ISSUE 6 I VOL 1 I APRIL TO JUNE 2025

Highlights

Upcoming WINICON 2025

WOMEN IN NEPHROLOGY INDIA

WINGS

WOMEN IN NEPHROLOGY GUP SHUP

OFFICIAL NEWSLETTER OF WOMEN IN NEPHROLOGY INDIA


WIN
WOMEN IN NEPHROLOGY
INDIA

CONTENTS

EDITORIAL

It gives us immense pleasure to present the second issue of this year's Women in Nephrology India (WIN) newsletter. As we continue our journey to create a platform for learning, sharing, and inspiring each other, this issue once again celebrates the growing contributions of women to the field of nephrology in India and beyond.

In this issue, we feature an intriguing case report on the diagnostic work-up for tubular balance disorders – a reminder of how meticulous clinical acumen and laboratory insights can guide us through complex renal physiology.

We also take this opportunity to commend the enthusiastic contributions of our young women nephrologists in research and scientific communication. Their active involvement in clinical studies, publications, and the creation of visual abstracts has added a fresh and creative dimension to how knowledge is disseminated. Such innovations not only enhance learning but also make nephrology more accessible and engaging to the wider medical community.

Another highlight of this quarter is the upcoming WINICON 2025 Conference in Chennai, which promises to be a vibrant platform to exchange knowledge, showcase research, and foster collaborations. We invite all members and colleagues to participate actively and make this meeting a milestone event for nephrology professionals across the country.

As we move forward, let us continue to champion each other's achievements, encourage young nephrologists, and reaffirm our collective commitment to excellence in kidney health. We hope you enjoy this issue and look forward to your continued engagement with WIN.

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Teaching Point

Broken Balance: Growth Failure and Acid–Base Turmoil in a Young Child of Cousins

Dr Shweta Phadke, Dr Payal Gaggar, Dr Sree Bhushan Raju
Nizams Institute of Medical Sceinces,
Hyderabad, Telangana, India.

Introduction:
Fluid, electrolyte and acid-base homeostasis are imperative for the preservation of life. This is accomplished by adjustments in glomerular filtration and tubular reabsorption ofsolutes and fluids in response to fluctuations in dietary intake and metabolic processes (2). Inherited and acquired defects can hamper these physiologic processes, resulting in varied clinical presentations which require detailed evaluation including genetic work up. Consanguineous marriages lead to increased expression of autosomal recessive disorders in next generation. (3) Here we present a similar case of distal renal tubular acidosis in a patient born of consanguineous marriage.

Case details
A 3 years 9 months child, third born to a second-degree consanguineous marriage, with mother being reliable informant was brought with chief complains of inability to gain adequate height and weight since 1 year of his age. He did not have any history of recurrent infections. Mother denied history of short stature in the family and both parents attained puberty at normal age. He was born at full term with low birth weight(2.4kg) and required NICU admission for 4 days. He was immunised for his age. He had not achieved developmental milestones appropriate for his age with developmental age being 2yrs 8 months and developmental quotient of 75%. His intellectual quotient was 80%below average. He was in calorie deficit of 258 kcal/day and a protein deficit of 1.5 gm/day. On anthropometric examination, height was 84 cm(<-3 z score), weight 10.3 kg (<-3 z score), head circumference -48.5cm (0- -2 z score), upper segment: lower segment ratio of 1.07. His height age (1 yr 6 months) was lesser than weight age (2 years) which was lesser than his bone age (3 years). On head-to-toe examination, he had mild pallor, knocked knees, coxa vera, costochondral beading and dysmorphic facies but no other leg deformities or midline facial defects. Systemic examination also revealed normal findings. No sensorineural hearing loss or history of renal stones. He was further subjected to blood investigations which revealed hypokalaemia, hyperchloremic metabolic acidosis and elevated urinary pH with normal renal function and normal ultrasound abdomen. The reports are enclosed in table 1.

Table 1

Haemoglobin	10.1 gm/dl	Calcium	9.5 mg/dl
Total leucocyte count	13090/ cmm	Phosphorous	3.8 mg/dl
Platelet count	440000/cmm	ALP	445 U/L
Creatinine	0.3 mg/dl	TSP/albumin	7.1/4.6 gm/dl
urea	18 mg/dl	TSH	4.2 microIU/L
Sodium/ potassium	135/2.3 meq/L	PH/pCO2/HCO3	7.3/22/12.1
Vit D	18.5 ng/ml	Urine pH	7.5
PTH	19.5 pg/ml	Spot urine:k+	20 mmol/g
Urine anion gap	17	Urine osmolality	130 mosm/kg

In view of history of consanguinity, growth retardation and biochemical abnormalities suggestive of renal tubular acidosis, particularly distal RTA , whole exome sequencing sent. It revealed SLC4A1 mutation at exon 19, homozygous, autosomal recessive associated with autosomal recessive distal renal tubular acidosis-4 with haemolytic anaemia (OMIM# 61590), a pathogenic variant. The child is being treated with alkali therapy, potassium supplementation and folate supplementation

Discussion

Being born of consanguineous marriage, our patient was at high risk of autosomal recessive inherited disorders. The clinical features favouring tubulopathy were growth retardation, hypokalaemia and metabolic acidosis, though he did not have nephrocalcinosis and sensorineural hearing loss. Evaluation of renal tubular acidosis should include venous/arterial blood gas analysis, serum potassium, calcium, magnesium, phosphorous, uric acid and urinary pH. Further evaluation is tailored as per reports to include urinary electrolytes, osmolality and 24 hours urinary excretion of electrolytes. Ultrasound imaging helps to detect nephrocalcinosis or nephrolithiasis. Child should also be evaluated for rickets, hearing abnormalities and ocular defects. Lastly, genetic testing should be done wherever feasible as it helps in confirmation of diagnosis, prognostication, antenatal counselling for future pregnancies and tailored therapies wherever available. Most of these disorders are currently treated with electrolyte and alkali supplementation, awaiting gene directed therapies.

Hypokalaemia with metabolic acidosis is a typical feature of both proximal tubular bicarbonate wasting and impaired acid secretion in the distal tubule(2).The pathophysiologic basis of renal tubular acidosis has been depicted in figure 1.(4), detailed explanation is beyond the scope of this article. .

