NZ-ISNCON-2026
30th Annual Conference of the
Indian Society of Nephrology Northern Zone
10th-12th April 2026, Hyatt Centric, Dehradun

Programme for NZ-ISNCON 2026

10th April 2026

Time	Topic
1 (04:00-04:20pm)	Rituximab in MN
2 (04-20-04:40Pm)	Thymoglobulin in Kidney Transplant- Personal experience.
3 (04-40-05:00pm)	Multiplex PCR in post-kidney transplant Diarrhoea
4 (05:00 – 05:20 PM)	START trial
5 (05:20– 05:40 PM)	Crescentic IgA nephropathy
6 (05:40– 06:00 PM)	Phenotype and Genotype correlation in Indian children with Renal Tubular Acidosis: unravelling the mystery of inheritance.
7 (06:00– 06:20 PM)	AVF angioplasty- Experience from SGPGI
8 (06:20 – 06:40 PM)	Hepatitis B- Experience
9 (06:40– 07:00 PM)	Management of central venous stenosis

11 April 2026

Time	Topic	Objective
8:00-8:15 am	Welcome address	
8:15-9:00 am	Slippery slopes of Nephrology	To discuss the low-value care in Nephrology and steps to address the same
9:00-09:45 am	**Case 1:** A 29yr old male with end stage kidney disease of unknown aetiology, on thrice weekly maintenance haemodialysis, underwent an ABOi kidney transplant with his mother (51yr old female) as donor. There were 5/12 mismatches in HLA and both CDC crossmatch and flow cytometry crossmatch were negative before transplant. There were also low-titre donor-specific antibodies detected against DPA1*01:03:DPB1*04:02 (MFI–1070). The baseline anti-B titre (IgG isohemagglutinin) was 1:16, and he underwent pretransplant desensitization with rituximab (200mg) given 2 weeks before transplant. Tacrolimus and MMF were started 1 week before transplant. He received ATG induction (75mg on POD0 and 75mg on POD1) and triple immunosuppression (steroid, MMF, tacrolimus). In the immediate post-operative period he had good urine output with Creatinine reducing to 3.2mg/dl on POD 2. Anti B titer repeated on POD 1 had reduced to 1:1. However, on the evening of POD 2, the patient developed oliguria and had to be started on diuretics. Labs showed evidence of severe thrombotic microangiopathy (severe anemia, thrombocytopenia and high LDH) with graft dysfunction. Complement factor C3 and C4 were normal and direct comb's test was negative. Tacrolimus was withheld due to suspicion of CNI induced TMA. Doppler of the graft kidney was normal. Serum creatinine continued to rise, peaking at 4.83 mg/dL.He was given 1 session plasmapheresis on POD 3 will full FFP replacement. Over the course of the next few days, he received multiple blood products (10 RDP, 1 SDP, 5 LDPRBC) due to ongoing haemolysis and thrombocytopenia. Despite plasmapheresis there was	**Case 1:** • How would you manage this patient?

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no clinical improvement. Given the unavailability of eculizumab at that time and the patient’s worsening condition, graft nephrectomy was contemplated.

Case 2:
40 y/o Female with ESRD and on dialysis for 1 month with unknown
etiology, with her husband as donor. Rest of her workup is normal.
After you rule out her husband as donor- due to crossmatch positivity,
one of her 2 sons come forth as potential donor though Ankita does
not want them to donate. Immunological workup is below.

<table>
<tr><td>Sensitization History</td><td>2 pregnancies</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>HLA typing</td><td>A</td><td>B</td><td>C</td><td>DRB1</td><td>DQB1</td><td>DQA1</td></tr>
<tr><td>Donor (Husband)</td><td>02:11, 24:07</td><td>08:01 , 52:01</td><td></td><td>03:01, 10:01</td><td>02:01, 05:01</td><td>01:01, 01:01/ 04</td></tr>
<tr><td>Elder Son</td><td>11:01, 24:07</td><td>35:03 ,52:0 1</td><td></td><td>10:01, 13:01</td><td>05:01, 06:03</td><td>Not done.</td></tr>
<tr><td>Recipient</td><td>11:01, 24:02</td><td>35:03 , 51:01</td><td></td><td>04:04, 13:01</td><td>03:02, 06:03</td><td>01:03, 03:01</td></tr>
<tr><td>XM</td><td>T cells</td><td>B Cells</td><td></td><td></td><td></td><td></td></tr>
<tr><td>CDC XM (husband)</td><td>Positive</td><td>Positive</td><td></td><td></td><td></td><td></td></tr>
<tr><td>Flow XM (Husband)</td><td>Positive</td><td>Positive</td><td></td><td></td><td></td><td></td></tr>
<tr><td>Single Antigen Bead (Ankita Report 1)</td><td>Class I : Multiple anti HLA Abs</td><td>Class II: Multiple Anti HLA Abs.</td><td></td><td></td><td></td><td></td></tr>
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Case 2:

Should we even consider Son as husband has a positive crossmatch?
Which of the antibodies are Donor Specific against husband?
Role and Importance of DQ and DP antibodies.?
How would you evaluate these donors and decide proceeding for transplant? How do you evaluate multiple donors from family?

