fascia also revealed rhomboid shaped crystals which were kappa restricted. Genome sequencing identifies pathogenic variation in MYD 88 gene.

Conclusion- This case highlights clinical, pathological, and genetic features of crystal storing histiocytosis with concurrent proximal tubulopathy in the kidney.

A rare case report of polyarteritis nodosa with SAA Amyloidosis: a bolt from the blue.

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Background:
Polyarteritis nodosa (PAN) is a systemic necrotizing medium vessel vasculitis. Renal artery involvement in form of aneurysmal dilation, thrombosis and rupture which can result in renal haemorrhage and infarction has been reported, but renal amyloidosis is rare. We describe a rare case of PAN complicated by renal amyloidosis.

Case report: A 26-year-old gentleman case of PAN with mononeuritis multiplex (nerve biopsy: vasculitic neuropathy), cardiomyopathy (ejection fraction: 15-20%), renal and splenic infarcts presented with swelling of bilateral lower limb with facial puffiness and periorbital oedema since one month. On workup 24-hour urine protein 7050 mg/TV, kappa/lambda ratio:1.4; viral markers, autoimmune workup and DADA2 were negative. A renal biopsy was done given renal parenchymal disease on USG KUB and sudden onset proteinuria (24hr protein 5.2g). Light microscopy showed expansion of mesangium by nodular acellular amorphous material which were weakly PAS-positive, silver negative, congophilic with apple green birefringence. Similar deposits were also seen in interlobular artery. DIF was negative for IgG, IgA, IgM, C1q, kappa and lambda. However, IHC for SAA shows diffuse positivity in areas of amyloid deposits. The patient was initiated on Adalimumab and follow-up had normalisation of serum albumin, reduction of nephrotic syndrome with normal TG levels. On extensive literature search, only a few cases of PAN with SAA have been described up to date.

Conclusion:
Classical PAN is an immune complex-mediated disease and inflammation-induced cytokines induce production of acute phase reactants including SAA. Amyloidosis complicating PAN is a rare condition hence warrants discussion because of prompt response post-therapy.

CASE REPORT

From Nodes to Nephrons: Renal amyloidosis associated with Castleman disease

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Introduction: Castleman disease (CD) is an atypical lymphoproliferative disorder with characteristic lymph node histopathology. CD can present with constitutional symptoms, lymphadenopathy, cytopenias, and multiple organ dysfunction. However, renal involvement has been infrequently described in CD. We hereby report a case of castleman disease that later developed renal amyloidosis.

Case report: A 25-year-old male presented with history of fever, pain abdomen, abdominal distension for 3 months and bilateral pedal edema for 2 months. On examination, hepatosplenomegaly was present. PET-CT which showed FDG avid large cervical, supraclavicular and mediastinal lymph node. Laboratory investigation revealed anemia Hb-3.8gm/dl, deranged renal function test with S. creatinine- 5.18mg/dl and 24hr urine protein-2.5g/day. Total protein/albumin was 6.4/2.1g/d, SFLC-Kappa-385mg/L, lambda-354mg/L; kappa:lambda ratio 1.08 and SPEP was negative. Supraclavicular lymphnode was excised and sent for histopathology examination (HPE). Grossly the lymph node was 2.0 cm in maximum dimension. On HPE, the lymph node showed maintained nodal architecture with marked interfollicular area expansion by sheets of plasma cells. On immunohistochemistry, the plasma cells were highlighted by CD138 and showed lambda light chain restriction. Three months later, renal biopsy sent for HPE showed diffuse complete occlusion of glomerular capillary loops due to mesangial expansion by deposition of amyloid. The arteriolar walls also revealed amyloid deposition. Further, SAA immunostain was positive.

Conclusion: This case highlights the crucial role of timely renal biopsy in unraveling diverse renal presentations associated with Castleman Disease. Such interventions are pivotal for accurate diagnosis and tailored management, ultimately improving patient outcome.

Polyarteritis nodosa masquerading as renal abscesses in a female child.

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Introduction: Polyarteritis nodosa (PAN) is a multisystem affecting medium vessel vasculitis characterised by transmural segmental necrotising inflammation of the vessel wall. Herein, we present a case of PAN mimicking as renal abscesses on imaging and diagnosed on kidney biopsy.

Case report: A 9-year-old female child presented with complaints of severe pain of bilateral lower limbs, blackish discolouration of limbs and intermittent diffuse abdominal pain. Routine investigations revealed anaemia, leucocytosis, thrombocytopenia, raised ESR (55mm/1hr) and normal renal function tests. She was hypertensive with a blood pressure of 117/92 (>95 percentile). Ultrasonography of the abdomen showed two hypoechoic areas in the right kidney, measuring ~ 1.9 x 1.7 cm and 1.7 x 1.5 cm in the lower pole and upper pole respectively, without internal vascularity. A strong possibility of renal abscesses was suggested. She also had decreased blood flow in major arteries of the lower limb on colour doppler. A targeted renal biopsy was performed and sent for microbiological culture and histopathological examination. No growth of microorganisms was noted on culture. Kidney biopsy revealed necrotizing arteritis of arcuate calibre blood vessels with surrounding dense mixed inflammation. In absence of glomerular involvement, a diagnosis of PAN was suggested. Subsequently, digital subtraction angiography revealed microaneurysms in the right renal arteries.