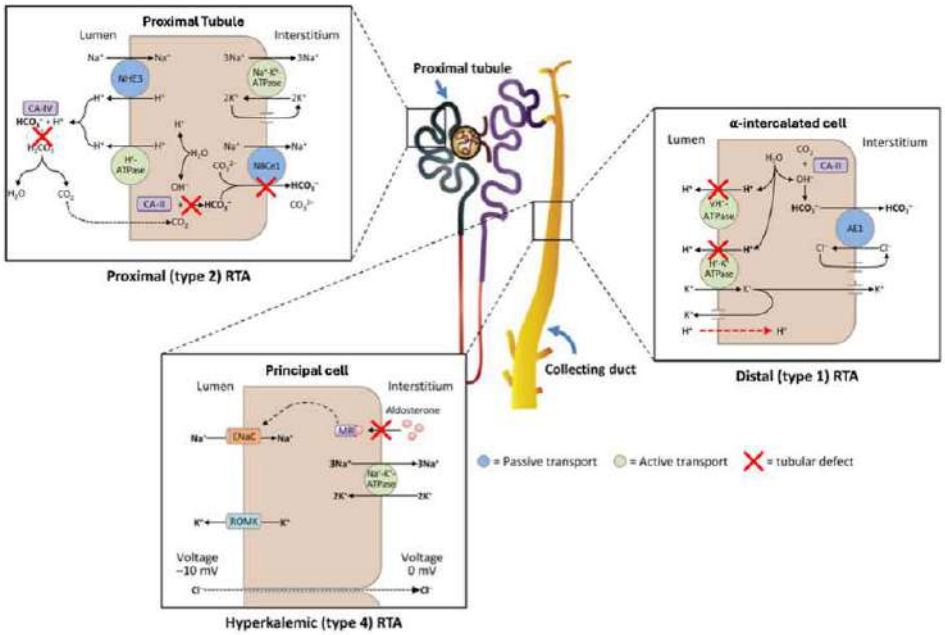

Figure 1: Pathophysiologic basis for proximal, distal, and type 4 RTAs. Abbreviations: AE1, kidney anion exchanger 1; CA-II, carbonic anhydrase II; CAIV, carbonic anhydrase IV; CO2, carbon dioxide; ENaC, epithelial Na+ channel; H+, hydrogen ion; H2CO3, carbonic acid; HCO3–, bicarbonate; H2O, water; MR, mineralocorticoid receptor; NBCe1, electrogenic sodium bicarbonate cotransporter 1;NHE3, sodium–hydrogen antiporter 3; H+-ATPase, hydrogen-exporting ATPase; ROMK, apical membrane K+ channel; RTA, renal tubular acidosis. Adapted from Palmer BF, Kelepouris E, Clegg DJ. Renal tubular acidosis and management strategies: a narrative review. Adv Ther. 2020;38(2):949-968. doi:10.1007/s12325-020-01587-5.

Hypokalaemia with metabolic acidosis is a typical feature of both proximal tubular bicarbonate wasting and impaired acid secretion in the distal tubule(2).The pathophysiologic basis of renal tubular acidosis has been depicted in figure 1.(4), detailed explanation is beyond the scope of this article. .

An approach to hypokalaemia with metabolic acidosis is enlisted in figure 2.

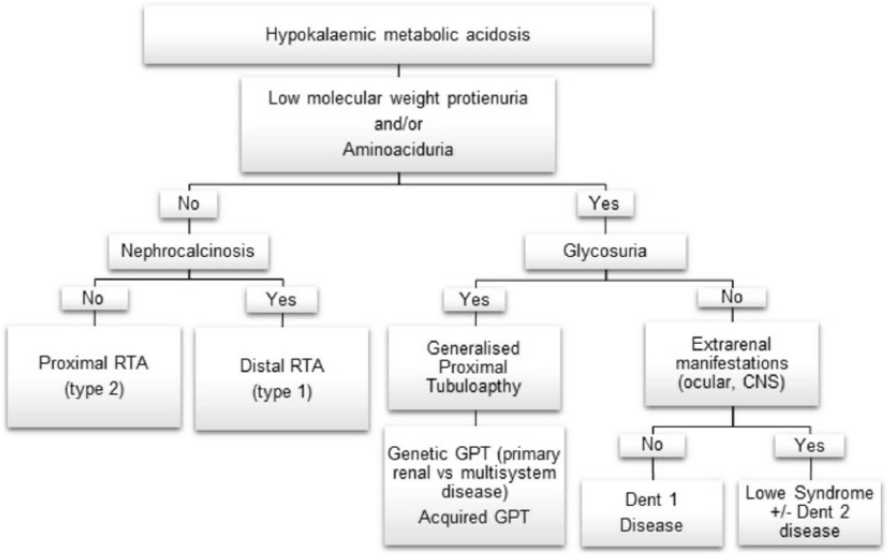

Figure 2- Diagnostic flow chart for hypokalaemia metabolic acidosis

The next step in our patient was genetic evaluation which yielded pathogenic variant of SLC4A1. Anion exchanger 1 (AE1), encoded by SLC4A1, facilitates Cl^-/HCO_3^- exchange. The erythroid isoform (eAE1) maintains red blood cell structure and enables CO_2 transport, while the kidney-specific isoform (kAE1), lacking 65 N-terminal residues, resides in α-intercalated cell basolateral membranes, aiding urinary acidification. (1) The pathophysiology of dominant or recessive SLC4A1 variant related dRTA has been linked with the mis trafficking defect of mutant kAE1 protein. However, in vivo studies in kAE1 R607H dRTA mice and humans have revealed a complex pathophysiology implicating a loss of kAE1-expressing intercalated cells and intracellular relocation of the H+-ATPase in the remaining type-A intercalated cells. These cells also displayed accumulation of ubiquitin and p62 autophagy markers.(5)The physiologic mechanism of urinary acidification is depicted in figure 3.

A case of an adult female with recurrent haemolytic anaemia and distal RTA with SLC4A1 mutation was reported in IJN.(6) Most of the cases are diagnosed during childhood with clinica features of failure to thrive.The autosomal recessive form is considered severe one and is commonly encountered in Asian population. Early diagnosis and prompt treatment are essential.