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<td>9:45-10:30 am</td>
<td>Management of anaemia</td>
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The role of HIF -PHI in the management of anaemia in CKD.
Role of HIF-PHI in special circumstances like PRCA
Indian experience in management of anaemia using HIF-PHI
Strategies to address ESA resistance in CKD

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<td>10:30-10:45 am</td>
<td>Tea Break</td>
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No specific objective

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<td>10:45-11:30 am</td>
<td>A 28-year-old man was found to have mildly elevated blood pressure during a routine health check, with a reading of 138/90 mmHg. He had no symptoms and felt well at the time. Initial urine testing showed abnormalities, with 2+ red blood cells and 2+ protein. Further evaluation showed normal kidney function, with a serum creatinine of 0.8 mg/dL. A 24-hour urine test revealed protein excretion of 1.2 g/day, indicating sub-nephrotic range proteinuria. Because of persistent protein in the urine, microscopic blood in the urine, and borderline high blood pressure in a young person with normal kidney function, he was referred to a nephrologist. After clinical evaluation and review of investigations, a kidney biopsy was advised to identify the cause. The kidney biopsy showed mesangial expansion with endocapillary hypercellularity. According to the Oxford classification, the biopsy was graded as M1 E1 S0 T0 C0, confirming a diagnosis of IgA nephropathy. These findings indicated active inflammation in the kidneys without evidence of chronic or irreversible damage. The patient was counselled about IgA nephropathy, its variable course, and available treatment options. He was started on supportive treatment, including renin–angiotensin system blockade with telmisartan 40 mg twice daily, to control blood pressure and reduce proteinuria. He was also advised on lifestyle measures such as salt restriction and regular blood pressure monitoring. As proteinuria persisted</td>
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The role of CR-Budesonide in management of IgA nephropathy
Evidence for the role of budesonide in IgA nephropathy
Discussion on the Indian data on CR-Budesonide in IgA nephropathy

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	despite supportive care and the biopsy showed active disease (M1E1) with preserved kidney function, treatment with controlled-release budesonide was initiated. He was advised to continue treatment for 9 months. The patient was informed in detail about the reason for treatment (numerous option- anti-proteinuric plus immunosuppression), expected benefits, and possible side effects. Close follow-up was planned, and he was asked to return in 2–3 weeks for reassessment of blood pressure, kidney function, proteinuria, and treatment tolerance.	
11:30-12:00 PM	Management of Vasculitis-ANCA	By experts in the field
12:00-12:30 pm	Management of Multiple Myeloma what nephrologist should know	By experts in the field
12:30-1:30 pm	Climate change is real—but our (global and not India) approach is hypocritical	Debate
1:30-2:15 pm (Lunch Symposium)	Newer Potassium Binders in Hyperkalemia management- Are they worth the debate in Indian Scenario	• Role of sodium zirconium cyclosilicate in management of acute hyperkalemia • Dosing protocol for its use for treatment of hyperkalemia • Role as a maintenance agent for hyperkalemia
2:15-2:30 PM	Break	
2:30-3:15 PM	A challenging case of complement-mediated TMA	• To discuss the role of eculizumab in CM-TMA • To discuss role of PLEX and eculizumab in anti-CFH-TMA • Interpretation of genetic testing in patients with CM-TMA • Functional testing for complement activity • Procurement of eculizumab using the rare-disease programme
3:15-4:00 PM	Two lectures by ICMR scientist on how to write proposals	
4:00-4:15 PM	**Tea Break**	
4:15-5:15 PM	A 73-year-old gentleman, a former small-business owner, presented with progressive lethargy, reduced exercise tolerance, anorexia, and generalized itching for nearly one year. His background medical history is notable for Type 2 diabetes mellitus for over 25 years, complicated by advanced diabetic retinopathy with significant visual impairment and diabetic autonomic neuropathy. Over the last 6–9 months, he has developed recurrent postural dizziness, early morning falls, and episodic presyncope, particularly on dialysis days. He is a known case of ischemic heart disease, having sustained an inferior wall myocardial infarction 6 years ago, followed by coronary artery bypass grafting. He remains on antiplatelet therapy and beta-blockers, with limited tolerance to aggressive volume shifts. In addition, he has a history of recurrent urinary tract infections, including one episode of emphysematous cystitis 18 months ago and another episode of urosepsis requiring ICU admission in the past year. He was diagnosed with secondary membranous nephropathy nearly 7 years ago in the setting of long-standing diabetes and chronic infections. Given his age, comorbidity burden, and repeated infective episodes, immunosuppressive therapy was deferred, and he was managed conservatively. Despite optimal supportive care, he progressed to end-stage kidney disease. Dialysis and Vascular Access History The patient was initiated on maintenance haemodialysis 11 months ago through a right internal jugular tunnelled dialysis catheter due to poor superficial veins and delayed referral. A left brachio-cephalic arteriovenous fistula was subsequently created. Although the fistula matured clinically, it was complicated by high venous pressures and suboptimal dialysis adequacy. Doppler evaluation revealed outflow stenosis, for which he underwent percutaneous transluminal angioplasty on two occasions. Currently, the fistula supports a blood flow of approximately 230–260 mL/min, with intermittent access alarms during dialysis sessions. Infectious and Nutritional Concerns The dialysis course has been complicated by one episode of catheter-related bloodstream infection and repeated lower urinary tract infections with prior cultures growing extended-spectrum beta-lactamase–producing organisms. He has experienced progressive weight loss	The patient now presents with worsening pruritus, generalized weakness, bone pain, and declining functional independence. Family members express concern regarding his quality of life and frequent hospital visits. The treating team is faced with multiple management challenges: • What is the dialysis optimising strategies in this case • Improving dialysis adequacy with HDF in the setting of limited AVF flow and intradialytic hypotension • What are the factors predicting a better outcome with HDF • Indian data on the quality of life with HDF • How do you manage skin discolouration in a patient with ESKD • How do you manage intractable pruritus in a patient with ESKD • Managing severe secondary hyperparathyroidism in an elderly patient with ischemic heart disease, what are the targets and indications for parathyroid surgery • Addressing recurrent infections and minimizing further vascular access–related complications • Optimizing anaemia and nutritional status amid chronic inflammation 10 minutes: Role of central dialysis fluid delivery system in a haemodialysis facility