Conclusion: Systemic PAN, though uncommon in the paediatric population, should be considered in the differential diagnosis of renal abscesses. Targeted kidney biopsy may be helpful in diagnosing PAN, particularly in absence of overt systemic manifestations.

CASE REPORT

Disseminated calcification in a case of multicystic dysplastic kidney

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Introduction: Paediatric renal cystic diseases are a common cause of morbidity, and are included in the spectrum of CAKUT (Congenital anomalies of kidney and urinary tract) anomalies. Here we report an interesting autopsy finding in an infant having bilateral cystic kidneys with disseminated calcification.

Case report: A 6 months old female infant was brought dead in the emergency department. At the time of previous admission five months back, baby had non-oligouric CKI with raised creatinine, hyperkalemia and metabolic acidosis. She was started on calcium supplementation, and follow-up after 2 weeks was advised. Autopsy examination reveal bilateral cystic kidney disease with multiple tiny cysts identified in cortex and corticomedullary junction ranging in size from 0.1 to 0.5 cm. Microscopic examination reveal variable sized cysts in both kidneys involving both cortical and medullary areas. These cysts are mainly formed because of dilatation of both proximal and distal tubules as highlighted by CD10 and CK7, respectively. Foci of glomerulosclerosis seen. In addition, there is extensive calcification involving tubules, vessels and glomerular compartment. A few foci showed renal dysplasia in the form of metaplastic cartilage, suggestive of multicystic dysplastic kidney (MCDK). Calcification was observed in every organs except liver. Liver had features of congenital hepatic fibrosis. Brain showed features of hepatic encephalopathy. Genetic study showed heterozygous PAX 2 mutation.

Conclusion: The present case highlights an important iatrogenic complication of excessive calcium supplementation in a patient of MCDK. Also we document the rare association of congenital hepatic fibrosis in presence of bilateral MCDK.

Abbreviation: CKI: Chronic kidney injury, MCDK: Multicystic dysplastic kidney, PAX2: Paired box gene 2

CASE REPORT

A case series on pigment induced nephropathy- the causes and outcomes

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Background: Pigment-induced nephropathy leads to a rapid reduction in kidney function due to the deposition of endogenous heme pigments, myoglobin and haemoglobin. Rhabdomyolysis and haemolysis cause pigment nephropathy that progresses to chronic kidney disease requiring haemodialysis in some patients.

Methods: We performed a retrospective observational study over a period of 2 years (2022-23) to analyze the etiology, clinical manifestations and histopathological features in patients with biopsy-proven pigment-induced nephropathy.

Results: A total of 5 patients were included. Age range was 25-50 years (males: female ratio-2:3). All these cases presented with acute renal failure. Two patients presented with oliguria, while the other three had anuria. Evidence of rhabdomyolysis alone was noted in 1 case, and the serum creatinine in this case was 5.7 mg/dl. Features of hemolysis alone were seen in 2 patients, while features of both rhabdomyolysis and hemolysis were present in 2 cases (mean serum creatinine at presentation were 6.49 and 10.7 mg/dl respectively in these cases). Etiology of rhabdomyolysis only case was malaria by plasmodium vivax (1/5). The causes of hemolysis only patients included one case each of vasculotoxic snake bite and plasmodium falciparum malaria. Cases with both rhabdomyolysis and hemolysis had autoimmune hemolytic anaemia (DAT positive; 1/5) and Dengue (1/5).

Perls stain for hemosiderin and immunohistochemistry for myoglobin was done in all the cases. Intraepithelial hemosiderin deposition in tubules was noted in all the cases (2 of which also showed haemoglobin casts). Myoglobin casts were seen in three cases suggesting rhabdomyolysis. Both the patients with history of malaria also had focal cortical necrosis and malarial pigment deposition in thrombosed glomeruli. Patient with snake bite also had patchy cortical necrosis. Cases with both rhabdomyolysis and hemolysis had more severe acute tubular necrosis than the other cases. No immune deposits were noted on immunofluorescence.

Conclusions: The causes of pigment nephropathy in this series included malaria, vasculotoxic snake bite, autoimmune haemolytic anemia and dengue. Most cases presented with acute renal failiure.

CASE REPORT

Renal Allograft failure resulting from Diffuse Cortical Necrosis

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Background

Cortical necrosis of the renal allograft is an extremely rare but serious complication of renal transplantation and can lead to graft failure.

Case Report

We present an unusual case of a 28-year-old female with chronic kidney disease since four years, who was referred for a live related renal transplant. At transplantation, warm ischemia time was 6 minutes and cold ischemia time was 1 hr 42 minutes. Post - transplant the patient was anuric and developed delayed graft function. On the 2nd post-transplant day, the patient developed tacrolimus toxicity with tacrolimus trough levels of 30 ng/ml.

