ISSUE 5 I VOL 3 I JULY TO SEPTEMBER 2025

Highlights

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

As we bring you the third edition of the Women in Nephrology–India (WIN-India) newsletter, we take this opportunity to celebrate the remarkable achievements of women nephrologists across the country. This edition continues to highlight WIN's ongoing commitment to mentorship, scientific growth, and collaboration.

In this edition, we feature a rare and insightful case report on **TRPM6-related hypomagnesemia with secondary hypocalcemia,** reminding us of the importance of careful metabolic evaluation and early genetic diagnosis in complex pediatric presentations.

We also present a vibrant summary of **WINICON 2025,** held in Chennai—a meeting that blended high-quality science, hands-on workshops, leadership dialogues, and cultural celebration. With national and international experts contributing to discussions spanning dialysis innovations, onconephro-pathology, and global opportunities, the conference embodied this year's theme: advancing nephrology through evidence, collaboration, and inclusivity.

A major milestone for WIN this year is the launch of the **WIN–India Clinical Research Training Fellowship**, designed to build research skills among young nephrologists and strengthen India's clinician-scientist pipeline.

We extend heartfelt thanks to our contributors, mentors, editorial team, and the entire WIN-India family. Your dedication and collective spirit continue to drive progress and create meaningful opportunities for women in nephrology.

We hope you enjoy this issue and look forward to an exciting year ahead filled with learning, leadership, and inspiration.

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Teaching Point

A Rare TRPM6 Gene Mutation Causing Hypomagnesemia and Secondary Hypocalcemic Seizures: Clinical Approach and Implications

Introduction:
Hypomagnesemia with secondary hypocalcemia (HSH) is a rare autosomal recessive disorder caused by mutations in the TRPM6 gene, which encodes a magnesium-permeable ion channel expressed in the intestine and distal convoluted tubule (DCT) of the kidney. Affected individuals typically present during infancy with seizures or tetany due to profound hypomagnesemia and resultant hypocalcemia. Early recognition and lifelong magnesium supplementation are crucial to prevent neurological sequelae.

Case Presentation
A 6-year-old female child, born of a consanguineous marriage with normal antenatal TIFFA scan, was delivered via lower segment cesarean section at 34 weeks of gestation due to oligohydramnios. Her birth weight was 1.9 kg. She cried immediately after birth and had no history of neonatal jaundice or seizures. Developmental milestones were appropriate for age, and she was exclusively breastfed until 4 months of age.

At 4.5 months, the patient presented with multiple seizure episodes. History revealed that she had received the DPT vaccine two days prior, followed by low-grade fever and an abscess at the injection site on the left thigh. On examination, the infant was drowsy, weighed 4 kg, and had no focal neurological deficits or other significant physical findings.

Investigation	Result	Normal / Reference Range	Interpretation
Serum calcium	3.2 mg/dL	8.5–10.5 mg/dL	Low (hypocalcemia)
Serum phosphate	4.8 mg/dL	2.5–4.5 mg/dL (adults; slightly higher in kids)	Mildly elevated
Serum magnesium	0.2 mg/dL	1.7–2.2 mg/dL	Low (severe hypomagnesemia)
Urine protein–creatinine ratio	1.2	<0.2 (no significant proteinuria)	Elevated
Serum albumin	2.0 g/dL	3.5–5.0 g/dL	Low (hypoalbuminemia)
CSF analysis	No infection	No organisms, normal cells/ biochemistry	Normal
MRI brain	Normal		No structural abnormalities
Neurosonogram	Normal	No hemorrhage/structural anomaly	No abnormalities detected
Urine exam	Unremarkable	No protein, blood, casts, or infection	Normal
Stool exam	Unremarkable	No ova, cysts, blood, or fat	Normal
Chest X-ray	Normal	Clear lung fields, normal heart size	No pathology
Skeletal X-rays	Normal	No fractures, rickets, or lytic/ sclerotic lesions	No rickets/other lesions
Metabolic panel	Normal	Normal Na, K, Cl, HCO_3, glucose, urea, creatinine, ammonia, lactate	Electrolytes and key metabolites normal
Vitamin D levels	23ng/ml	25(OH)D typically 20–50 ng/mL	Rules out vitamin D deficiency
Parathyroid hormone (PTH)	36pg/ml	~15–65 pg/mL (assay dependent)	Not consistent with primary PTH disorders