	and poor nutritional intake, with intermittent low-grade inflammatory markers, raising concern for chronic inflammation and protein-energy wasting. Current Clinical Assessment On examination, the patient appears elderly and frail, with reduced muscle bulk and slow gait. His skin shows diffuse hyperpigmentation with a greyish-brown hue, especially over the face, neck, and extensor surfaces. There is marked xerosis, excoriations, and pruritic nodules consistent with chronic uremic pruritus. He also reports altered taste sensation and restless sleep. Blood pressure measurements demonstrate significant orthostatic hypotension, with systolic blood pressure falling from 148 mmHg supine to 112 mmHg on standing. Dialysis sessions are frequently interrupted due to intradialytic hypotension, limiting ultrafiltration and resulting in persistent volume and uremic symptoms. Laboratory Investigations Recent laboratory parameters are as follows: • Haemoglobin: 9.1 g/dL, on erythropoiesis-stimulating agents • Blood urea nitrogen: 164 mg/dL • Serum creatinine: 9.8 mg/dL • Corrected serum calcium: 8.6 mg/dL • Serum phosphorus: 6.4 mg/dL • Intact parathyroid hormone (iPTH): 842 pg/mL • Serum albumin: 3.1 g/dL • Ferritin: elevated with borderline transferrin saturation These findings indicate suboptimal dialysis adequacy, advanced CKD-mineral and bone disorder, anemia of chronic kidney disease with inflammation, and protein-energy wasting.	
515-545	Esaxerenone- an antihypertensive and anti proteinuric therapy	Dr Amit Gupta Dr P P Varma Dr Raka Kaushal Dr Sanjiv Saxena Dr Vijay Kher
06-00 Pm	GBM	
07-30 Pm	Inauguration and Dinner	

12th April 2026

Time	Topic	
8:00-830 AM	A challenging case- Peritoneal Dialysis	Discussion on clinical challenges (not logistical or financial) while managing patients on peritoneal dialysis- 4 to 5 clinical scenarios
830-915 AM	**Case 1** A 32-year-old male with biopsy-proven IgA nephropathy progressed to end-stage kidney disease (ESKD) and is planned for a living donor kidney transplantation, with his mother as the donor. His sensitizing history includes transfusion of four units of packed red blood cells (PRBCs) approximately eight months prior, at the time of initial diagnosis and management of his primary disease. His current immunological workup reveals: • ABO compatible • Negative complement-dependent cytotoxicity (CDC) crossmatch • Negative flow cytometry crossmatch • Single antigen bead (SAB) assay negative for donor-specific antibodies (DSA) against HLA-A, HLA-B, HLA-DR, and HLA-DQ • Panel reactive antibody (PRA) low/absent He has no prior transplant history and no other known sensitizing events. His comorbidities include well-controlled hypertension, with no diabetes or cardiovascular disease. **Case 2** A 33-year-old woman with ESKD secondary to analgesic nephropathy has been on maintenance haemodialysis for three years. She is scheduled to undergo a living donor kidney transplantation, with her husband as the donor (spousal transplant).	• How would you risk-stratify patient immunologically? Would you consider this low-, intermediate-, or high-risk? • What would be your approach to induction immunosuppressive therapy in the two clinical scenarios • What is the Indian experience with use of Induction agents: Thymoglobulin • Evidence in favour of low-dose ATG in kidney transplant • Rold of CD3 monitoring in patients receiving thymoglobulin