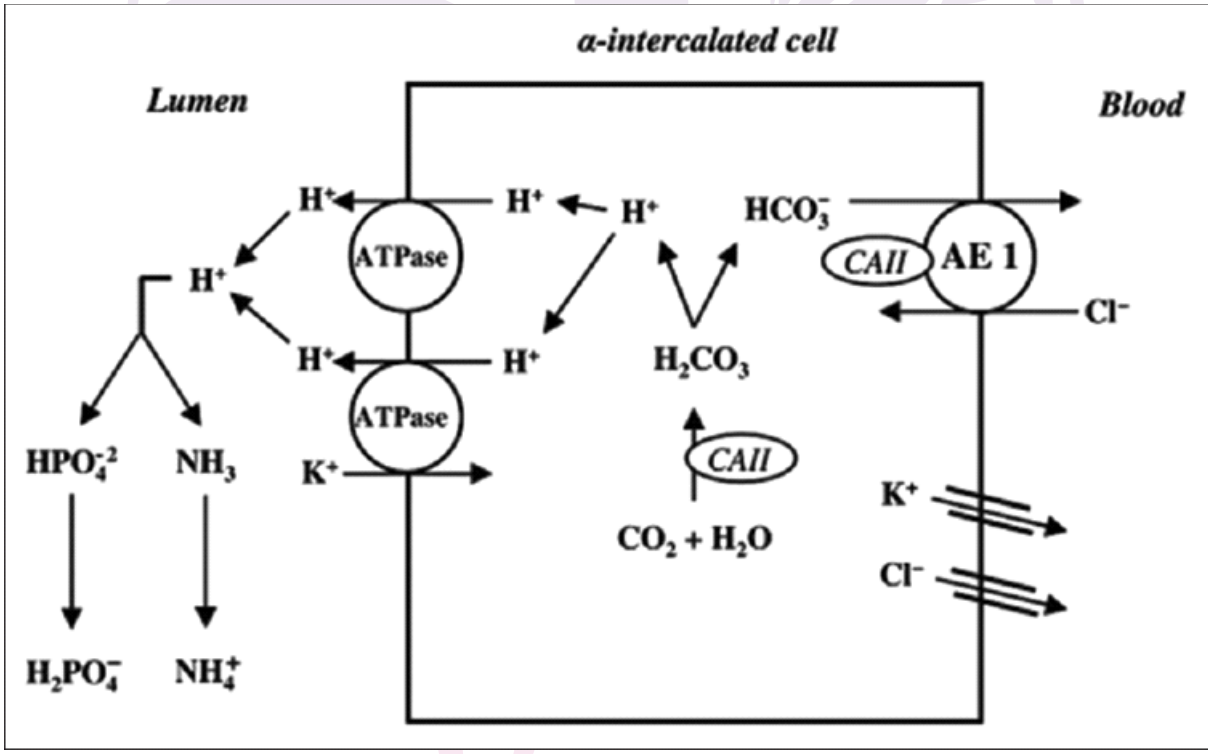

Figure 3- Schematic model of the α-intercalated cell and the H+ secretion in cortical collecting tubule

Conclusion

Tubulopathies highlight the importance of kidneys in maintaining homeostasis. A single gene defect gives rise to wide array of clinical manifestations which are non- specific to a single disease entity. Thus, amalgamation of clinical, biochemical, radiologic and genetic testing is required for timely diagnosis and prompt treatment.

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Events

WIN - Maharashtra Group Presents
Case Based Series Hypertension & Kidneys
Date: 22nd April 2025 | Time: 05:00 PM - 07:00 PM IST

Sr. No.	Topic (30 min)	Presenter (5 min)	Speaker (15 min) Q&A (10min)	Chairperson
CASE 1	Importance of ABPM	Dr Aditi Thawait	Dr Jyotsna Zope	Dr Kavita Vishwakarma, Dr Swati Mane, Dr Jasmine Sethi
CASE 2	Resistant Hypertension	Dr Shweta Singh	Dr Monali Sahu	Dr Divya Bajpai, Dr Ruchi Samdhani, Dr Aruna Acharya
CASE 3	Hypertension in a Dialysis patient	Dr Vaishnavi Gaur	Dr Rasika Sirsat	Dr Vidya Kadam, Dr Purva Bavikar, Dr Isha Tiwari
CASE 4	Hypertension in a renal transplant patient	Dr Aakarsha Sahay	Dr Kalpana Mehta	Dr Pooja Binani, Dr Aswathy Haridas, Dr Mythri Shankar

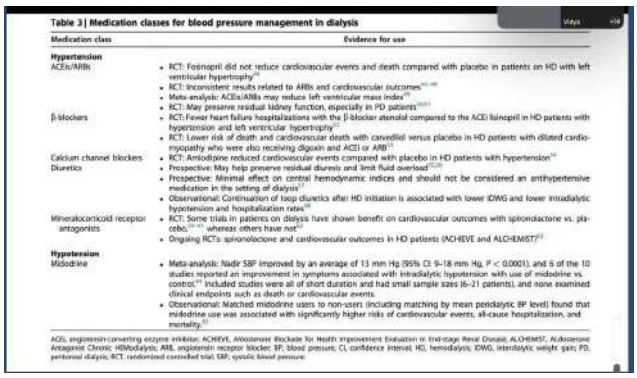
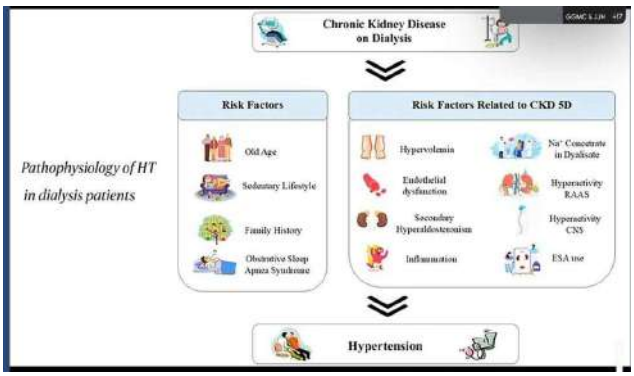
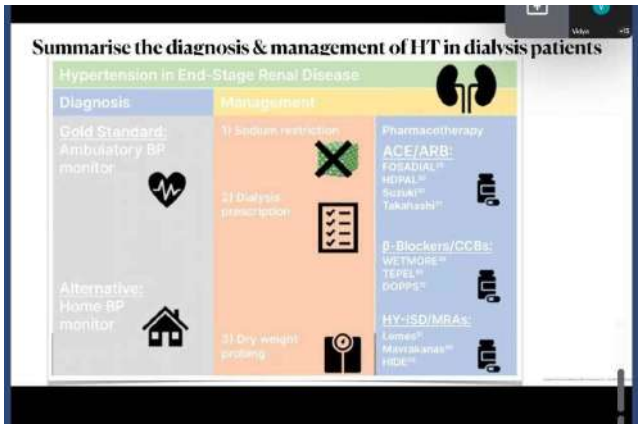