On the 3rd post-transplant day, she was restarted on hemodialysis due to rising creatinine levels. Transplant kidney biopsy showed acute tubular injury- ischemic type. C4d and donor specific antibodies were negative. On the 12th post-transplant day, USG Doppler of the renal allograft showed thrombosis of the renal vein. An open wedge biopsy was done which showed extensive renal cortical necrosis. A month and a half later, the patient developed pus collection anterior to the transplanted kidney and a fistula with persistent pus discharge. Two months later, a graft nephrectomy was performed and diffuse cortical necrosis of renal allograft with ureteral necrosis and thrombosis of both renal artery and vein were seen.

Conclusion

To prevent graft failures, it is critical to differentiate thrombosis caused by surgical complications from hyperacute or acute humoral rejection. Rapid diagnosis and treatment of renal cortical necrosis with its varied etiological factors can lead to better outcome in graft survival.

ACUTE OXALATE NEPHROPATHY SECONDARY TO EXCESSIVE DIETARY INGESTION

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Background- Acute oxalate nephropathy is a rare but important cause of severe acute kidney injury and is characterised by acute deterioration of renal function due to extensive deposition of calcium oxalate crystals in the renal tubules. We describe two cases of unexplained, severe acute kidney injury (AKI) with acute oxalate nephropathy on histopathology.

Case Report-

Patient 1: A 64-year-old man diagnosed with a neuroendocrine tumour of the colon with liver metastasis, presented with chronic diarrhoea, pedal oedema, oliguria, nausea, loss of appetite, and dyspnea for 6 months. On evaluation, he had anaemia, azotemia and moderate proteinuria. BUN was 140 mg/dl and creatinine was 7.9mg/dl and required hemodialysis till he succumbed.

Patient 2: A 42-year-old male with a history of untreated hypertension and nephrolithiasis presented with abdominal pain, nausea, loss of appetite, and pedal edema for 1 month. BUN was 93.4 mg/dl and serum creatinine was 10.3mg/dl. He required hemodialysis for 8 weeks.

Both of our patients had acute oxalate nephropathy with extensive deposition of oxalate crystals in the renal tubules. Factors like nephrolithiasis, untreated hypertension, and cancer in our patients also made them vulnerable to AKI. On inquiry, patient 1 revealed a history of daily intake of 40ml each of Amla and Aloe Vera juice. Patient 2 gave a history of excessive consumption of green tea immune-fortified with vitamin C freely available at the office vending machine.

Conclusion-

Knowledge and awareness of oxalate nephropathy due to excessive consumption of oxalate-rich foods can be helpful to diagnose and prevent acute oxalate nephropathy.

CASE REPORT

Case of Primary hyperoxaluria type 1

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BACKGROUND

Primary hyperoxaluria type 1 (PH1) is caused by a deficiency of the liver peroxisomal enzyme alanine glyoxylate-aminotransferase (AGT), which catalyzes the conversion of glyoxylate to glycine. When AGT activity is absent, glyoxylate is converted to oxalate, which forms insoluble calcium oxalate crystals that accumulate in the kidney and other organs. Age at onset of symptoms ranges from infancy to the sixth decade. The majority of individuals with PH1 present in childhood or early adolescence, usually with symptomatic nephrolithiasis and reduced kidney function along with nephrocalcinosis which can lead to end-stage renal disease (ESRD).

CASE REPORT

9 month old baby girl presented with acute kidney injury, anasarca, vomiting, loose stools and anemia. Serum Creatinine 9.27 mg/dl. Renal biopsy was done.

RESULTS

Renal biopsy showed Oxalate Nephropathy.
Clinical exome sequencing was run and detected presence of two clinically relevant variants in the AGXT gene with heterozygosity. Suggestive of Primary hyperoxaluria type 1.

CONCLUSION

The diagnosis of PH1 was established by identification of pathogenic variants in AGXT on molecular genetic testing.PH1 is inherited in an autosomal recessive manner therefore carrier testing for at-risk relatives and prenatal testing for a pregnancy at increased risk should be mandatory if both pathogenic variants have been identified in a family.

Acute phosphate nephropathy caused by epithelial malignancy

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BACKGROUND

Acute phosphate nephropathy (APN) is an uncommon disease that is known to occur in patients exposed to high doses of oral sodium phosphate for bowel preparations. However, there are other less known sources of phosphate that can cause APN. We describe a case of APN due to epithelial malignancy.

CASE SUMMARY

A 73-year-old hypertensive female presented with lower backache and myalgia for 1 month. She was treated with etoricoxib, proton pump inhibitors and parathormone injection. The patient had worsening kidney function and her serum creatinine levels increased from 1.2 – 7.10 mg/dl over a period of 1 month. She had sub-nephrotic range proteinuria. Autoimmune and complement work-up was negative. The patient has a breast lesion with biopsy features suggestive of infiltrative breast carcinoma with signet ring morphology. In view of multiple drugs intake, a clinical diagnosis of acute tubule-interstitial nephritis was kept and the patient underwent kidney biopsy.