The patient was managed with intravenous phenobarbital (5 mg/kg/day in two divided doses) and parenteral calcium gluconate 10% (5 mL/kg/day in three divided doses), along with oral calcium and magnesium supplementation. After seven days of treatment, the patient was active, feeding well, and neurologically normal. She was discharged on oral magnesium sulfate, calcium supplements, and antiepileptic therapy.she was on magnesium sulphate 200 mg bd initially Subsequently, she was followed up regularly. Over the following years, she experienced intermittent fasciculations, poor dentition, and enamel loss. Magnesium sulphate dose increased to 400 mg BD.At the age of 4 years, due to persistent hypomagnesemia with serum magnesium levels 0.7mg/dl despite supplementation, she was referred to Nephrology for further evaluation.

Further Evaluation @ 4 years of age in Nephrology

Physical examination revealed mildly upslanting palpebral fissures, a high-arched palate, dental caries with steel caps (Figure 1), and wrist joint hyperlaxity (Figure 2).

Fig 1: Dental Caries with Steel caps

Fig 2: Hyperlaxity at Wrist joint

Investigations @ 4 years of age:

Parameter	Value	Reference range	Interpretation
Hemoglobin	9.7 g/dL	~12–16 g/dL (adult female) / 13–17 g/dL (adult male)	Mild anemia
Serum sodium	136mEq/L	135–145 mEq/L	Within normal limits
Serum potassium	3.8mEq/L	3.5–5.0 mEq/L	Within normal limits
Serum calcium	6.7 mg/dL	8.5–10.5 mg/dL	Low (hypocalcemia)
Serum magnesium	0.8 mg/dL	1.7–2.2 mg/dL (typical)	Persistent hypomagnesemia despite supplements
24-hour urinary calcium	7 mg/day	60–300 mg/day	Low; no hypercalciuria
24-hour urinary magnesium	20 mg/day	1.5–2.5 mg/day	Elevated urinary magnesium
Fractional excretion of magnesium	4%	<2–3% (approx.)	Increased renal magnesium loss

Renal magnesium wasting was localized to the distal convoluted tubule. As there was no associated hypokalemia or extra-renal manifestations, disorders involving the DCT were considered. Whole exome sequencing was performed, which identified a **homozygous mutation in the TRPM6 gene**, confirming the diagnosis of a**utosomal recessive hypomagnesemia with secondary hypocalcemia.(Fig 3)**

Test Results and Interpretation

HOMOZYGOUS VARIANT OF UNCERTAIN SIGNIFICANCE (VUS) CONSISTENT WITH PHENOTYPE DETECTED.

Summary of Variants

Gene and Transcript	Exon/Intron Number	Variant Nomenclature [Variant depth/ Total depth]	Zygosity	Classification	OMIM Phenotype	Inheritance
TRPM6 (NM_017662.5)	Exon 36	c.5607dup p.Asn1870GlnfsTer7 [Depth - 82X/82X]	Homozygous	Uncertain significance	Hypomagnesemia 1, intestinal	Autosomal recessive

Patient was managed with oral magnesium supplements.On follow-up, serum calcium stabilized at 8.2–10.4 mg/dL and magnesium at 0.8–1.3 mg/dL. The child remained asymptomatic, highlighting the success of early magnesium supplementation.

Discussion:

Magnesium is a vital electrolyte that plays a crucial role in many biochemical reactions in the human body, affecting cellular function, nerve conduction, and other needs. Normal serum magnesium levels are between 1.46 and 2.68 mg/dL. Hypomagnesemia is an electrolyte disturbance caused by a low serum magnesium level of less than 1.46 mg/dL in the blood. However, this condition is typically asymptomatic until serum magnesium concentration is less than 1.2 mg/dL (0.5 mmol/L).