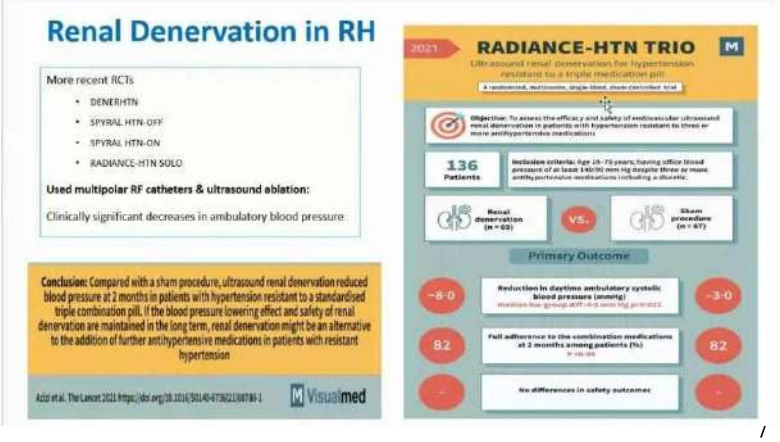
Table 3 | Medication classes for blood pressure management in dialysis

Medication class	Evidence for use
Hypertension	
ACEIs/ARBs	• RCT: Fosinopril did not reduce cardiovascular events and death compared with placebo in patients on HD with left ventricular hypertrophy[24] • RCT: Inconsistent results related to ARBs and cardiovascular outcomes[25–28] • Meta-analysis: ACEIs/ARBs may reduce left ventricular mass index[29] • RCT: May preserve residual kidney function, especially in PD patients[30,31]
β-blockers	• RCT: Fewer heart failure hospitalizations with the β-blocker atenolol compared to the ACEI lisinopril in HD patients with hypertension and left ventricular hypertrophy[32] • RCT: Lower risk of death and cardiovascular death with carvedilol versus placebo in HD patients with dilated cardiomyopathy who were also receiving digoxin and ACEI or ARB[33]
Calcium channel blockers	• RCT: Amlodipine reduced cardiovascular events compared with placebo in HD patients with hypertension[34]
Diuretics	• Prospective: May help preserve residual diuresis and limit fluid overload[35,36] • Prospective: Minimal effect on central hemodynamic indices and should not be considered an antihypertensive medication in the setting of dialysis[37] • Observational: Continuation of loop diuretics after HD initiation is associated with lower IDWG and lower intradialytic hypotension and hospitalization rates[38]
Mineralocorticoid receptor antagonists	• RCT: Some trials in patients on dialysis have shown benefit on cardiovascular outcomes with spironolactone vs. placebo,[39–41] whereas others have not[42] • Ongoing RCTs: spironolactone and cardiovascular outcomes in HD patients (ACHIEVE and ALCHEMIST)[43]
Hypotension	
Midodrine	• Meta-analysis: Nadir SBP improved by an average of 13 mm Hg (95% CI 9–18 mm Hg, $P < 0.0001$), and 6 of the 10 studies reported an improvement in symptoms associated with intradialytic hypotension with use of midodrine vs. control.[44] Included studies were all of short duration and had small sample sizes (6–21 patients), and none examined clinical endpoints such as death or cardiovascular events. • Observational: Matched midodrine users to non-users (including matching by mean periclalytic BP level) found that midodrine use was associated with significantly higher risks of cardiovascular events, all-cause hospitalization, and mortality.[45]

ACEI, angiotensin-converting enzyme inhibitor; ACHIEVE, Aldosterone Blockade for Health Improvement Evaluation in End-stage Renal Disease; ALCHEMIST, Aldosterone Antagonist Chronic HEModialysis; ARB, angiotensin receptor blocker; BP, blood pressure; CI, confidence interval; HD, hemodialysis; IDWG, interdialytic weight gain; PD, peritoneal dialysis; RCT, randomized controlled trial; SBP, systolic blood pressure.



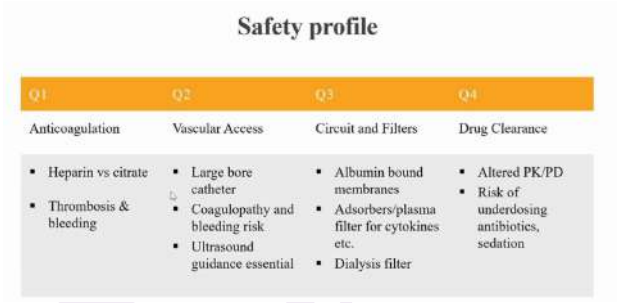


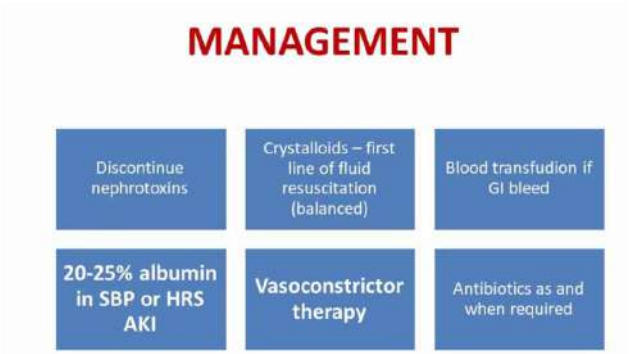


Women's Day Event by Women in Nephrology - Karnataka

Date: 25th April 2025 | Time: 06:00 PM IST Onwards

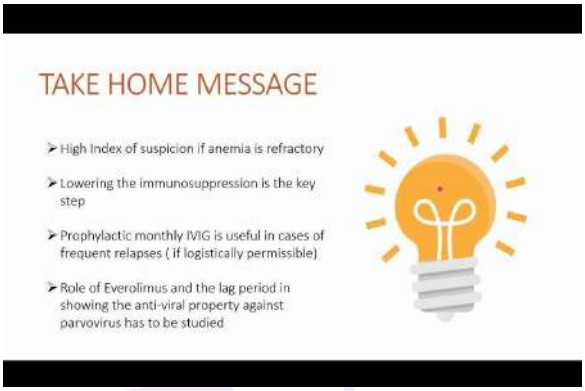
WIN – North Group
Exploring the kidney, Heart and Liver Crosstalk
Date: 22nd May 2025 | Time: 05:00 PM – 06:30 PM IST Onwards