Kidney biopsy showed several tubules containing granular to onion shell-like material indicating calcium phosphate calcification, which were highlighted on von-Kossa stain. These phosphate calcifications are also noted in occasional glomeruli inciting intraglomerular giant cell reaction. Apart from this, no significant glomerular pathology noted. No immune deposits were observed on direct immunofluorescence. A diagnosis of APN was established. Patient was kept on hydration. On follow-up after 2 months, her serum creatinine returns to 1.48mg/dl.

CONCLUSION

This case highlighted that APN may occur with other sources of phosphorus, highlighting the importance of good history taking and kidney biopsy in patients with predisposing factors for APN. Raising awareness on the possibility of APN and its timely recognition and management is imperative so that appropriate measures can be instituted to prevent or delay its progression to end stage renal disease.

Abbreviations – APN – Acute phosphate nephropathy

CASE REPORT

Renal Intravascular Large B-cell Lymphoma- A case report.

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Background: Intravascular large B cell lymphoma is a rare subtype of non-Hodgkins's lymphoma according to current WHO classification. Overall, the incidence is 0.95/100

Case report: Renal biopsy was done in a 66-year-old patient who had presented with complaints of fever with intermittent chills and body pain for 2 months. He also had history of Malena and loss of weight for 2 months. On evaluation patient had nephrotic range proteinuria. Renal biopsy showed atypical lymphoid cells in the glomerular capillary wall and also in occasional peritubular capillaries. On Immunohistochemistry study, atypical lymphoid cells were positive for CD20, confirming diagnosis of Intravascular large B cell lymphoma.

Conclusion: Intravascular large B cell lymphoma are aggressive lymphoma associated with poor prognosis, which requires high index of suspicion for diagnosis.

RENAL PRIMITIVE NEUROECTODERMAL TUMOR [PNET]: A RARE ENTITY

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INTRODUCTION:

Primitive Neuroectodermal Tumor [PNET] is a malignant small round cell tumor and typically arises from bone and soft tissue in adolescents and young adults. Renal PNET is an extraordinarily rare and exhibits highly aggressive biological behaviour with poor prognosis.

CASE REPORT:

A 16 years old female presented with complaint of left flank pain for last 2 months. Computed tomography scan revealed heterogeneously enhancing lesion in left kidney. Left nephrectomy specimen was received. On cutting open tumor was seen reaching upto pelvicalyceal system and showing grossly grey white to tan necrotic and haemorrhagic area measuring 8 x 9 x 5 cm. Sections examined revealed clusters, sheets and nests of tumor cells with high N:C ratio, scanty cytoplasm, pleomorphic nucleus with inconspicuous nucleoli. Numerous rosettes were also noted. Tumor was infiltrating the renal pelvis, perinephric fat and Gerota's fascia. Sections from ureter show infiltration by tumor cells. Lymphovascular Invasion also present. On immunohistochemistry, tumor cells were membranous positive for CD99 and NSE, patchy positive for Vimentin and negative for CK, CD10, CD56, Synaptophysin, WT1, PAX-8. Based on histomorphological features and IHC, a diagnosis of Primitive Neuroectodermal Tumor [PNET] was suggested.

CONCLUSION:

Renal PNET is a rare renal malignancy that should be kept in differential diagnosis of renal space occupying lesion especially when presenting in adolescent and young adults. It has a very aggressive course and multimodal therapy has to be considered in its treatment.

CASE REPORT

A rare association of Wilms tumor with incidentally detected renal tuberculosis

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AIIMS Manglagiri

Background: Fine needle aspiration cytology (FNAC) helps in the early diagnosis of paediatric renal tumours, especially in centres where preoperative chemotherapy is advocated in Wilms' tumour. There is a controversy regarding its role in operable paediatric renal tumours. Both SIOP (International Society of Paediatric Oncology) and NWTS (National Wilms' Tumour Study Group) state that a core biopsy increases the risk of relapse; as tumours biopsied are upstaged. SIOP protocol advocates a radiological or FNAC diagnosis permitting preoperative chemotherapy first, followed by surgery and further therapy. Rapid onsite evaluation cytology further aids in the rapid diagnosis. We report a rare case of pediatric Wilms tumor diagnosed on cytology with incidentally detected renal tuberculosis.

Case Report: We report a case of 3-year-old female child who presented with left flank swelling for 1 week. On examination an, ill-defined diffuse mass of 5 x 5 cm size was palpable. On ultrasonography, a lobulated hypoechic solid-cystic mass lesion was identified at the lower pole of left kidney with mild hydronephrosis. Child has given a single dose of vincristine based on the radiological diagnosis soon after which she developed high grade fever not responding to medication. FNAC was advised and hence a rapid onsite evaluation was done. Smears showed several linear, beaded negative shadows for which acid-fast bacilli staining was performed which was positive to our surprise. Soon patient was taken up for radical nephrectomy after the diagnosis of small round blue cell tumor with associated Tuberculosis infection. Specimen was received in the pathology department for Histopathological evaluation.