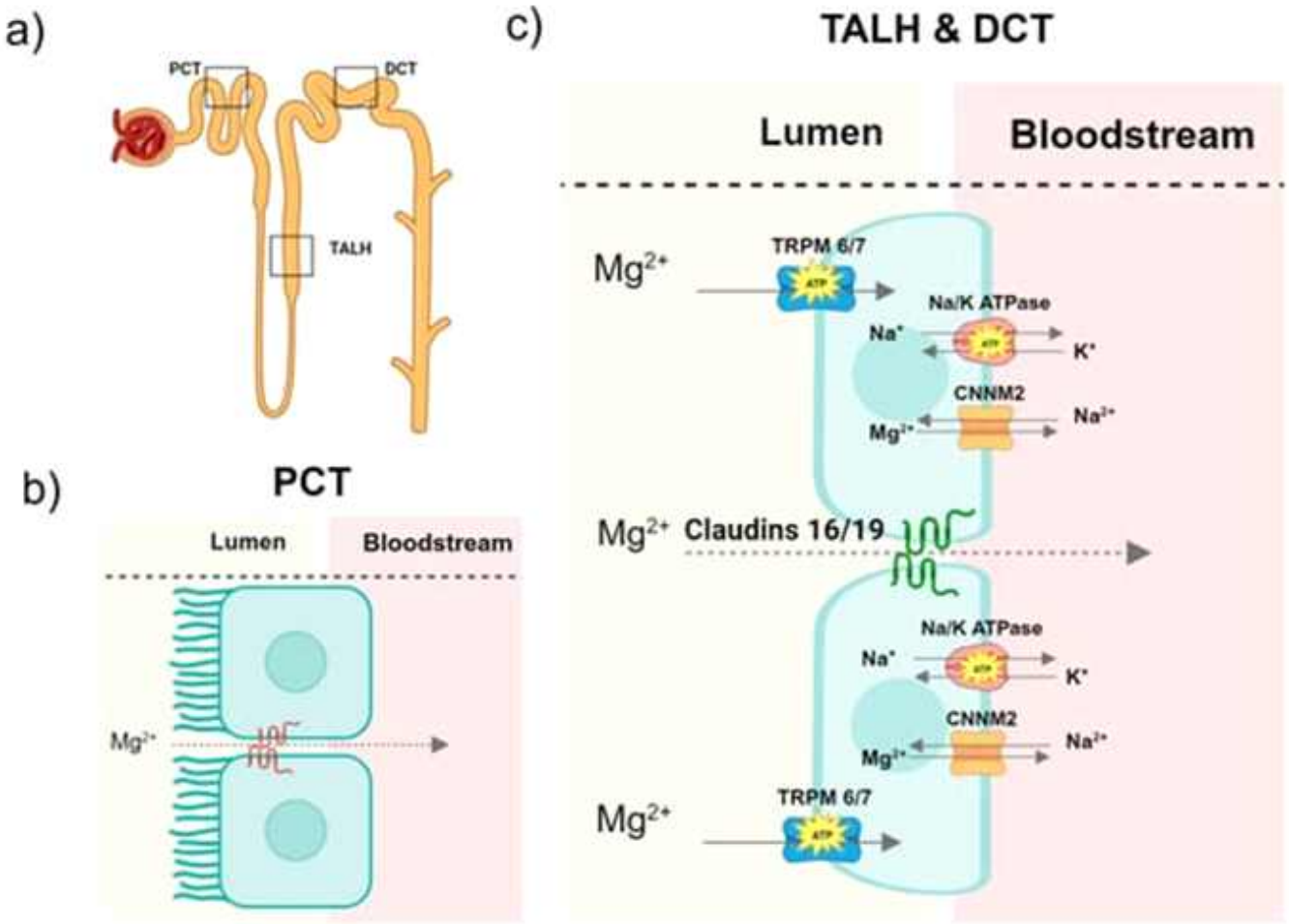
Magnesium handling:

Gastrointestinal absorption of Mg^{2+}

Mg^{2+} is acquired through daily food intake, where nearly 35%–80% of the ingested Mg^{2+} is absorbed . Specifically, around 30–50% of dietary Mg^{2+} intake is absorbed in the jejunum and ileum by way of passive paracellular route and in the colon through active transcellular route by transient receptor potential melastatin type 6 and 7 (TRPM6 and 7). The absorbed Mg^{2+} enters the bloodstream by CNNM4 and Na^{+} -Mg^{2+} exchanger at the basolateral side of cells

Renal regulation of Mg2+ reabsorption

Serum Mg2+ is subsequently excreted in the urine after storage in organs and cellular utilization. The kidney determines the final urinary Mg2+ excretion, and hence plays a major role in Mg2+ homeostasis. In the kidney, only 3–5% of filtered Mg2+ is excreted after reabsorption in renal tubules. around 15–25% of filtered Mg2+ is absorbed in the PCT via paracellular pathway. The positive intraluminal voltage is generated by apical Na + -K+-Cl-cotransporter (NKCC2)-mediated Na+, K+, Cl– reabsorption, and apical renal outer medullary potassium (ROMK)-mediated parallel K+ excretionThe majority of filterable Mg is actively reabsorbed in the thick ascending loop of Henle (TALH) via a divalent cation channel transient receptor potential melastatin 6/7 (TRPM6/7) and paracellularly with claudins 16/19. Once inside the cell, Mg is then transported into the bloodstream via a Mg/sodium antiporter, Cyclin, and the CBS domain Divalent Metal Cation Transport Mediator 2 (CNNM2) by utilizing the electrochemical gradient of sodium generated by the sodium/potassium ATPase. Mg transport in the distal convoluted tubule (DCT) is thought to be the same as in the TALH.8



Etiological and Genetic Framework of Hypomagnesemia

Category	Representative Causes	Mechanism	Key Genes Involved
Reduced Intake	Malnutrition, anorexia, chronic alcoholism, prolonged fasting, elderly, inadequate parenteral nutrition	Inadequate dietary magnesium leading to total body depletion	
Gastrointestinal Losses	Chronic diarrhea, malabsorption, celiac disease, pancreatitis, PPI therapy (TRPM6 inhibition), short bowel syndrome	Decreased intestinal absorption or excessive GI loss	**TRPM6, TRPM7** (intestinal Mg^{2+} transport channels)
Renal Magnesium Wasting — Drug Induced	Loop/thiazide diuretics, cisplatin, aminoglycosides, amphotericin B, calcineurin inhibitors (tacrolimus, cyclosporine)	Impaired tubular Mg reabsorption in TAL/DCT due to transporter inhibition or toxicity	**CLDN16, CLDN19, FXYD2, HNF1B, CNNM2** (renal tubular Mg handling)
Renal Magnesium Wasting — Genetic Tubulopathies	Gitelman syndrome, Bartter syndrome, familial hypomagnesemia with secondary hypocalcemia	Defects in NaCl or K^+ channels affecting Mg reabsorption	**SLC12A3** (Gitelman), **KCNJ1, CLCNKB,** (Bartter), **TRPM6** (FHSH)
Renal Magnesium Wasting — Endocrine/Metabolic	Hypercalcemia, hyperaldosteronism, diabetic ketoacidosis recovery	Altered tubular potential gradient or hormonal modulation of TRPM6 expression	**CASR** (calcium-sensing receptor modulating TAL Mg transport), **NR3C2** (mineralocorticoid receptor)
Post-Transplant State	Calcineurin inhibitor use, tubular injury, secondary hyperaldosteronism	Downregulation of TRPM6 expression, impaired DCT reabsorption	**TRPM6, CLDN16** (reduced expression under CNI therapy)
Redistribution / Intracellular Shift	Refeeding syndrome, insulin therapy, catecholamine surge, hungry bone syndrome, pancreatitis, massive transfusion	Shift of Mg^{2+} into cells or sequestration in bone/fat necrosis	
Mixed or Multifactorial	Alcoholism, critical illness, chemotherapy, post-transplant state	Combination of reduced intake, GI loss, renal loss, and redistribution	Multiple interacting pathways; possible variants in **TRPM6, CNNM2, HNF1B** contributing to susceptibility