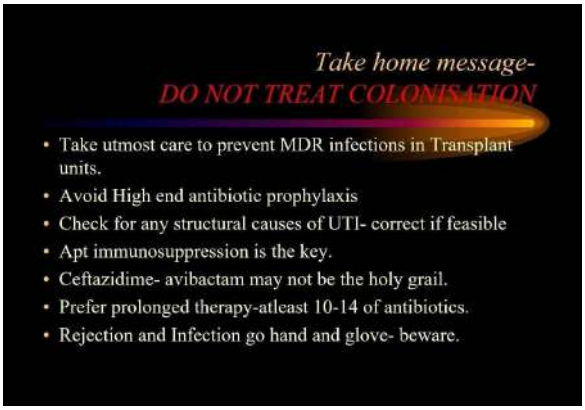


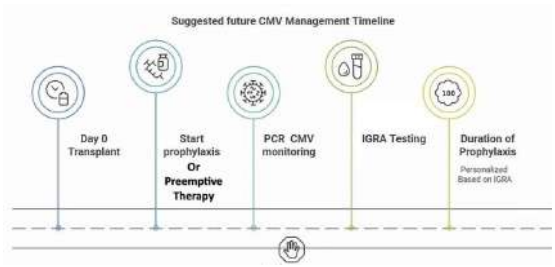


Events

TN and Puducherry Wing - Webinar
Managing Infections In Kidney Transplant Recipients
Date: 26th June 2025 | Time: 07:00 PM - 08:30 PM IST







Upcoming Events


ISN
INTERNATIONAL SOCIETY OF NEPHROLOGY
WIN
WOMEN IN NEPHROLOGY INDIA
WOMEN IN NEPHROLOGY-INDIA
WIN-ICON 2025
4th Annual Conference of Women in Nephrology India
WIN-ICON 2025
Chennai,Tamilnadu
The Residency Towers , T Nagar Chennai
WIN-ICON2025 Highlights
Pre conference workshop Renal pathology
Hybrid session international speakers
Allied health sciences workshop
Panel discussion Cardio Kidney Metabolism
Newer treatment strategiesin renal transplant
Lunch Symposia -Important Drug Molecules
Debates , Quiz
Oral & Poster paper sessions
Serene ambience, state of the art cuisine
'WIN- Gala Night'- a feast to all eyes.
Register Now
www.winicon2025.com

ISN
INTERNATIONAL SOCIETY OF NEPHROLOGY

4th Annual Conference of Women in Nephrology India

WIN-ICON 2025

The Residency Towers , T Nagar Chennai

Welcome to WIN ICON 2025

Warm Greetings from Chennai

It is with immense joy that we welcome you to the 4th Annual Conference of WIN-INDIA—WIN -ICON 2025, hosted in the vibrant city of Chennai on August 30th and 31st, 2025.

As the Tamil Nadu and Puducherry wing of Women in Nephrology-India, we are proud to uphold the legacy of this advancing scientific community. WIN-ICON 2025 promises engaging academic sessions, inspiring discussions, and an opportunity to connect with peers from across the nation.

Come be part of this celebration of science and sisterhood in a city known for its rich cultural heritage and lip-smacking cuisine. We look forward to hosting you with warmth, learning, and unforgettable experiences!

Dr Manisha Sahay
President WIN INDIA

Dr Swarnalatha Guditi
Secretary WIN INDIA

Dr S Jayalaskmi
Organising Chairman

Dr Ranjane Muthu
Organising Secretary

Register Now

TO REGISTER AND MORE DETAILS VISIT

Website : www.winicon2025.com

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Cultural Committee


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Dr Mookambika

Dr.A.Aarthi (JIPMER)

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ISN's endorsement is for the promotion of education in general, therefore the specific content of the event/course is the responsibility of the organizer.

4th Annual Conference of Women in Nephrology India

WIN-ICON 2025

Chennai, Tamilnadu

The Residency Towers, T Nagar Chennai

Scientific Programme

Hall A - Day 1 Program Schedule (IMPERIAL Hall)

30th August Saturday

Time	Program	Speaker	Chairpersons
8am - 8.30 am	**REGISTRATION**		
8.30 am - 10am	Pre conference workshop on Onco Nephropathology	Dr Anila Abraham	Dr Geetika Singh Dr Vinita Agarwal
10am-10.30am	aHUS - Treatment update	Dr Anuja Java (Online) USA	Dr Kalaivani Ganesan Dr K Venkataramanan Dr Jasmine Sethi Dr Chandani Bhagat
10.30 am-10.45 am	**Tea Break**		
10.45 am-11.45 am	**Inaugural Ceremony**		
	Dr Muthu Jayaraman Oration		
11.45am-12.15 pm	Topic : "Clinical wisdom from the bedside : what my critically ill patients taught me"	Dr Vimala A	Dr Abirami K WIN TN President Dr Indumathi E WIN TN Scientific Chair
12.15pm-12.45 pm	Monogenic Hypertension in Children	Dr Bobbity Deepthi	Dr Indira Agarwal Dr Kinnarivala Dr Prabha Sengottuvan Dr Prahlad Nageshwaran
12.45pm-1.30pm	Industry symposium (Finerenone) a. Comparing steroidal and non-steroidal MRA b. Evidence of Finerenone in DKD c. Practical aspects of adding on Finerenone therapy (ADR, monitoring and costing) d. Finerenone in non-diabetic kidney disease (mechanisms and on- going trials)	Moderator: Dr Ranjanee Muthu Panelists: Dr Rubina Vohra Dr Sugan Gandhi Dr Kiranmai Ismail Dr Ishani Haldar	Dr Payal Gaggar Dr V Chandrasekhar Dr Sayali Thakare Dr Uttara Das
1.30 pm - 2 pm	**Buffet Lunch - Senator Hall / Hall A annexe (TOWN Hall)**		
	Glomerular Symposium		
2.00pm-2.30pm	Unraveling Podocytopathies: New Mechanisms and advances in management	Dr Sukanya Govindan	Dr Deepthi Ayanavelli Dr Miranda Pegu Dr Geeta S Sheth Dr Ramprabhahar
2.30pm-3.00 pm	C3GN Redefined: The Rise of Complement-Targeted Therapies	Dr Suceena Alexander	Dr Manisha Sahay Dr Arpita Ray Choudhary Dr Indumathi E Dr N Gopalakrishnan

