



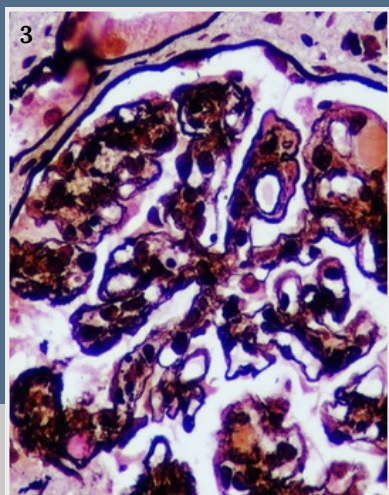
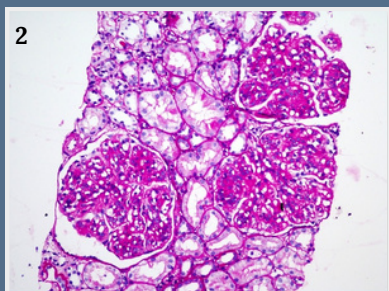
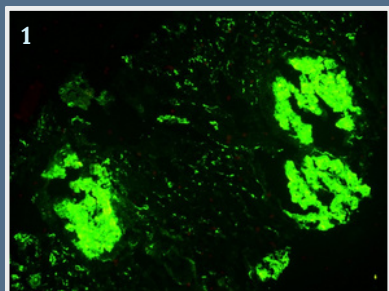
KIDNEY KOLUMNNS

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KIDNEY KOLUMNNS

VOL 3 | ISSUE 3 | JULY TO OCTOBER 2025

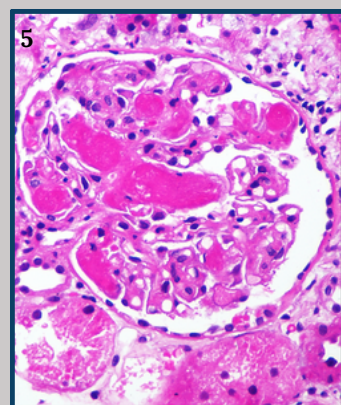
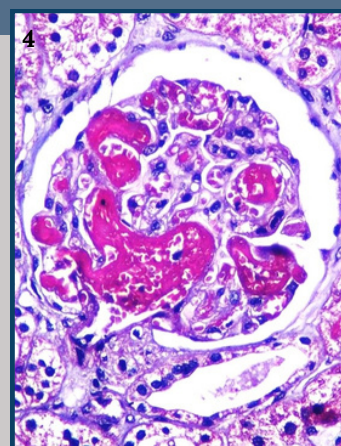


Dear Readers,

We are delighted to bring you this edition of Kidney Kolumns, the official newsletter of the Indian Society of Nephrology. The theme of this issue is Complement-Mediated Kidney Diseases—a rapidly advancing area that is redefining our understanding of glomerular disorders. The complement system has emerged as a critical driver not only in rare syndromes but also in many common renal conditions. In this issue, our experts highlight new insights into pathophysiology, diagnostic challenges, genetic underpinnings, and emerging therapies, with a special focus on complement inhibitors that are transforming patient care. We hope these perspectives will stimulate discussion and inspire further exploration into this fascinating domain.

We also take this opportunity to welcome you to **ISNCON 2025**, scheduled from **18th-21st December** in **Lucknow**. The meeting promises rich scientific exchange, collaboration, and warm hospitality in the historic city of Nawabs. We look forward to seeing you there!

Warm regards
Editors



As always, we welcome your **feedback & contributions** at **education@isn-india.org**

Kidney Kolumns is the official newsletter of the Indian Society of Nephrology (ISN). Launched in 2023, it is published quarterly and serves as a vibrant platform for the nephrology community to exchange knowledge, share updates, and foster professional growth.

The newsletter currently accepts contributions by invitation only. All content is reviewed by the editorial board prior to publication. However, student contributors are encouraged to submit their work without an invitation and should contact the student editors for further guidance.

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Kidney Columns

SECRETARY'S DESK

Dear members,

Greetings from the ISN Secretariat!

The 54th Annual Conference of the Indian Society of Nephrology ISNCON 2025 is going to take place between 18th - 21st December 2025 at SGPGI Convention Centre, SGPGI Campus, Lucknow.

I want to highlight some of the key attractions of ISNCON 2025.

1. This year, ISNCON will offer numerous workshops on a pre-conference day (December 18th) for our members, featuring renowned National and International faculty.



Dr Shyam B Bansal
Honorary Secretary,
Indian SN

You can register for one of the workshops

1. **Intervention Nephrology** - Dr Tushar Vacharajani
2. **Critical Care Nephrology and POCUS** - Dr R Murugan, Dr Rupesh Raina and Dr Vandana Dua (US)
3. **Onconeurology and Histopathology** - Dr Rimda Wanchoo and Dr Prakash Gudsoorkar (US)
4. **Pediatric Nephrology** - Dr Rulan Parekh, Dr Ajay Sharma (Canada), Dr Rukshana Shroff (UK), Dr Carla Nester (US)
5. **Transplant Immunology** - Indian Faculty

Registration for workshops is complimentary; however, the seats are limited.

Main Scientific Program

We have renowned international and national faculty -

1. ASN-Indian SN joint session on CKM and Transplantation - Dr Katherine Tuttle,

Dr Prabir Roy Chaudhry, Dr Balram Bhargava, Dr Madhuri Kanitkar, Dr Amit Govil, Dr Vasistha Tatapudi

2. ISGD - Indian SN Joint symposia on Glomerular diseases - Dr Carla Nester, Dr Suceena Alexander, Dr Koyal Jain and Dr Rupesh Raina
3. ERA-Indian SN Joint session on ADPKD, Genetics - Dr U Muller and Dr Mark Valvelot
4. Dr Jai Radhakrishnan (Editor KI) - Membranous Nephropathy, MGRS
5. Dr Rajnish Mehrotra (Editor JASN) - Peritoneal Dialysis
6. Dr Silvi Shah - Pregnancy and AKI
7. Dr Katherine Tuttle - CKM and GLP-1 Agonist

We have received the maximum number of abstracts, more than 500 this year

Do not miss this opportunity to be part of this international event and register for

ISNCON 2025 at www.isncon2025.com

Looking forward to seeing you at Lucknow in December.

Thrombotic Microangiopathy

IN PREGNANCY-ASSOCIATED ACUTE KIDNEY DISEASES

Dr Arun Prabhakar, Assistant Professor in Nephrology

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PGIMER, Chandigarh

Thrombotic microangiopathy (TMA) is a clinicopathologic process in which microvascular injury leads to a characteristic triad: microangiopathic hemolytic anemia (MAHA), thrombocytopenia (platelet count $<150 \times 10^9/L$), and end-organ dysfunction. MAHA is suggested by falling hemoglobin, elevated lactate dehydrogenase (LDH), reduced or absent haptoglobin, and schistocytes on the peripheral smear, in the absence of immune-mediated hemolysis on a direct Coombs test. End-organ damage predominantly affects the kidneys, nervous system, and heart.

The incidence of pregnancy-related acute kidney injury (PrAKI) ranges from 40 to 100 per 10,000 pregnancies, and despite major improvements in maternal health care - even in resource-limited nations - the occurrence of PrAKI has not shown a substantial decline in

recent years. In low- and middle-income countries (LMICs) such as India, the underlying etiology is often not systematically evaluated due to: (1) deliveries occurring in peripheral obstetric centers with limited diagnostic facilities, (2) the risk of bleeding associated with kidney biopsies during pregnancy and postpartum, and (3) delays in referral or transportation to tertiary centers capable of managing severe AKI (4) limited awareness of conditions such as TMA. Events like hemorrhage, infections, and surgical stress can trigger complement activation and precipitate TMA, yet are frequently misattributed as the direct causal factors.