Algorithmic Approach to Hypomagnesemia

↓ LOW SERUM MAGNESIUM (<1.7 mg/dL or <0.7 mmol/L)

STEP 1: Confirm True Hypomagnesemia
· Repeat serum magnesium
· Exclude pseudohypomagnesemia (hyperlipidemia, paraproteinemia

STEP 2: Assess Associated Abnormalities
· Serum K^+, Ca^{2+}, PO_4^{3-}, PTH
→ Hypokalemia or hypocalcemia s

STEP 3: Evaluate Clinical Context
· GI loss: diarrhea, vomiting, PPI use
· Renal loss: diuretics, cisplatin, calcineurin inhibitors
. Redistribution: insulin therapy, refeeding, catecholamine surge
. decreased absorption: alcoholism, malnutrition

STEP 4: Assess Urinary Magnesium
Study 24-hour urine magnesium or FEMg (%)
- FEMg <2% → Extrarenal (GI) loss
- FEMg >4% → Renal magnesium wasting

STEP 5: Identify Underlying Cause
· Endocrine/metabolic (hyperaldosteronism, DKA)
· Genetic (Gitelman, Bartter, TRPM6 mutation)

In our study, a structured approach was applied to evaluate patient with documented hypomagnesemia. This framework enabled systematic differentiation between renal and extrarenal causes

Table Comparative summary of published TRPM6 cases

	No. of patients / Family	Mutation(s)	Age at presentation	Main presentation	Treatment / Outcome
1. Schlingmann et al., 2002	28 individuals - 21 families	Various loss-of-function TRPM6 mutations	Infancy	Seizures due to hypocalcemia secondary to hypomagnesemia	Serum magnesium levels remained in the subnormal range despite adequate therapy. Delay of diagnosis resulted in permanent neurologic damage in three patient

Study	No. of patients / Family	Mutation(s)	Age at presentation	Main presentation	Treatment / Outcome
2. Walder et al., 2002	Families with familial HSH	TRPM6 mutations (familial)	Infancy	Seizures, tetany	several variants in TRPM6 gene have been reported to cause intestinal hypomagnesemia can also cause renal magnesium wasting
3. Apa et al., 2008 (Indian J Pediatr)	Case report (single patient)	TRPM6 mutation (reported)	Infancy	Hypomagnesemia with secondary hypocalcemic seizures	Responded to Mg therapy; case-level outcome favorable
4. Altıncık et al., 2016	Single-family / case	Homozygous TRPM6 mutation	Infancy	Hypomagnesemia, seizures	Oral Mg replacement, favorable outcome
5. Han et al., 2022 (case report + review)	Twins	Novel TRPM6 mutations reported	early childhood	Febrile seizures	convulsions caused by the novel mutation might have been potentially induced or aggravated by fever
6. kamali et al	Case report	Novel TRPM6 mutations om exon 26	infancy	Seizure,watery diarrhea	Failure to thrive , improved with magnesium supplement
7. Chandreyee et al	Case report	Novel TRPM6 heterozygous mutation	infancy	Seizure	Improved with magnesium supplement

Conclusion

This case underscores the importance of considering TRPM6-related hereditary hypomagnesemia with secondary hypocalcemia in infants presenting with recurrent or calcium-resistant seizures. Early recognition through targeted biochemical screening and confirmatory genetic testing is crucial, as timely magnesium supplementation can lead to rapid and sustained resolution of neurological symptoms. The identification of a novel TRPM6 mutation expands the known genetic spectrum of this rare disorder and reinforces the need for heightened clinical awareness, particularly in populations with high rates of consanguinity. Prompt diagnosis not only prevents unnecessary investigations and treatments but also significantly improves neurological outcomes

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Reflections from WINICON 2025.