Conclusion: It's one of the kinds of rare case reports of Wilms tumor associated with incidentally detected renal tuberculosis.

"Unraveling the Enigma: Non-Lupus Full House Nephropathy - A Comprehensive Exploration"

Sharumathi. E, Dr. Aasma Nalwa1, Dr. Vikarn Vishwajeet1, Dr. Vikrant Verma1, Dr. Deepika Gupta1, Dr Ayushi Saxena1, Dr. Manish Chaturvedy2, Dr. Nitin Kumar Bajpai2, Dr. Rajesh Jhorawat2.

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Background: Full-house nephropathy (FHN) is characterized by an immunofluorescence pattern displaying simultaneous deposition of immunoglobulins - IgG, IgA, and IgM - along with complement system components - C3 and C1q - in renal tissue, primarily in glomeruli and occasionally impacting tubules and vessels. We comprehensively analyzed adult cases of non-lupus full-house nephropathy, studying etiology, clinical presentation, and outcomes, comparing with lupus nephritis.

Aim: Our aim in undertaking this study was to investigate the clinicopathologic spectrum of these "full-house" nephropathies (FHN) that do not stem from lupus nephritis.

Case report: In Case 1, a 64-year male presented with two-week history of fever, two days of hematuria, pedal edema, abdominal distension, and non-blanching purpuric rash on limbs. Case 2, a 55-year female, with fever for five days, along with skin rashes, pedal edema, and shortness of breath. Case 3, a 52-year male, presented with recurrent vomiting, lower limb and periorbital edema, headache, fever, and burning micturition for five days, coupled with decreased urine output.

Case	Age/sex	Proteinuria	S.Creatinine (mg/dL)	Light microscopy
1	64/M	3+	2.97	MPGN with **fibro cellular** crescents
2	56/F	4+	5.1	MPGN with **cellular crescents**
3	53/M	3+	1.9	MPGN with **cellular to fibro cellular** crescents

Results: Three non-lupus FHN patients, aged 40-50 years, with creatinine 2-5 mg/dL, 3+ to 4+ proteinuria; negative ANA, anti-DNA, and ANCA. The prevailing light microscopy pattern observed was membranoproliferative glomerulonephritis (MPGN), accompanied by crescents and a full-house pattern on immunofluorescence.

Conclusion: Non-lupus full house nephropathy serves as a comprehensive term for instances that do not meet the established criteria for systemic lupus erythematosus (SLE). The etiology and pathogenesis of idiopathic non-lupus FHN are yet to be fully explained. Nonetheless, there is a need for further studies to establish more effective approaches for diagnosing and treating non-lupus FHN.

CASE SERIES

"Diagnostics and Dilemmas: Pathological Insights into Cryoglobulinemic Glomerulonephritis"

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Background: Cryoglobulinemic glomerulonephritis (CGN) is a rare renal disorder with varied etiology and clinical-pathologic manifestations characterized by deposition of cryoglobulins in the glomeruli, leading to inflammation and kidney damage. Most cases present a membranoproliferative pattern, often associated with glomerular intraluminal pseudothrombi. Immunofluorescence (IF) findings vary, reflecting the serum cryoprecipitate composition.

Case series: A retrospective analysis of renal biopsy specimens from patients with the diagnosis of CGN were included. Serum cryocrit levels and histopathological examination, including light microscopy and IF, were conducted to characterize cryoglobulin deposits within the glomeruli. 3 patients of CGN were included in the study; none of the patients had a history of autoimmune disorders. The mean age of presentation was the 3rd to 5th decade, with female predominance and serum cryocrit positivity was seen in one case. Renal biopsy revealed characteristic features, including pseudothrombi in the viable glomeruli with exudative MPGN pattern, vasculitis and positivity of DIF with predominance of one light chain(kappa) and one heavy chain (IgG) with a trace of C3 and C1q.

Conclusion: In conclusion, cryoglobulinemia is a rare clinical entity. Awareness of its clinical spectrum is important because diagnosis is facilitated by the demonstration of cryoglobulins in the serum. Treatment is challenging, given the end-organ damage and frequent relapses. Currently, all treatment options are designed to either treat the underlying disease or to provide immunosuppression, without any emerging disease-specific interventions.

Anti-Glomerular Basement Membrane Disease: Case series of 5 cases, evaluating clinicopathological spectrum of one of the leading causes of RPRF

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Background: Anti-glomerular basement membrane (anti-GBM) disease is a rare autoimmune disorder with autoantibodies targeting NC1 domain of alpha-3 chain of type IV collagen. This leads to rapidly progressive glomerulonephritis (RPGN) and also involves pulmonary capillaries, resulting in pulmonary haemorrhage.