TMA as a cause of PrAKI is underreported. A recent review of Indian studies cited sepsis, pregnancy-induced hypertension (PIH), and hemorrhage as



leading causes; however, biopsy data from the same review revealed that among 346 cases, acute cortical necrosis (ACN) (49.8%) and TMA (13.5%) were major pathologies. ACN represents the morphological outcome of fulminant TMA. In our series of 21 patients presenting with ACN during pregnancy, all had evidence of hematological or biopsy-confirmed TMA, despite 19 showing clinical features of sepsis, hemorrhage, or PIH.

In pregnant or postpartum women developing acute MAHA with thrombocytopenia, the key differential diagnoses include preeclampsia with severe features/Hemolysis, Elevated Liver Enzymes and Low Platelets (HELLP) syndrome, thrombotic thrombocytopenic purpura (TTP),

and pregnancy-associated TMA (P-TMA (Refer to Table 1). Clinical overlap is common, but response to delivery aids differentiation: resolution suggests PE/HELLP, while persistence or progression of MAHA and kidney dysfunction indicates TTP or P-TMA. Severe thrombocytopenia with minimal renal involvement favors TTP, whereas severe AKI with anuria is more typical of P-TMA. P-TMA is characterized by MAHA, uncommon neurological involvement, and hypertension usually developing after AKI onset, unlike HELLP syndrome, where hypertension triggers AKI. A serum creatinine >1.9 mg/dL and LDH >600 U/L at 72 hours postpartum has >95% specificity for differentiating P-TMA from HELLP syndrome.

Feature	Pre-eclampsia/HELLP	TTP	P-TMA
Time of occurrence	Usually, late pregnancy starts resolving within 24–36 h of delivery	May occur throughout pregnancy, often near term and persists postpartum	Near-term and postpartum
MAHA	Moderate	Severe	Moderate to severe, often transient
Kidney Involvement	Mild to moderate	Minimal	Severe AKI, often anuric
Liver function tests (AST, ALT)	Markedly elevated	Normal or slightly elevated.	Normal or Markedly elevated (in early stages)
Neurological Symptoms	If associated with seizures	Prominent (confusion, seizures)	Rare

Feature	Pre-eclampsia/HELLP	TTP	P-TMA
Hypertension	Usually present early	May be absent	Develops after AKI onset
LDH / Creatinine	Moderately elevated	Elevated LDH, creatinine often normal	LDH >600 U/L, Creatinine >1.9 mg/dL
Diagnostic tests	sFLT/PlGF ratio	ADAMTS13 activity <10%	Genetic and functional testing for regulators of ACP
Response to Delivery	Rapid resolution	Symptoms persist	Usual onset is after delivery
Treatment	Delivery, supportive care	PLEX, immunosuppression if inhibitors are present	Complement inhibitors +/- PLEX

TABLE 1

Abbreviations:

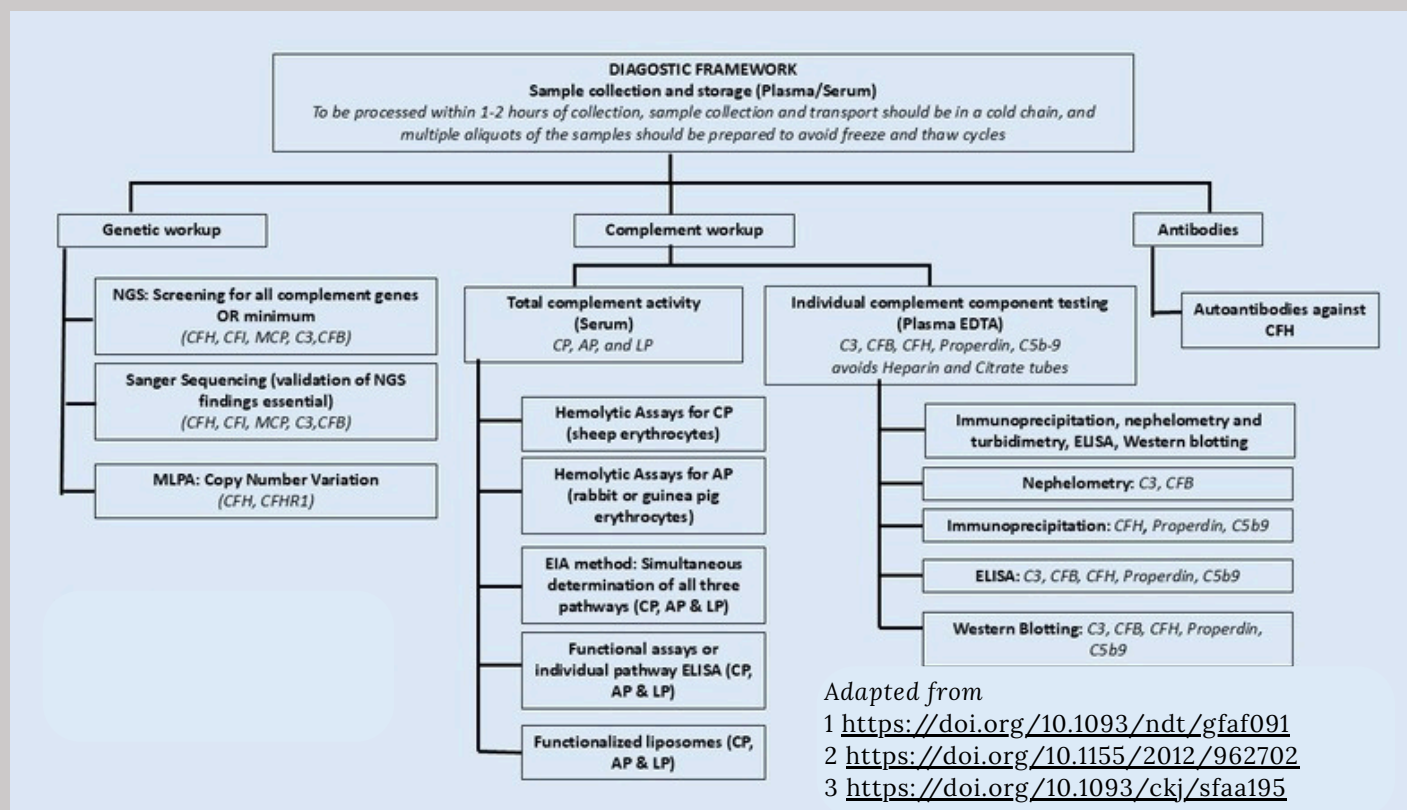
ACP – Alternative Complement Pathway; ADAMTS13 – A Disintegrin And Metalloproteinase with Thrombospondin Type 1 Motif, Member 13; AKI – Acute Kidney Injury; ALT – Alanine Aminotransferase; AST – Aspartate Aminotransferase; HELLP – Hemolysis, Elevated Liver Enzymes, and Low Platelets; LDH – Lactate Dehydrogenase; MAHA – Microangiopathic Hemolytic Anemia; PLEX – Plasma Exchange; P-TMA – Pregnancy-associated Thrombotic Microangiopathy; sFLT – soluble fms-like Tyrosine Kinase-1; PlGF – Placental Growth Factor; TTP – Thrombotic Thrombocytopenic Purpura

Table updated from <https://doi.org/10.1182/asheducation-2015.1.644>

Normal pregnancy is associated with heightened but physiologically regulated complement activity, both systemically and locally within the placenta. In normal pregnancies, C3 rises from 93.5 to 119.9 mg/dL (~28%) and C4 from 35.7 to 56.7 mg/dL (~59%) between the first trimester

and 36 weeks. Loss of feto-placental complement regulators after delivery may unmask maternal complement dysregulation, explaining the high incidence of P-TMA in the postpartum period.