WINICON 2025, the 4th Annual Conference of Women in Nephrology–India, was held in Chennai on August 30th and 31st, 2025, co organised by Women in Nephrology Tamilnadu and Puducherry Wing and Keondy events, uniting over 300 nephrologists, researchers, and healthcare professionals from across India and abroad for an inspiring celebration of science, clinical innovation, and gender equity in kidney care. Themed **"Practicing Advanced, Evidence-Based Nephrology with Impactful Collaboration,"** the conference focused on advancing kidney health through leadership, partnership, and professional development.

Event Highlights

WINICON 2025 showcased a dynamic blend of academic sessions led by eminent national and international faculty, including interactive panel discussions on clinical challenges, hands-on workshops on Onconephro-pathology and Dialysis Technology, and a vibrant cultural program. The **4th Prof Muthu Jayaraman Oration** was delivered by Dr. A Vimala, an embodiment of empathy, compassion and dedication; from Trivandrum for her exemplary work towards growth of Nephrology in Kerala over 4 decades and project on snake envenomation project. The Souvenir, **WINSPIRE** was released in the presence of Dr Sundar Sankaran, a Nephrology Giant from Bangalore. It is beautiful compilation of powerful messages from various Nephrology leaders throughout the country, motivating ideas from the Gems of the field, a glimpse into the vibrant city of Chennai and magic of Women beyond Nephrology, put forth by our colleague, Dr P. Rajeevalochana.

Dr Harshini D Perera from Sri Lanka shared her experience on CRRT with dialysis technicians and gave update on ADPKD topic. Charu Malik, executive director ISN chose online platform to address young minds to discuss various opportunities in ISN.Dr Anuja Java from USA, Dr Roser Torra from Spain, Dr Soumita Bagchi from Australia and Dr Aarthi M from UK were other online speakers who shared their expertise on various topics.

Quiz competition for postgraduates with 5 teams entering final round and each giving other tough time for the trophy left all glued to their seats till end. 5 best oral presentations and 70 odd posters kept the judges enthralled and engrossed. Delegates participated in networking events, fostering robust professional connections and knowledge exchange. Trade exhibition was well attended with 26 pharma stalls and an ISN booth to facilitate and showcase its upcoming events through brochure.

Leadership and Vision

The conference was inaugurated by Chief Guest Dr. Soumya Swaminathan (Former Chief Scientist, WHO), alongside prominent leaders like Dr. Shyam Bansal (Indian Society of Nephrology), Dr. Arpita Ray Choudhary (ISOT), and Mrs. Vasundhara Raghavan (Kidney Warriors). Leaders from WIN India, including Dr. Manisha Sahay (President), Dr. Swarnalatha Guditi (Secretary), Dr. Jayalakshmi Seshadri (Organising Chairman), and Dr. Ranjanee Muthu (Organising Secretary), emphasized the importance of mentoring, solidarity, and innovation in driving the future of nephrology. Prof Amit Gupta, Retd. Professor and HOD, Dept. of Nephrology, SGPGI and currently Director Nephrology, Apollo Medica, Lucknow also participated in the conference.

Scientific and Cultural Impact

Over its two days, WINICON 2025 championed the empowerment of women specialists, promoted research, nurtured early-career nephrologists, and underscored the need for collaborative approaches to kidney health. The event also celebrated Tamil Nadu's cultural heritage, highlighting the unique spirit of Chennai as a centre for both tradition and innovation. The ambience of the venue, The Residency Towers, T Nagar right in the heart of the city, surrounded by shopping centres displaying esp. rich saree collections and its easy access from the airport excited the delegates and above all the hospitality services were impeccable. The cuisine was a blend of comfort food with traditional and creative topping with mouth-watering desserts.