Case Series: In this study a retrospective analysis of clinicopathological characteristics of five patients with diagnosis of anti-GBM disease was done (mean age: 41 years; age range: 22-73 years). All the cases showed linear deposits for IgG along the glomerular capillary walls on immunofluorescence, along with linear deposits for C3, Kappa and Lambda. However, ELISA for Anti-GBM antibodies was positive in four of these cases only. One case also showed associated MPO ANCA antibodies, and the biopsy from this patient revealed crescents in all the 6 viable glomeruli, while three glomeruli were globally sclerosed and there was fibrosis of 60% of the biopsied cortical parenchyma. Most common clinical manifestations were fever, reduced urine output, and edema. Laboratory data showed mean creatinine of 9.3 mg/dl (range: 3.5 to 18.8 mg/dl). The number of glomeruli with crescents ranged from 8% to 100 %, with a mean of 68.7% glomerular involvement. The crescents were mostly cellular and fibrocellular, with fibrous crescents also noted along in one case. Segmental fibrinoid necrosis of the tuft was noted in one case (involving 28% of the glomeruli). None of the cases showed endocapillary hypercellularity. Interstitial fibrosis and tubular atrophy ranged from 35% to 60% with average of 47%.

Conclusion: Cases of Anti-GBM disease present with RPRF. Double seropositivity for Anti-GBM and ANCA is associated with more interstitial fibrosis and tubular atrophy, and a worse outcome as noted in this study as well as on literature review. Distinguishing between anti-GBM disease and other causes of RPGN is essential for appropriate management.

CASE SERIES

"Unveiling PGNMID: Understanding a Rare Renal Disorder"

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Background: Proliferative glomerulonephritis (GN) with monoclonal IgG deposits (PGNMID) is part of monoclonal gammopathy of renal significance (MGRS) affecting middle-aged to elderly patients who present with proteinuria, haematuria and renal insufficiency.

Case report:

Case 1: 34-year female presented with anasarca, nephrotic range proteinuria, raised serum creatinine and axillary lymphadenopathy. Renal biopsy showed focal segmental glomerulosclerosis, severe acute tubulointerstitial nephritis and capillary wall granular deposits of IgG with kappa restriction on immunofluorescence (IF).

Case 2: 49-year female presented with macroscopic haematuria, proteinuria and renal dysfunction. Renal biopsy showed acute diffuse exudative glomerulonephritis with irregular, capillary wall and mesangial granular deposits of IgG, C3, kappa and lambda on IF. The subsequent renal biopsies showed diffuse proliferative glomerulonephritis with crescents and secondary segmental sclerosis with deposits of IgG, C3 with kappa light chain restriction on IF suggestive of PGNMID.

Case 3: 32-year female presented with oedema, haematuria and was clinically suspected as Lupus nephritis. Renal biopsy showed mild interstitial fibrosis and glomerular immune complex deposition of IgG with lambda light chain restriction in mesangium and glomerular capillary wall on IF. Ultrastructural examination showed occasional subendothelial and subepithelial electron dense deposits.

Conclusion: While PGNMID can mimic glomerulonephritis of various etiologies clinically, a meticulous evaluation of the patient entailing correlation with renal biopsy features and IF findings is essential to reach an accurate diagnosis.

Vasculotoxic Snake Bites: Effect on clinical and histopathological findings in kidney

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Background: Snake bite envenomation is a major public health concern in India. Vasculotoxic snakes like Viper, Russell's viper and saw-scaled viper cause haemolysis and acute kidney injury (AKI).

Aim: This study was done to evaluate the clinical, biochemical and histopathological findings on renal biopsy of patients with history of vasculotoxic snake bite.

Methods: We performed a retrospective analysis of renal biopsies with history of vasculotoxic snake bite at AIIMS Jodhpur from July 2021 to December 2023. Relevant data was collected from HIS and clinical and histopathological findings were evaluated.

Results: The study included three cases biopsied for the presentation with abrupt derangement of renal parameters and anuria after vasculotoxic snake bite. Haematological findings showed anaemia, leukocytosis and thrombocytopenia with presence of schistocytes in all three cases. Mean LDH levels were 1136 IU/L, and the average serum creatinine and BUN levels were 5.98 mg/dL and 80 mg/dL respectively. Sub-nephrotic 24-hour proteinuria was seen in all cases with a mean 24- hour proteinuria of 1.055 g/dl. All three cases required haemodialysis. Haematuria was present in 2 out of three cases (66%). There was patchy cortical necrosis in 1 case, and diffuse cortical necrosis was noted in other case. IFTA was mild in 2 cases and could not be interpreted in 1 case due to presence of cortical necrosis. There was moderate interstitial inflammation (comprising of chiefly lymphocytes and plasma cells, along with admixed neutrophils) in two biopsies with viable preserved part of cortical tissue, however, this could not be assessed in the biopsy with diffuse cortical necrosis. In 1 of the cases, hemosiderin pigment was noted in interstitium and tubules (highlighted with Perl's stain). No immune complex or complement deposits were noted on immunofluorescence microscopy. The review of literature Sanjay et al (2017), clinico-pathological spectrum of snake bite-induced acute kidney injury from India showed ATN along with mild to moderate AIN in 20 cases, moderate AIN in 1 case and patchy RCN in 1 case.