Evidence from studies in Western Europe



suggests that TMA in pregnant patients is complement-mediated, based on genetic testing, therapeutic response, and clinical outcomes. Fakhouri et al. (pre-eculizumab) reported abnormally activated complement in P-TMA, with >60% progressing to End-Stage Kidney Disease (ESKD) by month 1, predominantly postpartum. Complement evaluation at a median of 19 months from onset revealed abnormalities in 86%, most commonly CFH mutations (45%). Similarly, in the study by Bruel et al., 56% of P-TMA cases had genetic mutations, most commonly in the CFH gene. In an Indian cohort of 17 patients with P-TMA (Kandori et al.), 15 patients (88.2%) had complement abnormalities, entirely due to CFHR3-CFHR1 deletions (homozygous = 4, heterozygous = 11), with no CFH, CFI, C3, or MCP variants detected. Notably, heterozygous CFHR3-CFHR1 deletion is reported in one-third of the healthy individuals

(DOI: 10.2215/CJN.01260212), therefore, its high background frequency limits its causal attribution to genetic TMA. In our series of 15 ACN patients, 9 demonstrated low C3 levels and/or reduced alternative pathway functional activity. These findings signals the role of dysregulated alternative complement pathway leading to P-TMA, though limitations include small sample sizes, delayed complement testing, lack of functional correlation, population-specific differences, and absence of control groups.

In P-TMA, MAHA may go unrecognized due to its transient nature, late presentation, or when patients are first evaluated in the context of ESKD or during pre-transplant assessment. An ideal evaluation after P-TMA, including all cases with MAHA and/or ACN diagnosed by radiology or kidney biopsy demonstrating TMA or ACN confirmation, includes genetic analysis of complement

proteins and functional testing (at least soluble membrane attack complex [sC5b-9] and AH50). The validated Next Generation Sequencing (NGS)-based panel includes 15 genes: ADAMTS13, C3, CD46, CFB, CFH, CFHR1-5, CFI, DGKE, THBD, MMACHC, and PLG. A diagnostic framework of P-TMA is given in Figure 1.

Before C5 inhibitors, PLEX was the mainstay for P-TMA, although efficacy was limited. C5 inhibitors, including eculizumab and ravulizumab, improve renal outcomes, with eculizumab relatively safe in pregnancy and lactation. Most evidence comes from observational and retrospective studies. In

India, eculizumab is selectively (free) available through designated Centres of Excellence under the National Policy for Rare Diseases; however, PLEX remains frequently used. Careful patient selection and vaccination are essential, as infections can complicate the course during C5 inhibitor therapy. For patients who recover from AKI, subsequent pregnancies are possible. For those who progress to ESKD, kidney transplantation is an option. In both scenarios, comprehensive testing, proper counselling, continuous monitoring, and individualised therapeutic interventions, potentially including C5 inhibitors based on genetic or functional risk, are advised.

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Diagnostic Tests

IN THROMBOTIC MICROANGIOPATHY

Dr Valentine Lobo, Head of the Department of Nephrology, KEM Hospital, Pune

The International Society of Nephrology Hemolytic Uremic Syndromes International Forum's 2023 statement has pointed out that current terminology of atypical HUS is confusing and does not reflect underlying pathogenesis. Thrombotic Microangiopathy (TMA) with qualifiers indicating the possible cause would be a more appropriate and inclusive term. The priority therefore is recognition of a clinical syndrome consistent with TMA, and confirming the presence of microangiopathic hemolysis (MAHA) [Hemoglobin level <10 g/dl, LDH serum level >1.5 times the upper limit of normal, undetectable serum haptoglobin, negative DCT, and the presence of schistocytes on a peripheral blood film], followed by elimination of systemic infection, malignancy, or autoimmune disease by history and accessible clinical tests early, as secondary TMAs require specific treatments like control of blood pressure or elimination of offending drugs and rarely warrant a search for a genetic cause which is no more frequent

than in the general population. Common clinical conditions like pregnancy or organ transplantation may be a second hit or trigger in a susceptible individual.

Other clinical situations with a high likelihood of TMA are shown in the box Box 1.

Thrombocytopenia and hypertension

End-organ damage with a predilection for the renal, cardiac, and nervous systems

Renal limited TMA (without any hematological manifestations), often associated with autoimmune diseases, drugs, or solid organ transplantation or Incomplete presentations

Evidence of alternate pathway complement consumption (low C3 with normal C4)

Proteinuria, hematuria and casts

Post partum or pregnancy related AKI

Snake bite with AKI without DIC

Post solid organ or stem cell transplant

Any unexplained AKI in adults or pediatric cases including the 1st year of life.

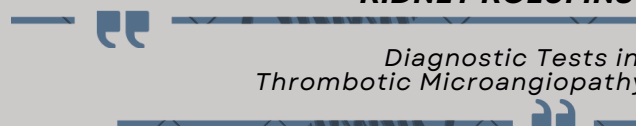
In rare situations (organ limited) a renal biopsy with characteristic histological findings may be required for the diagnosis but this is an exception.

Investigations in a case of TMA should

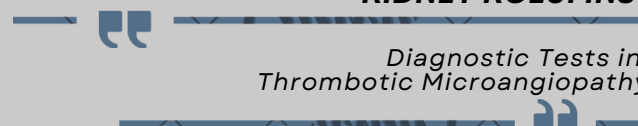
identify the underlying etiology/pathogenic mechanism as well as rule out the numerous mimics of TMA. Tests which estimate the state of complement pathway activation including autoantibody estimation should be conducted on samples drawn prior to initiating therapy as both plasma related interventions and complement inhibitors interfere with the interpretation of these results. In contrast genetic testing is uninfluenced by treatment, rarely required urgently and may even be withheld in patients where an autoimmune or secondary cause is revealed.

Table 2 lists the recommended tests in a case of TMA along with special precautions regarding their use

Test	Sample Required	Result Interpretation	Comment
Culture. rtPCR for Stx genes.	Stool/rectal swab	Indicates an infection related TMA and usually requires only supportive treatment ⁴	Also triggers MCP related disease and possibly complement pathway dysregulation in predisposed individuals
PCR/serology for influenza A/B, CMV, EBV, HIV4	Serum	Infection related or triggered TMA	May require a search for an underlying genetic abnormality especially if evidence of persistent complement dysregulation.
Blood/CSF culture, 16S rRNA PCR Thomsen-Friedenreich antigen	Blood in appropriate medium Serum		DCT may be positive early in which case plasma therapy should be avoided.



Test	Sample required	Result Interpretation	Comment
ANA/Anti DNA, APLA, Anti Scl 70, ACA	Plasma/Serum	Indicate a secondary TMA and treatment should be directed towards the appropriate cause.	
ADAMTS 13 activity and antibodies	Citrated plasma transported at -70oC ideally	Establishes the diagnosis of TTP which requires plasma exchange and/or immunosuppression	A Bethseda assay establishes the presence of a functional inhibitor. If absent genetic testing is required
Anti FH antibodies,	Serum ideally on ice or at -20oC	<u>Commonest cause of childhood HUS in India</u> , probably significant in adults also. <u>Titers and Factor H levels determine prognosis and can be monitored for response, relapse probability, and effect of immunosuppression</u>	Requires a reference or standardized method including sample specific background estimation
MCP expression on PBMC by FACS	EDTA whole blood sample	Homozygous and heterozygous variants associated with decreased MFI in patients with decreased function. However a second hit or additional risk factor including risk	<u>Useful rapid screening test prior to WES, gives an idea of prognosis(good) and need for anti complement therapy</u>



Test	Sample required	Result Interpretation	Comment
		haplotype is required to cause disease	
sFlt-1/PlGF	Serum	Mandated in post partum /pregnancy associated cases to distinguish HUS from HELLP Syndrome	Invaluable for distinguishing the TMAs of pregnancy which have diametrically different treatments. <u>HELLP only requires delivery of the fetus.</u>
C3, C4, CH50 AH50, Hemolytic Assay	Serum frozen at -80 ° C, preferably within 120 min	Analysis of complement function, confirming alternative pathway overactivity. Levels of C3 associated with prognosis and relapses. Absent CH50 and AH50 activity is seen in patients appropriately treated with Eculizumab. Spurious low level by improper sample handling.	Samples need to be immediately frozen after serum separation, avoiding freeze thawing, and handled on ice and aliquoted on receipt by the laboratory.
Analysis of copy number variation in the CFH/CFHRs genomic region by MLPA Genotyping	EDTA sample	Large duplications or deletions or rearrangements better identified than by massive	Often not reported. Must be asked for.