Closing Reflection

WINICON 2025 stands as a milestone in the ongoing journey to advance Nephrology in India, combining academic excellence, cultural richness, and a resounding commitment to gender-inclusive leadership—each delegate left inspired and equipped with knowledge, professional networks, and renewed vision for patient-centered kidney care and with the lingering memories of filter coffee aroma ,saga of sarees and authentic tamil cuisine.

Dr Ranjanee Muthu

DM,CHS ,SCE (Neph),FASN, FIMSA
WINICON 2025,
Organising Secretary.
Senior Consultant Nephrologist and Transplant Physician
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Women in Nephrology – India

FROM IDEA TO IMPACT: CLINICAL RESEARCH TRAINING FELLOWSHIP

(NOVEMBER 2025 TO MARCH 2026)

Are you a nephrology resident or consultant who wants to publish impactful research, secure grants, and present on global stages?
This unique Clinical Research Training Fellowship is built for nephrologists – blending rigorous methodology, practical tools, and mentorship.

Why Join?

- Structured Learning: From research question → study design → biostatistics → writing & publication.

Sandwich Model:

- Inaugural Session (Hyderabad, Nov 2, 2025) – Kickstart with live workshops.
- Interactive Online Modules (Nov–Mar, Tuesdays 7–8:30 pm) – Learn, discuss, and practice.
- Valedictory (Bengaluru, Mar 28, 2026) – Showcase your work, celebrate, and network.

Events

- Hands-On Training: Journal clubs, case discussions, visual abstracts, grant writing, ethics & regulatory insights.
- Faculty Mentorship: Learn from a stellar panel of nephrologists and clinical research experts.

Who Can Apply?

- Nephrology Residents & Consultants
- Age no bar – just curiosity and commitment!

Key Takeaways

- Develop a complete research proposal
- Master biostatistics essentials with practical tools
- Learn to write, publish & present effectively
- Understand grants, ethics & research regulations
- Build confidence to lead research projects in nephrology

Scan QR to register

REGISTER NOW

Dates

- 2 Nov 2025 (Hyderabad) – Orientation & Foundation
- Nov 2025 – Mar 2026 – Weekly Online Modules
- 28 Mar 2026 (Bengaluru) – Closing & Awards

For registrations & details:

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Seth G.S.M.C. and K.E.M. Hospital,
Mumbai

Dr. Priyadarshini John
Assistant Professor of Nephrology
Osmania General Hospital
Hyderabad

Nephrology Residents & Consultants Clinical Research Training Program -Curriculum

November 2025 to March 2026; Tuesday 7:00 pm to 8:30 pm

SL.NO.	DATE	TITLE / TOPICS COVERED	METHODOLOGY
1	2 November 2025	• In person Hyderabad • Inaugural • Program Orientation • 1.How to find and structure a Research Question • 2. Study, Objectives, primary, secondary. • 3. Overview of Study designs • 4. Efficient Literature review using modern tools • 5. Project proposal outline • 6. Best practices for online learning. Methods for the day: Short lectures, group discussion and interactive sessions.	
SECTION 1: Research Question and Study Designs (Online)			
2	10 Nov 2025	• Cross Sectional • Registry	• Short lectures focusing on methods, results and interpretation • Examples from relevant journals • STROBE, CONSORT, PRISMA, COREQ
3	22 Nov 2025	• Case Control Studies	
4	24 Nov 2025	• Cohort Studies	
5	2 Dec 2025	• Randomized Controlled Trials • JC on Cross Sectional design	
6	9 Dec 2025	Use of AI for Literature search	
6	16 Dec 2025	• Systematic Reviews & Meta Analysis • JC on Case Control Studies	