Time	Program	Speaker	Chairpersons
Dialysis Forum			
3pm – 3.30pm	Case based approaches to trouble shooting in CRRT	Dr Subashri M	Dr Balasubramaniyam R Dr Siva Parvathi Dr Krithika Mohan Dr Abirami K
3.30 pm -4pm	Oral anticoagulants for Atrial Fibrillation in dialysis patients - scrutinizing the evidence and a guide for clinical practice	Dr Sai Vani	Dr Pallabi Bardoloi Dr N R Venkatraman Dr Anitha R Dr Hitaishi
4 pm - 4.30 pm	Peritoneal Dialysis in tough situations: navigating challenges with confidence	Dr Radha Vijayaraghavan	Dr Amit Gupta Dr Renuka S Dr Urmila Anandh Dr Jayalakshmi S
4.30 pm-4.45 pm	**Tea break**		
4.45 pm -5.15pm	Pregnancy in Transplant Recipients	Dr Aarthi Muthukumar (Online) UK	Dr Divya Bajpai Dr Manjusha Yadla Dr Manjuri Sharma Dr Sampathkumar K
5.15 pm -6.00pm	**Cardio-Kidney Metabolic Panel Discussion**		
5.15 pm-5.30pm	CKM syndrome - Exploring the Mechanisms Behind the Burden	Dr Namrata Rao	Moderator : Dr Geetha M Nair Chairpersons:
5.30 pm-5.45pm	Identifying heart failure in CKD: diagnostic challenges	Dr Anitha A	Dr Jikki Dr Tamilarasi V
5.45pm-6pm	An algorithm-based approach to treatment of CKM syndromes	Dr Kajaree Giri	Dr Manju Thampi Dr Abraham Oomman
6.00pm-6.30 pm	Opportunities for Young Nephrologists in ISN	Dr Charu Malik (Online) Belgium (EU)	
6.30 pm -7pm	**WIN India General Body Meeting**		
7.30 pm -10pm	**"WIN Gala Night"- Culturals and Dinner**		

Hall B - Day 1 Program Schedule (RAJ Hall) 30th August Saturday

Time	Program	Speaker	Chairpersons
12 pm - 1pm	Oral presentation - 5 Best Papers	8+2 minutes	Dr P Srijjaa Dr Beena Unnikrishnan
1pm - 1.30 pm	Poster Viewing Session Judges: Dr Ezhilarasi A Dr Anupama YJ Dr Anitha Jagannath K Dr Susan Uthup		
1.30pm -2 pm	**Lunch-Senator Hall / Hall A Annexe (TOWN Hall)**		
6.30pm -7 pm	**TN State General Body Meeting**		

Hall A- Day 2 Program Schedule (IMPERIAL Hall)
31st August Sunday

Time	Program	Speaker	Chairpersons
Acute Kidney Injury Forum			
8am - 8.30 am	Revolutionizing AKI: advanced prediction models and sub- phenotyping of AKI	Dr Priti Meena	Dr M Edwin Fernando Dr Chetna Gothwal Dr Shilpa Shetty Dr Vijaya Sethumadhavan
8.30 am - 9 am	Septic AKI- what's new in the management?	Dr Nisha Jose	Dr T Balasubramanian Dr Gayatri Pegu Dr Swati Mane Dr Shabana Nazneen
9 am - 9.30 am	ADPKD –treatment update	Dr Harshini D Perera Srilanka	Dr Georgi Abraham Dr Garima Aggarwal Dr Isha Tiwari Dr Christine Mary Jane
Transplant Consortium			
9.30 am - 10am	Newer diagnostics in kidney transplant (Identifying antibodies and rejection early)	Dr Shruti Tapiawala	Dr N Keerthiga Dr Jayaprakash Dr Gomathy S Dr Priyamvada P S
10am -10.30 am	Treatment of Antibody-Mediated Rejection - Emerging Strategies and Innovations	Dr Dhanapriya J	Dr Rajeevalochana Parthasarathy Dr Sowrabha Rajanna Dr Vasudevan C Dr Swarnalatha Gudithi
10.30am - 11am	BK Virus Nephropathy Post Transplant: Emerging Insights and Evolving Strategies	Dr Soumita Bagchi (Online) Australia	Dr Subba Rao B Dr Muthu Jayaraman Dr Poornima Tadkal Dr Ratna Prabha
11am - 11.30am	**Tea Break**		
Chronic Kidney Disease Forum			
11.30am-12pm	Role of Genetics in CKD	Dr Roser Torra (Online) Barcelona	Dr Sakthirajan R Dr Sangeetha G Dr Sreelatha M Dr Piyali Sarkar
12pm -12.45 pm	Debate: Precision Medicine in Nephrology: Game-Changer or Overhyped?	PRO: Dr Swati Raju CON: Dr Sakshi Jain	Dr Sanjeev Nair Dr Sneha Joy Dr Mythri Shankar Dr Radha Venkatramanan
12.45 pm-1.30pm	Desidustat Industry Symposium (To block HIF or not ?) a. How does Desidustat work? b. Advantages of Desidustat over conventional EPO c. Gaps in our wisdom on Desidustat A pragmatic guide to usage of Desidustat in day-to-day practice	Moderator : Dr Priya John Panelists: Dr Pragya Pant Dr Nithya D Dr Jyothi R Dr Jaymeena P	Dr Syeda Hurmath Dr Dinesh Dr Anupama Kaul Dr Anita Thangavelu
1.30pm - 2pm	**Lunch – Senator Hall / Hall A Annexe (TOWN Hall)**		
2pm - 3pm	**Final Round Quiz**		
3pm -3.30pm	**Prize Distribution and Valedictory Function**		

Hall B - Day 2 Program Schedule
(RAJ Hall) 31st August Sunday

Time	Program		
8.00 am-9.00 am	**Quiz Round 1** Quiz Masters : Dr Sajmi Shaji, Dr Medhavi Gautam, Dr Sandhya, Dr Myzhivizhiselvi		
Allied Health Sciences Workshop and Hands on Session			
9am - 9.15 am	**Welcome and Introduction**		
Time	**Session**	**Topic**	**Speaker**
9.15 am -10.30	Automated Peritoneal Dialysis	• Settings and Applications • Preventing Peritonitis	Dr Harrini Devi Dr Pooja Prabhu
10.30 am – 11am	**Tea break**		
11 am - 11.30am 11.30 am - 12 pm 12pm - 12.30 pm	Continuous Renal Replacement Therapy	• Understanding the Circuits • Prescription and Precautions • Challenges in CRRT - Sri Lanka Experience	Dr Shamini Ajit Kumar Dr Jyothipriya J Dr Harshini D Perera Sri Lanka
12.30 pm -1 pm 1pm - 1.30 pm	Hemodiafiltration	• Nuances of HDF • Advantages of HDF vs High-Flux HD	Dr A R A Changnidhi Dr Nithyashree N
1.30 pm - 2 pm	**Lunch - RAJ Hall**		
2 pm - 3 pm	**CRRT and HDF-Hands on Session**		
3 pm - 3.30 pm	**Valedictory Function**		

Mistresses of Ceremony : Dr Nithyashree N, Dr Anitha R

Publications

1. **Meena P, Aggarwal G, Anandh U, Yadla M, Sahay M, Chaudhury AR,** et al. Sex inequities in kidney transplantation: a persistent and multifaceted challenge. Am J Kidney Dis. 2025;86(1):7–9.