	Her sensitizing history includes:
	• Two prior pregnancies
	• One spontaneous abortion
	• Two PRBC transfusions
	Immunological evaluation shows:
	• ABO compatible
	• Negative CDC crossmatch
	• Flow crossmatch pending/weakly positive (if relevant to discuss)
	• SAB assay demonstrating anti-HLA antibodies, including:
	  ○ HLA-A*02:02 with MFI 2400
	  ○ HLA-B*03:01 with MFI 2100
	Calculated PRA is moderately elevated. No prior transplant history.
	She has secondary hyperparathyroidism and mild left ventricular hypertrophy but is otherwise clinically stable.	

9:15-10:00 AM	Debate (AI will take away our prescription job)	
10:00-10:15 AM	**Tea Break**	
10:15-10:45 AM	Prof KK Malhotra Oration: Prof Narayan Prasad, SGPGIMS, Lucknow	
10:45-11:15 AM	Presidential oration	
11:15-1130 AM	ATG-Grafalon in Kidney Transplant	
11:30-12:10- PM	**Case 1** A 54-year-old gentleman with type 2 diabetes mellitus, long-standing hypertension, and heart failure with reduced ejection fraction (HFrEF, EF 30%) presents with worsening shortness of breath, orthopnea, and bilateral pedal edema. On examination, he has elevated jugular venous pressure, bilateral basal crackles, and pitting edema up to the knees. Over the past 72 hours, his urine output has declined. His serum creatinine has increased from a baseline of 1.1 mg/dL to 2.3 mg/dL. Serum potassium is 4.9 mEq/L, sodium 132 mEq/L, and NT-proBNP is elevated. He is currently on: • Torsemide 10 mg once daily • Sacubitril/valsartan • Metoprolol succinate • Spironolactone 25 mg daily • Empagliflozin Chest X-ray shows pulmonary congestion. Blood pressure is 104/68 mmHg. **Case 2** A 21-year-old woman is admitted with generalized swelling and biopsy-proven minimal change disease (MCD). She presented with nephrotic syndrome characterized by: • Proteinuria 8 g/day • Serum albumin 1.6 g/dL • Total cholesterol 320 mg/dL • Serum creatinine 0.9 mg/dL at admission Two days after the renal biopsy, she develops worsening edema and reduction in urine output to 400 mL/day. She remains normotensive. There is no gross hematuria. Color Doppler ultrasound of the renal vessels is normal, with no evidence of renal vein thrombosis or arterial compromise. **Current medications include:** • Prednisolone (initiated after biopsy) • Pantoprazole • No diuretics yet She has tense ascites, periorbital edema, bilateral pleural effusions on ultrasound, and difficulty ambulating due to anasarca.	**Case 1** How would you escalate diuretic therapy in this setting? • Oral dose escalation vs. intravenous loop diuretics • Bolus vs. continuous infusion • Dose conversion strategy How would you assess for diuretic resistance? When would you introduce sequential nephron blockade (e.g., thiazide-type diuretic such as metolazone)? What parameters will you monitor to assess response? • Urine output (target per hour/day) • Daily weight • Net fluid balance • Spot urine sodium • Renal function and electrolytes How would you decide whether to increase or decrease the diuretic dose? At what point would you consider ultrafiltration? How would you differentiate cardiorenal syndrome from over-diuresis? **Case 2** • What are the possible causes of acute oliguria in this setting? • How will you assess intravascular volume status in nephrotic syndrome? • How will you assess intravascular volume status in nephrotic syndrome? • How will you assess intravascular volume status in nephrotic syndrome? • What complications should you watch for during aggressive diuresis? • Would you initiate anticoagulation prophylaxis in this patient? If yes, what are your criteria? • How will you monitor response to therapy?
12:10-12:40 PM	Vaccine in kidney diseases	To discuss the vaccine schedule in patient with kidney disease with special focus on Herpes Zoster vaccination and PCV20
12:45 PM	Valedictory function, Lunch and Departure	