Conclusion: AKI resulting from vasculotoxic snake bites is a severe condition, with a significant proportion of patients being dialysis dependant. The histopathological picture in most cases is of acute tubular necrosis (ATN), renal cortical necrosis (RCN), or acute interstitial nephritis (AIN).

CASE SERIES

Cystic Diseases of Kidney: a clinicopathologic study of five cases

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Background:
Cystic diseases of kidney are uncommon disorders and are heterogeneous in origin, pathogenesis, and distribution. Herein we describe the pathological findings of cystic kidney disease from our Institutional database.

Case details
A total of five cases were retrieved over a period of 1 year. Three of them were infants, while two cases were adult. Two cases had history of consanguineous marriage and also strong family history of similar complaints in siblings. Of the three infants, two had autopsy tissue available and both were diagnosed with multicystic dysplastic kidney. These two cases had associated extrarenal congenital malformation- one had VACTERAL (Vertebral anomaly, Atresia, Cardiac hypertrophy, Tracheo-Esophageal fistula with intestinal atresia, polycystic renal malformation, and Limb anomalies) syndrome and another had congenital hepatic fibrosis. One infant case had renal biopsy, which showed feature consistent with autosomal recessive polycystic kidney disease. Two adult cases were diagnosed on renal biopsy and both of them presented with unexplained renal failure. On light microscopy, diagnosis of glomerulocystic kidney disease was offered in both cases while direct immunofluorescence results were negative. One case in 22-year female had bilateral bronchiectasis, primary amenorrhea, and primary sensorineural hearing loss. Molecular testing revealed mutation in DNAH1 which encodes an inner dynein arm heavy chain.

Conclusion:
The study reports the clinical and pathological features of cystic kidney disease and supports the heterogenous nature of these lesions. Histopathological examination and genetic testing are essential for accurate classification of these lesions and for guiding clinical management decisions.

Post-transplant malignancy in the native kidney of renal transplant recipient; Single center experience.

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Background: Post-transplant malignancy (PTM) is an important complication of kidney transplantation. Incidence & type of PTM differ considerably depending on geographic location & ethnicity. Increased risk of PTM in native kidney is multifactorial, complex & changes in immune surveillance.

Aim: To describe 3 cases with of PTM in native kidney of renal transplant recipients with associated history & prognosis.

Methods: We reported 3 cases of PTM in native kidney of renal transplant recipients from July 2019 to July 2023. Patients' records were evaluated for age, date of transplant, past history, history of immunosuppressive therapy, laboratory, radiological & histopathological reports.

Results: Cases include 3 males ranging from 28 to 65 years of age. Histopathological diagnosis included renal T-cell lymphoma, high grade invasive urothelial carcinoma of the renal pelvis & papillary renal cell carcinoma. Mean ± SD age at time of kidney transplant was 37±16.39 years (range=18-58 years), Age at renal malignancy diagnosis was 46.67±15.11 years (range=28-65 years). Mean time from transplantation to diagnosis of renal malignancy was 120±23.55 months. Mean time on pretransplant dialysis was 76±33.12 months. 2 patients presented with clinical symptoms of flank pain & hematuria at time of diagnosis. In 1 patient, tumor was detected in incidental findings on ultrasound. 2 patients died within 1 week of surgery due to septic shock, suggests poorer prognosis.

Conclusions: PTM in native kidney is rare clinicopathological finding in transplant recipients & has poorer prognosis. We hereby suggest regular radiological screening of native kidneys in post-transplantation that could be beneficial in early detection of PTM.

Starfruit induced Oxalate nephropathy – A case series

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Background: Starfruit induced oxalate nephropathy remains an under-recognized cause for acute and chronic kidney disease. Star fruit (Averrhoa carambola), belongs to the same Oxalidaceae family as A.bilimbi, already known for its potential to cause oxalate induced nephrotoxicity. Both are popular in many tropical countries as indigenous medications especially for diabetes. Starfruit is also rich in oxalic acid and caramboxin, a potential neurotoxin. In 2016, Barman et al reported starfruit associated acute kidney injury (AKI) from India for the first time. However, the available literature on this important community health problem is very limited.

Case reports: Here, we report 3 cases of acute renal failure associated with starfruit ingestion that we encountered over a period of one year.

Case 1: 38/male; no comorbidities, presented in acute renal failure - serum creatinine 9.9mg%.

Renal biopsy: Severe acute tubular injury (ATI) with crystals having fractured glass appearance consistent with oxalate.

Case 2: 73/female, known diabetic for 15 years, presented with acute worsening of serum creatinine from 2.5mg% to 7.1mg%.