2. **Meena P, Aggarwal G, Anandh U, Yadla M, Sahay M, Chaudhury AR, Bhargava V, Priyamvada PS, Gopalakrishnan N, Guditi S, Sharma M** et al. Evolving gender dynamics in India's nephrology workforce: a decadal analysis (2013–2023). Nephrology (Carlton). 2025 May;30(5):e70047. doi: 10.1111/nep.70047. PMID: 40312800.

3. **Meena P, Kattah A.** Dialysis during pregnancy: are we any closer to clarity? Kidney Int Rep. 2025 Jul;10(7):2100–2.

4. **Nelson-Piercy C, Srisawat N, Kashani K, Lumlertgul N, Murugan R, Rhee H,** et al. Pregnancy-associated acute kidney injury – consensus report of the 32nd Acute Disease Quality Initiative workgroup. Nat Rev Nephrol. 2025 Jul 18. doi: 10.1038/s41581-025-00979-6. Epub ahead of print. PMID: 40681846.

5. **Kher V, Sharma R, Abraham G, Shah B, Gang S, Gulati S,** et al. Diagnosis, evaluation and management of osteoporosis in chronic kidney disease: navigating treatment approaches – Indian consensus statement. Front Nephrol. 2025 Jul 10;5:1601610. doi: 10.3389/fneph.2025.1601610. PMID: 40708576; PMCID: PMC12286787.

6. **Tapiawala S, Jogale S, Danielle D, Handoo A.** Unfolding the mysteries of cell-based human leukocyte antigen crossmatching in solid organ transplantation. Indian J Transplant. 2025 Apr–Jun;19(2):125–34. doi: 10.4103/ijot.ijot_128_24.

7. **Kamath N, Nesargi S, Bajpai D, Singarayar P, Vidal E, Luyckx VA** Impact of maternal health on neonatal and long-term kidney outcomes. Pediatr Nephrol. 2025 Jun 3. doi: 10.1007/s00467-025-06771-9. Epub ahead of print. PMID: 40459611.

8. **Shankar M** et al. Not all IgA nephropathy are the same: Clinical implications of recurrent versus de novo disease posttransplant. Kidney Int Rep.

9. **Shankar M** Against the odds: Nivedita's fight for survival and hope. J Nephrol. 2025 Jun 14. doi: 10.1007/s40620-025-02300-x. Epub ahead of print. PMID: 40516026.

10. **Shankar M, Lamech TM.** Treatment burden in glomerular diseases: advances and challenges in immunosuppressive therapy. Front Nephrol. 2025 May 1;5:1545373. doi: 10.3389/fneph.2025.1545373. PMID: 40376095; PMCID: PMC12078155.

11. **Gaggar P, Nair RR, Thakur AP, Raju SB.** Post Transplant Membranous Nephropathy: A 10-year Single-Centre Experience. Indian J Nephrol. 2025;35:552-4. doi: 10.25259/IJN_150_2025

12. **Shankar M, Udayashankar A, Rajanna S, Anandh U, Chaudhury AR.** Barriers and Constraints in Scientific Manuscript Preparation Among Nephrologists: Insights From India. Int J Nephrol. 2025 Feb 23;2025:9008616. doi: 10.1155/ijne/9008616. PMID: 40034191; PMCID: PMC11872293

13. **Anandh U, Guditi S.** Chronic kidney disease screening in LMICs: benefits and challenges. Nat Rev Nephrol. 2025 Mar;21(3):145-146. doi: 10.1038/s41581-025-00929-2. PMID: 39809947.

Achievements

LAB JOURNAL

To catch 'em young

Researchers and start-ups are turning to microRNAs for early detection of critical illnesses.

GAURI KAMATH

Some years ago, nephrologist Mythri Shankar noticed patients, as young as in their 30s, turning up with end-stage kidney disease (ESKD) at the Karnataka government's Institute of Nephro Urology-Bengaluru. The reason was IgA nephropathy (IgAN) – a silent, autoimmune disease that, when left undetected, progresses over 10-15 years to ESKD. "The kidneys were burnt out," runs Mythri, Associate Professor in the Department of Nephrology. "There was nothing much we could do." Dialysis, while awaiting a transplant, was the only option. IgAN typically strikes people in their 20s and is more common in South and South-East Asia, explains Mythri. However, if detected early, treatment can retard disease progression.

The only diagnostic test for IgAN is a biopsy: inserting a needle into the organ to remove a piece of tissue to be studied under a microscope. This is an invasive process needing hospital admission – and with some risk of complications – that most would not volunteer for, until too late. There are also patients who cannot undergo a biopsy for medical reasons. Could an alternative for early detection be found?

Mythri sought answers in short sequences of nucleotides called micro ribonucleic acids (microRNAs or miRNAs) found in different body fluids. She engaged a private laboratory to extract miRNAs from urinary exosomes – extracellular vesicles shed by kidney cells and an abundant source of intact, un-degraded miRNAs.

Comparing urine samples of diseased patients and healthy controls, she identified five miRNAs, a so-called signature, whose presence was associated with 100% specificity and sensitivity. The findings were published in December 2021 in *Scientific Reports* (*bit.ly/miRNA-IgAnephropathy*). The next step is to collaborate with a company to create an RT-PCR screening kit. A successful miRNA screen could lead to new guidelines to detect IgAN early, she says.

Nephrologist Mythri Shankar reckons that diagnostics using miRNAs could be the future of disease detection.

There is a growing body of literature that shows the utility of specific miRNAs – and their upward or downward regulation – in flagging a range of conditions from infection to cardiovascular disease, neurodevelopmental disorders and cancers. Their potential to signal incipient disease has garnered interest from researchers and start-ups alike.