Events

SL.NO.	DATE	TITLE / TOPICS COVERED
SECTION 2: Biostatistics and Data Management (Online) • Short lecture on key principles • Several illustrations • Examples from Nephrology journals		
7	6 Jan 2025	• Types of data • T tests • **JC on Cohort design**
8	13 Jan 2025	• ANOVA • MANOVA • ANCOVA • **JC on Randomized Controlled Trials**
9	20 Jan 2025	• ·Chi squared tests • Mann Whitney U • Kruskall Wallis • Wilcoxon's
10	27 Jan 2025	• Correlation: Pearson's and Spearman's • Regression: Linear, Multiple Linear & Multiple Logistic Regression
11	3 Feb 2025	· Survival analysis; Kaplan Meier curves, Hazard ratio, Cox models
12	10 Feb 2025	• Sample Size Estimation • Data needed for SS • Principles in SS Estimation • Free Software • How to work with Statisticians
13	17 Feb 2025	• Data Collection Instruments (DCI or CRFs) • Data Bases • Quality Checks
SECTION 3: Research Managment: Regulatory & Ethics, Grants, Presentn & Publicn (Online)		
14	24 Feb 2026	• Key Principles in Research Ethics • Essential Regulations in India • Registering Studies · Dealing with Ethics Committees
15	3 March 2026	• Funding for Research · • Internal, External and International Grants • Key principles in Grant Writing • Submitting grants • Grant Review Process · • Managing Research Funding

Events

SL.NO.	DATE	TITLE / TOPICS COVERED
16	10 March 2026	• Scientific Writing • Sections of a Journal Article • Plagiarism Check • Submitting to Journals • Responding to Reviewers • Publ. Metrices • **Zen Protocol 1 & 2**
17	17 March 2026	• Oral/ Podium Presentation • Poster Presentation • Online Presentation • **Zen Protocol 3 & 4**
18	23 March 2026	• How to create Visual abstract?
19	28 March 2026	• **IN-PERSON – Bangalore:** • Lecture on Building a Research Culture • Feedback, Valedictory & Prize Distribution

For any queries, please contact:

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4th ANNUAL CONFERENCE OF
WOMEN IN NEPHROLOGY INDIA
WIN-ICON 2025
Date : 30th - 31st August 2025
Venue : The Residency Towers, T Nagar Chennai, Tamilnadu
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4th Annual Conference of
Women in Nephrology India
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On Onco Nephropathology


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2025
Date : 30th - 31st August 2025
Venue : The Residency Towers,
T Nagar Chennai, Tamilnadu

GALLERY

@ WINCON 2025

GALLERY

Day 1 @ Clinical Research Training Fellowship

WOMEN IN NEPHROLOGY INDIA (WIN INDIA)

In Association With

THE INTERNATIONAL SOCIETY OF NEPHROLOGY (ISN)

IS PLEASED TO SPONSOR ISN MEMBERSHIP FOR NEPHROLOGY RESIDENTS IN THEIR

2nd and 3rd year

Limited slots available! Hurry up and grab the opportunity!

Eligibility Criteria:

- Must be a member of WIN India
- 1st-year member of the International Society of Nephrology (ISN)

SPONSOR $15 PER YEAR FOR THE NEXT TWO YEARS

APPLICATIONS WILL BE ACCEPTED ON A FIRST-COME, FIRST-SERVED BASIS,
LAST DATE 5TH NOVEMBER 2025

Click & apply through the following link

https://winindia.org/form.php

In Patients **At Risk** of Rejection

Rx Thymoglobuline®

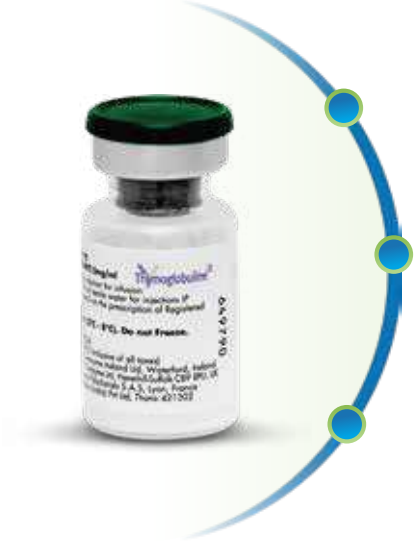
Anti-human thymocyte immunoglobulin (Rabbit) E.P.

More than 35 years of worldwide experience[2]

WHEN TRUST MATTERS...

First T cell-depleting therapy approved by USFDA*[1]

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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