Test	Sample required	Result Interpretation	Comment
for the risk haplotypes CFH-H3 and MCP ggaac		parallel sequencing Risk haplotypes increase disease susceptibility	
Whole exome/whole genome sequencing	EDTA blood sample	Rare variants (minor allele frequency < 0.1% [population specific]) in complement genes: CFH, CD46, CFI, C3, CFB, CFHR1 and non-complement genes: DAGKE, MMACHC, MTR5, MTHFD121); (TSEN25,6; EXOSC35); C5 polymorphism (p.R885H) which predicts eculizumab non-response	Does not distinguish variants of uncertain significance especially with SNPs. A functional assay or in silico prediction may be helpful.

In conclusion, only a C3 level, ADAMTS 13, anti factor H antibody and CH50/AH50 may need to be processed prior to emergent therapy.

Paediatric Thrombotic Microangiopathy

Prof Aditi Sinha, Professor, Division of Nephrology, Department of Pediatrics, All India Institute of Medical Sciences, New Delhi
Prof Arvind Bagga, Director of Pediatrics & Senior Consultant Nephrology, Indraprastha Apollo Hospital, New Delhi

Hemolytic uremic syndrome (HUS), characterized by microangiopathic hemolytic anemia, thrombocytopenia and acute kidney injury (AKI), is a rare condition that affects children more often than adults [1]. The underlying pathology is thrombotic microangiopathy (TMA), characterized by endothelial injury in multiple vascular beds, including the kidneys. Despite its rarity, it is the leading cause of AKI requiring kidney support therapy (KST) and if untreated, carries high risk of chronic kidney disease (CKD) and mortality [1].

HUS has multiple clinical and genetic phenotypes [1,2]. Worldwide, the chief cause of HUS is that following gastrointestinal infections with Shiga toxin-producing organisms, chiefly Shiga toxin-associated enterohemorrhagic *Escherichia coli* (STEC-HUS), and more rarely, infection due to *Shigella* sp. Infection due to these

organisms results in a dysenteric illness, that might progress to HUS in 10-12% of cases. The diagnosis requires demonstrating STEC on culture and presence of shiga toxin gene(s) on PCR of stool or rectal swab; presence of anti-lipopolysaccharide antibodies against common STEC serogroups may aid the diagnosis. The management of these patients is supportive; almost 50% patients require KST, and approximately 25% show CKD on follow-up. While anecdotal reports suggest activation of the alternate complement pathway in the pathogenesis of STEC-HUS, prospective studies have failed to show benefit of pharmacologic C5 blockade on disease course. This phenotype of HUS is currently uncommon in India and South Asia [2].

The second most common phenotype of HUS is atypical HUS (aHUS) [1,2]. This condition, characterized by severe AKI, frequent extrarenal involvement,

multiple relapses, and high risk of kidney failure, is increasingly diagnosed in diverse clinical settings in adult as well as pediatric patients. Patients with aHUS present with pallor, oliguria, fatigue and edema, often following a viral infection or prodrome comprising of abdominal pain and vomiting. Seizures, jaundice and/or severe hypertension might be present. Bleeding manifestations are uncommon. Past history of similar illness or family history of HUS-like illness might be elicited. Dysregulation of the alternate complement pathway, caused by significant variants in genes encoding factor H (CFH), factor I (CFI), membrane cofactor protein (MCP or CD46), factor B (CFB), or complement C3 (C3) or a hybrid CFH/CFHR gene, is detected in 20-30% cases. Rare genetic defects associated with HUS include mutations in WT1, INF2, HSD11B2, EXOCS3, EXOCS5, TSEN2 and POLR3B [3]. Recognising the role of dysregulation of the alternate complement pathway in pathogenesis of aHUS, the term complement-mediated TMA is preferred [1,3].

Acquired dysregulation of complement, secondary to antibodies to factor H (anti-FH), accounts for 25-55% of pediatric patients, chiefly those presenting at 5-15 years of age [1,2,4]. In India, antibodies to factor H (FH) constitute almost 50% of all pediatric patients with aHUS. This disorder chiefly affects children in school-going age group and is a severe form of illness characterized by severe anemia, thrombocytopenia and frequent need for KST. The condition is uniquely amenable to therapy with intensive plasma exchanges (PEX) that replaces IgG anti-FH antibodies and circulating FH/anti-FH immune complexes with fresh frozen plasma from healthy donors. Clinicians should be aware that 88-92% patients with anti-FH antibodies have a homozygous deletion in CFHR1/3, a

defect that is present in approximately 9% of healthy individuals [1,2,4].

A diagnosis of thrombotic thrombocytopenic purpura (TTP) is considered in patients presenting with severe thrombocytopenia, less severe AKI, and often, prominent neurologic manifestations. Congenital TTP, resulting from congenital deficiency of the von Willebrand protease, ADAMTS13 (a disintegrin and metalloproteinase with thrombospondin type 1 motifs, member 13) is an uncommon cause of TMA in childhood. In contrast, acquired TTP, resulting from autoantibodies to ADAMTS13 is more common than aHUS in adults. The diagnosis of TTP is confirmed by demonstrating low ADAMTS13 activity (<10%) with or without presence of autoantibodies [1,2].

Rare causes of HUS include *Streptococcus pneumoniae* (Sp)-induced HUS, which should be considered in young children (typically <2-yr-old) presenting with invasive pneumococcal disease (sepsis, pneumonia, empyema or meningitis) and diagnosed following demonstration of organism on culture or PCR of blood, bronchoalveolar lavage, cerebrospinal fluid or pleural fluid [1,2]. Disseminated intravascular coagulation (DIC) must be excluded; while useful to confirm exposure of the Thomsen-Friedenreich antigen on cell surfaces, a positive peanut agglutination test is often not feasible. Metabolism-associated TMA refers to defects in cobalamin pathway, which are considered in patients with elevated levels of homocysteine in plasma and methylmalonic acid in urine; however, B12 deficiency must be excluded. The term secondary TMA implies another etiology, e.g., malignant hypertension, systemic lupus, scleroderma, and antiphospholipid syndrome. Coexisting conditions, such as pregnancy, malignancy,

stem-cell and solid organ transplantation, and use of certain medications (calcineurin inhibitors, oral contraceptives or quinine), are common in adult patients. A proportion of such cases might have an underlying inherited complement dysregulation that is triggered by a second 'hit' [1,2].

Guidelines from the Indian Society of Pediatric Nephrology (ISPN) have helped refine the standard of care for aHUS in developing countries [2]. Clinical Practice Recommendations of the International Pediatric Nephrology Association (IPNA) are expected later this year. The diagnosis of aHUS requires careful exclusion of multiple entities, including infections that mimic or trigger HUS, e.g., malaria, leptospirosis and dengue [1,2]. When clinically indicated, one should evaluate for and exclude DIC, TTP, infection-associated and secondary forms. Investigations confirm the triad of microangiopathic hemolytic anemia (presence of schistocytes $\geq 2\%$, increased corrected reticulocyte count, lactate dehydrogenase ≥ 450 U/L, and reduced or undetectable haptoglobin), AKI (as defined by KDIGO) and thrombocytopenia (platelet count $<150,000/\text{mm}^3$); however, the latter may be lacking. Complement levels of C3, but not C4, may be reduced. Measurement of plasma levels of complement factors FH, FI and FB, is neither feasible nor recommended routinely. All patients presenting in childhood or adolescence should be screened for the presence of anti-CFH antibodies, before initiating specific therapy with plasma exchanges or complement inhibitors. While whole exome sequencing is recommended to examine for mutations in six key genes of the complement or coagulation pathway, multiplex ligation-dependent probe amplification (MLPA) is necessary to detect copy number variations (CNV) associated with

hybrid CFH-CFHR [1,2].