The master gene regulator

miRNAs are often referred to as master switches of gene regulation. They control gene expression by binding to messenger RNAs (mRNAs) to destabilise or silence their translation into proteins that impact human body functions. In so doing, they control a range of biological processes – from cell division to programmed cell death. The scientists who discovered miRNA and its role in gene regulation 30 years ago were awarded the 2024 Nobel Prize in Physiology or Medicine.

Over the years, research has pointed to miRNA's diagnostic and therapeutic value. A change in miRNA expression signals that "something is brewing", explains Lekha

40 IITM SHAASTRA | February 2025

Dr Mythri Shankar

selected for 2025-2026
Clinical Kidney Journal editorial fellowship

Glad to share that our research work on urinary miRNA in IgA Nephropathy is published as a special feature in IIT Madras Shastra Magazine.

Link here: https://shaastramag.iitm.ac.in/special-feature/catch-em-young

Dr Megha Pai won the

first place in women's doubles badminton Third place in mixed doubles badminton 3rd place in women's single table tennis
In the Indian medical association Udupi Karavali sports meet held in Ajjarkadu Udupi

Dr Payal Gaggar- selected for NDT Editorial fellowship 2025-26

GALLERY

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GALLERY

Women's day event by WIN Karnataka

GALLERY

Workshop on Mastering Abstract techniques @ St Johns National Academy of health sciences

In Patients **At Risk** of Rejection

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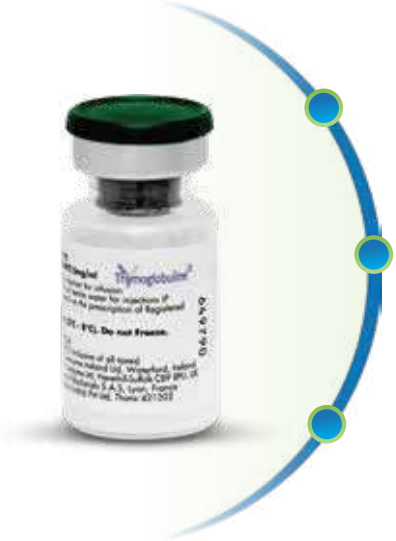
rATG is an **effective and well-tolerated** induction therapy in Indian patients undergoing renal transplantation[3]



Low incidence of acute graft rejection of 7.7% at 12 months[3]

Rejection-free graft survival rate of 92.3% at 12 months[3]

Low incidence of overall infection rate of 17.7%[3]

Abbreviations: rATG: Rabbit Anti-human thymocyte Globulin, USFDA: U.S. Food and Drug Administration

References: 1. Alloway RR, et al. Anti-human thymocyte immunoglobulin (Rabbit) for the prevention of acute rejection in kidney transplantation. American Journal of Transplantation. 2019 Aug: 19(8):2252-2261. 2. Gaber AO et al, Rabbit Antithymocyte Globulin (Thymoglobulin) 25 Years and New Frontiers in Solid Organ Transplantation and aematology. Drugs 2010: 70 (6): 691-732.
3. Adapted from Ray DS, et al. Poster Presented at 58 ERA-EDTA Congress, Fully Virtual, June 5-8, 2021, Please refer to the link for RISE abstract available on page 543 of the PDF: https://www.era-edta.org/en/virtualcongress2021/wp-content/uploads/sites/5/2021/05/NDTJ_36_Suppl-1_LR.pdf. *For the prophylaxis of acute rejection in patients receiving a kidney transplant.

Abridged Prescribing Information
Antihuman thymocyte immunoglobulin (Rabbit) E.P.
THYMOGLOBULINE@ 5mg/ml
Powder for concentrate for a solution for infusion
COMPOSITION: After reconstitution with 5 ml Water for Injection (WFI) I.P., the solution contains 5 mg rabbit anti-human thymocyte immunoglobulin/ml (concentrate) corresponding to 25 mg/5 ml of rabbit antihuman thymocyte immunoglobulin per vial. **THERAPEUTIC INDICATIONS:** Immunosuppression in transplantation: prophylaxis and treatment of graft rejection: Prophylaxis of acute and chronic graft versus host disease in haematopoietic stem cell transplantation: Treatment of steroid-resistant, acute graft versus host disease; Haematology: treatment of aplastic anaemia. **DOSAGE AND ADMINISTRATION:** The posology depends on the indication, the administration regimen and the possible combination with other immunosuppressive agents. Recommendations may be used as reference. The treatment may be discontinued without gradual reduction of dose. Administer doses of corticosteroids and antihistamines are required prior to infusion of rabbit anti-human thymocyte immunoglobulin. **SAFETY-RELATED INFORMATION:** Contraindications: Acute or chronic infections, which would contraindicate any additional immunosuppression. Hypersensitivity to rabbit proteins or to any product excipients. **Pregnancy and Lactation:** Thymoglobuline should not be given unless absolutely required. Breast feeding should be discontinued. **Warnings and Precautions:** Must be used in a hospital setting. Acute Infusion Dassociated reaction (IARs) may occur following the administration of Thymoglobuline and may occur as soon as the first or second infusion during a single course of Thymoglobuline treatment. In the event of an anaphylactic shock, the infusion has to be stopped immediately and any further administration must only be carried out after the benefits and the risks have been carefully weighed up. Thrombocytopenia and/or leucopenia have been identified: white blood cell and platelet count must be monitored during and after the treatment. Infections, reactivation of infection, and sepsis have been reported after administration of Thymoglobuline in association with several immunosuppressive agents. The use of immunosuppressive agents, including Thymoglobuline may increase the Incidence of malignancies. Reactions at the infusion site can occur and may include pain, swelling, and erythema. Immunization with attenuated live vaccines is not recommended for patients who have recently received Thymoglobuline. **ADVERSE REACTIONS:** Infection (including reactivation of infection). Sepsis, Lymphoproliferative disorder, Lymphomas (which may be virally mediated), Neoplasms malignant (Solid tumors), Febrile neutropenia, Disseminated intravascular coagulopathy, Coagulopathy. Cytokine release syndrome (CRS), Anaphylactic reaction, Serum Sickness (including reactions such as fever, rash, urticaria. arthralgia, and/or myalgia), Transaminases increased, Hepatocellular injury, Hepatotoxicity, Hepatic Failure, Infusion related reactions (IARS).
For full prescribing information please contact: Sanofi Healthcare India Private Limited, Sanofi House, CTS No. 117-B. L&T Business Park, Saki Vihar Road, Powal 400072.
Updated: November 2021

Source: 1) CCDS version no. 2 dated 16 July 2015. 2) UK Summary of Product characteristics dated 03 May 2015.

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