Biopsy: ATI with oxalate crystals in a background of marked global glomerulosclerosis and tubular atrophy.

Case 3: 44/male, presented with rapidly progressive renal failure, serum creatinine 11.5mg%. Clinically, NSAID induced acute interstitial nephritis was suspected.

Biopsy: Marked ATI with oxalate crystals.

The history of starfruit ingestion was obtained only on detailed interrogation in all cases.

Conclusion:

Community acquired AKI due to ingestion of toxic herbs and other natural remedies for health benefits poses a major challenge in developing countries. Starfruit (Averrhoa carambola) and its juice are very popular in and around the Indian subcontinent as an indigenous medicine for diabetes. Star fruit intoxication is potentially life-threatening and can be easily missed unless this history is specifically sought, especially in patients with unexplained AKI and/ or neuropsychiatric symptoms. Timely referral, prompt treatment with intensified haemodialysis, close monitoring and follow up are warranted and baseline renal function determines renal recovery.

A public health approach, including campaigns to raise awareness about safe, scientific and judicious use of alternative remedies as well as seeking timely medical help might help improve overall outcome.

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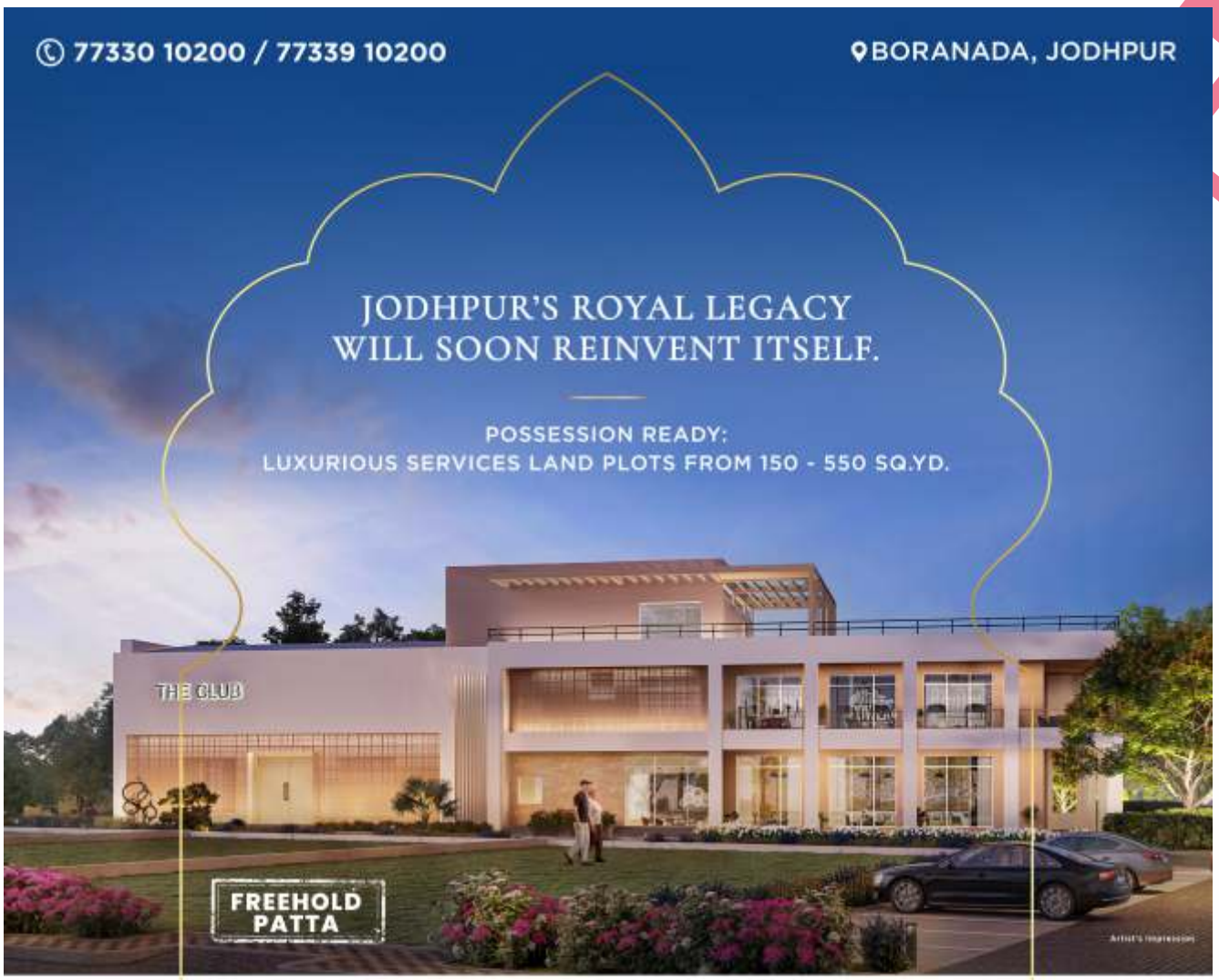

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