All forms of HUS require urgent initiation of both specific measures and supportive care [1,2,5,6]. The latter includes prevention or correction of dehydration (especially in STEC-HUS); avoiding nephrotoxic medications; management of volume overload, hypertension and electrolyte or pH abnormalities; initiation of kidney support therapy, if indicated; transfusions of red blood cells, if necessary; and management of organ-specific complications secondary to neurological, intestinal, pancreatic and cardiac involvement. Treatment of STEC-HUS and Sp-HUS is based around appropriate antibiotic therapy and supportive care. Patients with metabolism-associated TMA (CblC-HUS) require intramuscular hydroxocobalamin. Patients with DGKE or WT1 defects do not benefit from plasma exchanges or complement inhibition. Management of secondary forms of HUS focuses on treating the underlying disease or withdrawing the coexisting exposure [2,5,6].

Specific therapy for aHUS has evolved over the decades. Until recently, intensive PEX comprised the predominant strategy for managing aHUS since it replaces aberrant complement factors and/or removes circulating anti-FH antibodies. Evidence from the Indian registry on pediatric HUS and other cohorts indicates that PEX in combination with immunosuppression is associated with satisfactory outcomes in patients with aHUS associated with anti-FH antibodies. However, the role and optimal duration of PEX is not established for patients with aHUS due to inherited complement defects, many of whom may be refractory to PEX, or relapse following its discontinuation. As such, data from European

registries indicates unsatisfactory medium- and long-term outcomes in patients managed with PEX or plasma infusions alone [1,2,5,6].

Evidence from prospective uncontrolled studies in early 2000s indicated high rates of hematological remission and recovery from AKI following therapy with eculizumab, a monoclonal antibody against C5 that prevents formation of the membrane attack complex. Hence, international guidelines since 2009 recommend that all patients with aHUS receive complement blockade; the only exception is anti-FH associated HUS in which PEX with immunosuppression is considered a satisfactory management strategy [1,6]. Recent data from large European observational cohorts show significantly improved outcomes in the post-eculizumab era, both for aHUS in native kidneys and reduced risk of post-transplant recurrence [3]. While the bulk of data on complement blockade is on eculizumab therapy, its long-acting congener ravulizumab is increasingly preferred for maintenance therapy. Ongoing studies are evaluating the role of therapy with crovalimab, a C5 inhibitor with different epitope specificity than eculizumab, thus allowing for management of individuals with eculizumab-refractory illness due to a rare C5 polymorphism that prevents eculizumab binding to C5 [5,6].

As access to complement blockade improves in India and other developing countries, in a recent review, we have summarized the available evidence and expert opinion on the indications, prescription and duration of complement blockade, keeping in mind the cost of care, risks of infections and feasibility of monitoring [5]. While the use of C5-blockade in India is expected to be limited by lack of reimbursement for most patients, there are high risks of misuse for inappropriate indications, and inadequate dosing or premature discontinuation leading to aHUS relapses. Since therapy carries high risk of serious bacterial infections, adherence to appropriate vaccinations, antibiotic prophylaxis and heightened vigilance must be ensured [5,6].

Patients with aHUS require long-term therapy and follow up for the occurrence of relapses, hypertension, proteinuria and CKD. While early guidelines advocated for lifelong complement blockade, expert groups now agree that cessation of therapy with complement inhibitors is feasible in a significant proportion of cases with underlying genetic defects in complement regulation. For detailed guidance on the evaluation and management of aHUS and use of eculizumab, readers are referred to consensus guidelines and expert opinions [2,5,6].

aHUS - A Patient's Perspective

Dr Kamal Shah

Co-founder, NephroPlus

Founder, Dialysis in India

Founder, The Atypical HUS India

Foundation

Trustee Director, The aHUS Alliance
Action

An accident of genetics is bad enough, but when paired with an accident of geography, it can be disastrous.

In July 1997, at the age of 21, I was diagnosed with atypical Hemolytic Uremic Syndrome (aHUS) – a complement-mediated Thrombotic Microangiopathy (cmTMA), a disease that affects blood vessels, primarily those in the kidneys. The news was shocking: my kidneys had failed, and I needed dialysis right away.

Doctors tried many treatments to save my kidneys, but nothing worked. Eventually, my kidneys reached end-stage failure, meaning I had to rely on long-term dialysis. Even a kidney transplant didn't help because I had a recurrence of aHUS. So, I've been on dialysis ever since.

There are many challenges that aHUS patients and their families face when they

learn about the disease. One big problem is the lack of reliable information that is easy to understand. Many people don't even know that aHUS is a very complicated disease that can cause kidney failure and even lead to death. We try to learn more about it but often don't realize how bad things can get if we don't take it seriously.

The fact that this is an ultra-rare disease, where the average nephrologist has seen none or maybe one aHUS patient in their entire career, does not help. Specialists with experience in treating this disease are difficult to find. Because of this, many people end up either dying or needing dialysis simply because they didn't have the right information or didn't see the right doctors.

Genetic testing is very important for aHUS patients. Sadly, many patients and their families don't know what genetic testing really means, where to get it done, how much it costs, or which tests to ask for. If we are lucky enough to find a doctor who knows about aHUS, we might get the right advice. But often, this help is hard to find, leaving us confused about how to get genetic testing done. As a result, many patients end up spending a lot of money without getting the right answers.

It is also very frustrating to learn about the lack of treatment options. Typically, after meeting with a doctor and understanding

more about the disease, patients realize there are no treatments available in India. We feel helpless when we find out that importing the needed drugs is possible but extremely expensive. Only the very wealthy can afford it, which adds to our frustration.

A few lucky patients go into remission, but they and their families spend years worrying about a possible relapse. So, most people either die from complications of the disease or end up on dialysis because the complement inhibitor isn't available in India.

Those who end up on dialysis have to deal with all the problems that dialysis brings. While other dialysis patients often have a good chance of a successful kidney transplant, people with aHUS cannot opt for one, as the chances of recurrence are often very high. This means that patients need to spend years on dialysis, which can be very onerous. Mortality among dialysis patients is fairly high, and those who are able to live long have to contend with various side effects such as cardiac issues, bone problems, and sexual dysfunction. There is hardly an organ in the body that is not impacted by kidney failure.

While good-quality dialysis is becoming more accessible – and this helps improve the quality of life of patients – it is still difficult for many patients to find a good-quality dialysis clinic close enough to their homes.

Government support has been initiated in recent years for rare diseases like aHUS through the National Policy for Rare Diseases (NPRD). However, the grant offered is ₹50 lakhs, which is barely enough for a few months of treatment. Also, the entire process of availing of this grant is fairly complex, with the treating doctor having to register their

patients with the authorities. The processing of this grant is also possible only through certain Centres of Excellence, which makes it difficult for patients in other locations to register and avail themselves of the grant.

All this leaves aHUS patients ruing the combination of poor genetics and poor geography we have to grapple with. It can be very disheartening to know that there is a drug available in other parts of the world that can give us a much better quality of life, that can make us avoid dialysis altogether, and help us lead a normal life. However, just by virtue of being born in a country that cannot afford to give its citizens good-quality healthcare, we are condemned to lead compromised, shortened lives.

Through online groups that include patients from countries where the drugs are available, we often see patients advocating for easier access to treatments, reducing hospital visits for infusions, and even subcutaneous or oral complement inhibitors, which prevent them from getting into kidney failure in the first place or, in the worst case, allow a successful kidney transplant. Meanwhile, we struggle with just getting the drug itself. The priorities are completely different.

Thankfully, things are starting to change. Recently, clinical trials for some complement inhibitors were conducted in India, and some Indian aHUS patients participated. However, there's still uncertainty about what happens after these trials in terms of access to these drugs.

Another piece of good news is that Eculizumab, currently the only approved drug for this disease worldwide, may soon become available in India. But the pricing is still unknown, and it's a big concern among

patients and advocates as to how costly the drug will be and whether many patients will be able to afford it even if it becomes available.

aHUS patients in India are currently cautiously optimistic. After many, many years of despondency, there is finally some hope that things are beginning to change. India is

beginning to be looked at by companies that are working on complement inhibitors. It is critical, though, that these companies recognize the completely different context that exists in this country compared to the developed world. For a better future, the government and pharmaceutical companies must come together and collaborate with a genuine intent to change things.



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Plasmodium Vivax Malaria

CAUSING THROMBOTIC MICROANGIOPATHY

Dr Kandarp Patel, Dr Neha Athale, Dr Avinash Chaudhari, Dr Amar Kulkarni, Dr Sachin Naik

Introduction

Malaria is caused by the protozoan Plasmodium, transmitted by the Anopheles mosquito. Five species - falciparum, vivax, malariae, ovale, and knowlesi - infect humans. Globally, malaria affects over 200 million people annually. Acute kidney injury (AKI) is most frequent with P. falciparum infection, but P. vivax has also been increasingly linked to renal complications. Malaria contributes to 2–39% of hospital admissions for AKI, and up to 60% of patients with severe parasitemia may develop AKI. Thrombotic microangiopathy (TMA), a disorder of endothelial injury leading to microvascular occlusion, often presents with AKI due to glomerular susceptibility.

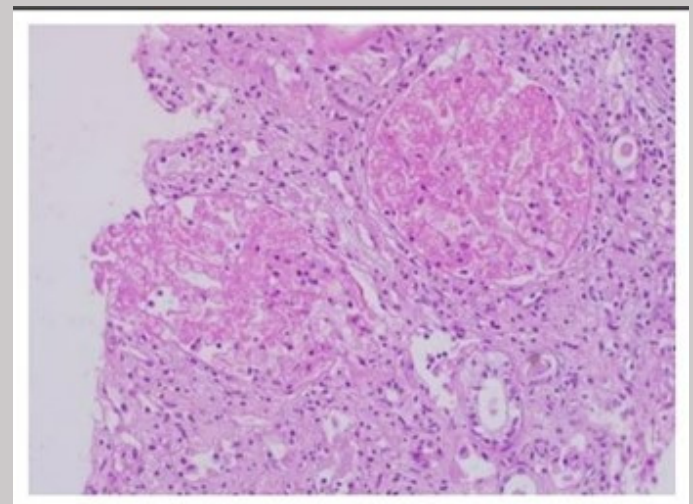
Case Report

A 28-year-old woman presented with fever, chills, nausea, and decreased urine output for three days, progressing to anuria. She had no significant past history. On admission, vitals were stable. Examination revealed pallor, icterus, pedal edema, and mild abdominal

tenderness. Laboratory reports showed anemia, thrombocytopenia, and P vivax positivity on peripheral smear.

She was treated with intravenous artesunate, antibiotics, and initiated on hemodialysis. Workup for vasculitis, connective tissue diseases, and other infections was negative. With regular dialysis, her clinical status improved, and urine output returned. A renal biopsy performed two weeks later demonstrated diffuse cortical necrosis, secondary glomerular sclerosis, ischemic changes, and features of TMA with focal arterial thrombotic occlusion. She was discharged but lost to follow-up.

Investigations





RENAL BIOPSY: Biopsy showed evidence of diffuse cortical necrosis. Viable areas exhibited secondary glomerular sclerosis,

accompanied by ischemic glomerular changes and thrombotic microangiopathy. Focal arterial luminal thrombotic occlusion.

	ON ADMISSION	AFTER 1 WEEK	AFTER 2 WEEKS
HEMOGLOBIN (g/dl)	9.2	6.9	11
TOTAL WBC COUNT (UL)	10	12	10
PLATELETS (UL)	30	203	248
CREATININE (mg/dl)	5.67	5	5.5
TOTAL BILIRUBIN (mg/dl)	4.1	1	1
DIRECT BILIRUBIN (mg/dl)	3.1	0.6	0.6
P VIVAX	POSITIVE		

Other results: urine demonstrated haematuria and proteinuria (2+blood, protein trace, RBCs 6-7); LDH was 994; serum haptoglobin <10; Coombs test negative; complement levels normal; ANCA and anti-dsDNA negative. Peripheral smear showed no schistocytes.

Discussion and Conclusion

Complicated malaria can mimic TMA, as both present with hemolytic anemia, thrombocytopenia, jaundice, and renal failure. Though P. vivax has long been considered a relatively benign parasite, recent reports from endemic regions describe severe complications, with AKI incidence ranging from 10 - 19%.

The pathophysiology of P vivax-induced TMA is multifactorial:

- Infected RBCs adhere to endothelium, causing hypoxia.
- Cytokine production is exaggerated compared with P falciparum.
- Toxin-like effects may damage endothelial integrity.

Case of Plasmodium Vivax Malaria
Causing Thrombotic Microangiopathy

- Increased endothelial activation and procoagulant activity trigger inflammation, coagulation, and vascular injury.

This case illustrates that P vivax malaria can lead to life-threatening renal complications such as TMA and cortical necrosis. Clinicians

should maintain high suspicion for TMA in patients with malaria-associated AKI, particularly when anaemia and thrombocytopenia persist despite parasitological clearance. Early renal biopsy is essential for diagnosis and guiding interventions, which may improve outcomes.

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Tea and Toast Syndrome

WITH ACUTE HYPONATREMIA IN AN ELDERLY MALE

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DrNB 1st year, Department of
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Abstract

This case report summarizes the clinical presentation, diagnosis, and management of an elderly male patient exhibiting signs of "Tea and Toast Syndrome" characterized by hyponatremia. The case illustrates the interplay between dietary habits and electrolyte imbalances in geriatric patients.

Introduction

Tea and Toast Syndrome is a diet-related phenomenon seen predominantly in the elderly, characterized by a limited intake of nutrients often consisting of only tea and toast. This dietary pattern may lead to various nutritional deficiencies and health complications, including electrolyte imbalances such as hyponatremia. Hyponatremia, defined as a serum sodium concentration < 135 mEq/L, can result in significant morbidity, particularly in the elderly.

Case Presentation

An 71-year-old male with a history of hypertension and mild cognitive impairment presented to the emergency department with complaints of confusion, weakness, and recent falls. His family reported that he primarily consumed tea and toast daily, with little intake of fruits, vegetables, or proteins. Upon examination, he was hypotensive and disoriented.

Laboratory Findings

Initial laboratory tests revealed:

- SERUM SODIUM: 112 MEQ/L (NORMAL: 135-145 MEQ/L)
- SERUM OSMOLALITY: 234 MOSM/KG (NORMAL: 285-295 MOSM/KG)
- BUN = 13
- RANDOM BLOOD SUGAR = 98mg/dl
- LIPID PROFILE = WNL
- SPOT URINE SODIUM = 25
- URINE OSMOLALITY = 77 mOsm/k (Normal = 300-900 gH2O)
- COMPLETE BLOOD COUNT: NORMAL
- COMPREHENSIVE METABOLIC PANEL: Unremarkable except for low sodium levels

Discussion

The patient's dietary history, in conjunction with his lab results, suggested Tea and Toast

*Severe Recurrent Hyponatremia Secondary
to Tea and Toast Syndrome in an Elderly Male*

Syndrome leading to his recurrent hyponatremia. The limited dietary intake resulted in insufficient sodium and other essential nutrients. Additionally, excessive tea consumption can exacerbate hyponatremia due to its high content of caffeine and potential diuretic effect.

It is similar to beer potomania and is associated with high carbohydrate, low protein and low solute diet leading to a dilute urine with suppressed ADH.

Management

The patient was treated with sodium chloride infusions and prescribed a tailored dietary plan emphasizing balanced nutrition including fruits, vegetables, and protein

sources. His cognitive status was reassessed post-rehydration, revealing significant improvement.

Conclusion

This case highlights the importance of dietary assessment in elderly patients presenting with hyponatremia. Tea and Toast Syndrome exemplifies how inadequate nutrition can lead to electrolyte disturbances, necessitating multifaceted management strategies that include both medical treatment and dietary modification.

Keywords

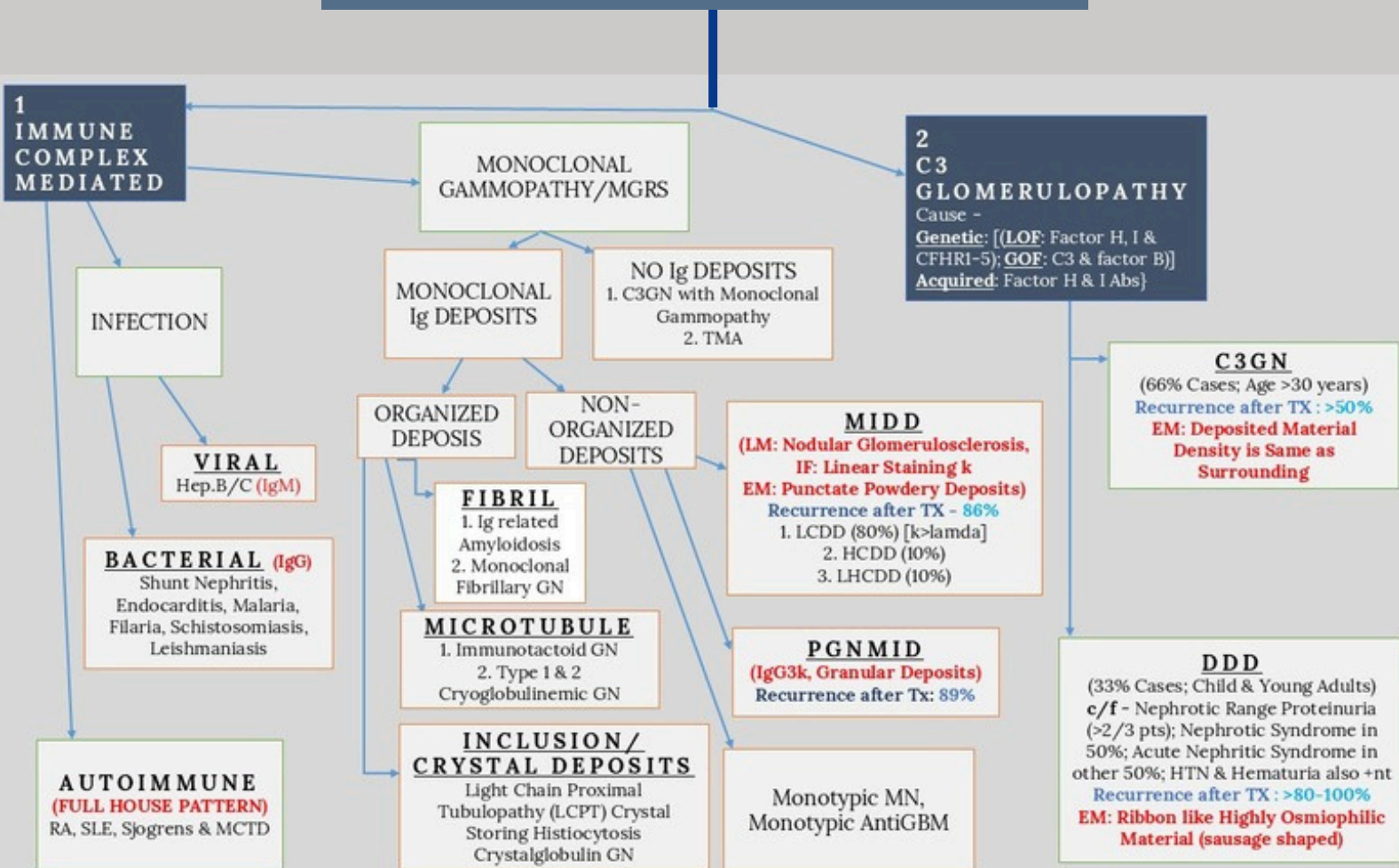
Tea and Toast Syndrome, Hyponatremia, Elderly, Case Report, Nutritional Deficiency.

MPGN Lesion

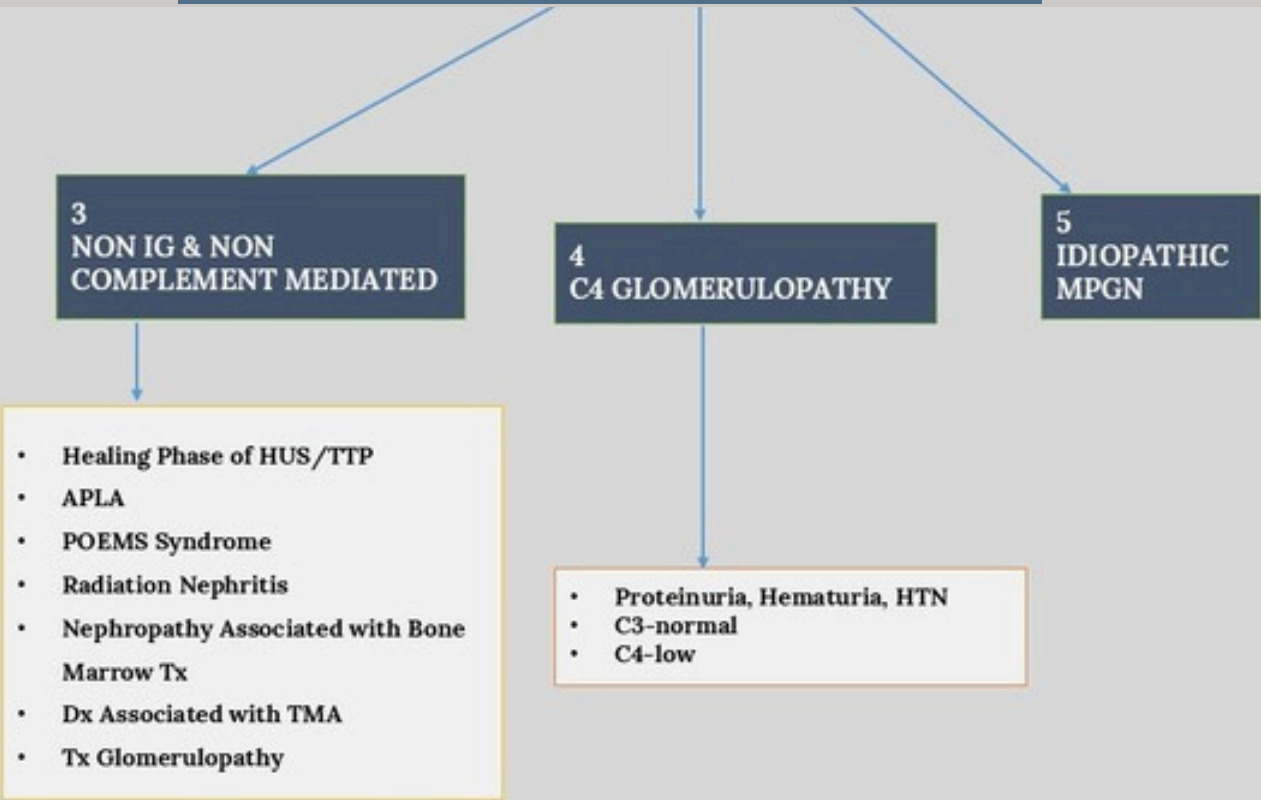
HOW TO APPROACH TO THIS?

Dr Tanmay Vagh
MD, DM Nephrology
BIMS Hospital, Bhavnagar, Gujarat

HOW TO APPROACH TO MPGN LESION?

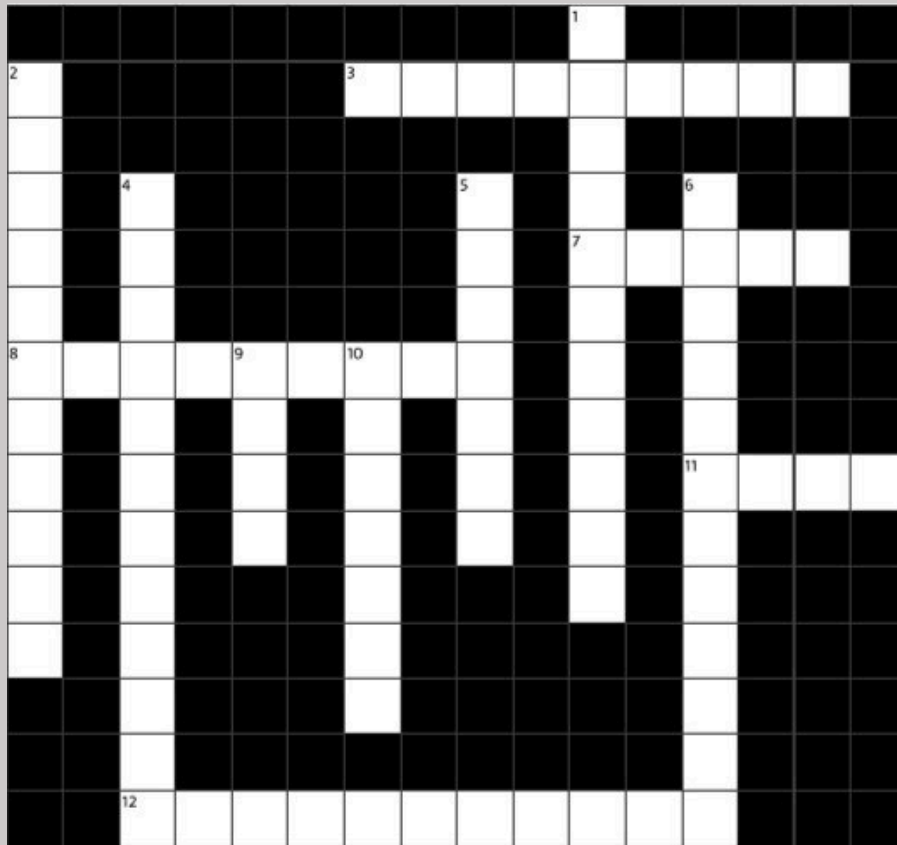


HOW TO APPROACH TO MPGN LESION?



TMA Teaser

Dr Pallavi, Dr Subashri, Dr Sandhya, Dr Ambily



**Solve the
crossword
ONLINE**



KK Crossword
Answers @ Pg 30

ACROSS

- 3 This name has been coined to distinguish fragile immature RBCs in newborns from abnormal schistocytes
- 7 Lactate Dehydrogenase exhibits five isomeric forms assembled in tetramers of either of the two types of subunits, namely muscle (M) and _____
- 8 APPELHUS is a pivotal phase 3 trial of this drug in atypical HUS
- 11 This test can be used to monitor complement activity during Eculizumab therapy
- 12 A human immunoglobulin G4 monoclonal antibody that is studied in the treatment of transplant-associated TMA

DOWN

- 1 He described the first case of TTP in 1924
- 2 Long acting congener of Eculizumab
- 4 While using complement inhibitor therapy, this drug should be used for prophylaxis to prevent infection
- 5 This antimalarial is known to be associated with TMA
- 6 This drug which is FDA approved for acquired TTP can also be used in SLE associated with TMA
- 9 Patients with this genetic mutation causing TMA may improve after kidney transplantation with low recurrence risk; eculizumab discontinuation can be considered early
- 10 A scoring system used to diagnose patients with severe ADAMTS13 deficiency

ACROSS

3 Pyknocyte - The name "pyknocyte" has been coined to distinguish fragile immature RBCs in newborns from abnormal schistocytes. Pyknocytes of up to 2% of the RBCs have been observed in full-term neonates and in up to 6% in premature babies until the first six months of life.

7 Heart (H) - Lactate dehydrogenase is a cytoplasmic enzyme that is present in almost all tissues but at high concentrations in muscle, liver, and kidney. Red blood cells also contain moderate concentrations of this enzyme. LDH exhibits five isomeric forms assembled in tetramers of either of the two types of subunits, namely muscle (M) and heart (H). These five isoforms, although catalyzing the same overall reaction, differ in their affinity to the substrate, inhibition concentration, isoelectric point, and electrophoretic mobility. These five isoforms can be visualized in the active state using LDH zymography.

8 Iptacopan - is an oral, first-in-class, highly potent, proximal AP inhibitor that specifically binds factor B (FB).

11 CH50 - assesses the ability of serum to lyse sheep erythrocytes optimally sensitized with rabbit immunoglobulin M (IgM) antibody and detects a deficiency of the classical pathway. CH50<20% indicates optimal complement blockade at 7 days of treatment initiation.

12 Narsoplimab - Narsoplimab inhibits MASP-2, the effector enzyme of the lectin pathway of the complement system. In this retrospective study, which evaluated the safety and efficacy of narsoplimab administered under a compassionate use program in a cohort of pediatric and adult patients with high-risk TA-TMA, showed high response rates and the encouraging survival outcomes underscore the potential of narsoplimab as a valuable therapeutic option, particularly for high-risk cases.

1 Moschcowitz - Dr Eli Moschcowitz described a 16-year-old female who became acutely ill and died after a short hospital stay, and the postmortem examination demonstrated widespread microvascular thrombi involving the heart, kidneys, and the spleen.

2 Ravulizumab - long acting congener of eculizumab, a C5 inhibitor, which prevents formation of membrane attack complex. It requires dosing every 8 weeks instead of every 2 weeks for eculizumab. It is approved for use in PNH and aHUS.

4 Erythromycin - Patients should get prophylaxis with ciprofloxacin or ceftriaxone till vaccination is not complete and for first 2 weeks after complete vaccination. Once meningococcal vaccination is complete, patients should be shifted to penicillin V, erythromycin or azithromycin which should be continued for at least 2 months after last dose of eculizumab.

5 Quinine - Quinine was one of the most common causes of immune mediated DITMA (Drug induced TMA) until 2010.

6 Caplacizumab - single-variable domain immunoglobulin that recognizes the human vWF A1 domain and inhibits the vWF-platelet GPIb - α interaction. Initially developed for ACS but was discontinued and now FDA approved for acquired TTP.

9 CD46 - CD46 coding for membrane co-factor protein (MCP) is a transmembrane protein abundantly expressed in the kidney. Post kidney transplantation, patients often improve with low risk of recurrence of TMA. Eculizumab, however, may still be required in case of significant defects in CD46 gene to prevent recurrence post transplantation.

10 PLASMIC - The PLASMIC score for TTP prediction comprises components such as platelet count, hemolysis, history of solid organ cancer or stem cell transplant, MCV less than 90, INR less than 1.5, and creatinine less than 2.0. A systematic review and meta-analysis from 2020 stated that a PLASMIC score of 5 or higher provided a sensitivity of 99% and specificity of 57%.