

DIABETES UPDATE

DREAM2026

Diabetes Research, Education, Advocacy and Management

Diabetes Update

Third edition of DREAM. Conference record and key points from every session, written from the speakers' own slides

Sunday, 20 September 2026

API Hall, Oasis Plaza, Abids, Hyderabad

ORGANISED BY



DAYS

Diabetes and You Society
SINCE 1999

IN ASSOCIATION WITH



Association of Physicians of
India, Telangana State Chapter

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Dr Ch Vasanth Kumar MD FACP

Founder and President, DAYS

Organising Chairman, DREAM 2026

It gives me great happiness to present this Diabetes Update of DREAM 2026, the third edition of our conference.

When I founded DAYS in 1999, our aim was to reach children with diabetes whose families did not know how to manage it. DREAM grew out of the same belief: that good diabetes care depends on doctors who are up to date and confident. This year's programme covered thyroid disease, adult vaccination, newer insulins and incretins, infections, the skin, the kidney and the diabetic foot, along with the DREAM Oration, a debate and a panel discussion.

DAYS has always worked closest to children with type 1 diabetes and their families, many of whom cannot afford the cost of lifelong insulin and monitoring. Alongside free check-ups and counselling for these children, the society works to spread awareness of healthy eating, early detection and prevention, because the rise in diabetes around us will be slowed by awareness long before it is slowed by medicines.

I thank every member of our faculty for sharing their time and knowledge so generously, and the Association of Physicians of India, Telangana State Chapter, for standing with us. Most of all, I thank the delegates. Your questions and your presence are what make this meeting worth holding.

This book carries the key points from each session, so that the learning of the day stays with you in your clinic. I hope you keep it close, and I look forward to seeing you at the next DREAM.

A handwritten signature in black ink, appearing to read 'Ch Vasanth Kumar'.

Dr Ch Vasanth Kumar MD FACP

Founder and President, DAYS

Organising Chairman, DREAM 2026



Dr Srirang Abkari MD

Scientific Chairman, DREAM 2026

Consultant Physician, Udai Omni Hospital

A good scientific programme should answer one question: what does a practising doctor need to know on Monday morning?

With that in mind, we chose topics that come up in everyday clinics: subclinical hypothyroidism, adult vaccination, secondary diabetes, infections, skin signs and the prevention of diabetic foot complications. Alongside these, we gave time to the newer therapies, insulin icodec, semaglutide, GLP-1 receptor agonists and finerenone, so that delegates could weigh the evidence for themselves. The DREAM Oration, the debate on conventional and newer drugs, and the panel discussion made room for different points of view.

I am grateful to each speaker for the care they put into their talks, and to the chairpersons who kept the sessions on time and the discussions focused. The session notes in this souvenir are written from the speakers' own slides. I hope you find them useful for revision and in your daily practice.

A handwritten signature in blue ink, appearing to be 'S. Abkari'.

Dr Srirang Abkari MD

Scientific Chairman, DREAM 2026

Consultant Physician, Udai Omni Hospital



Dr Naval Chandra MBBS, MD, FICP, FIACM

Chairman, API Telangana State Chapter

Professor and Unit Head, Department of General Medicine, NIMS

Chairman Elect, RSSDI Telangana

On behalf of the Association of Physicians of India, Telangana State Chapter, I congratulate the Diabetes and You Society on another successful DREAM.

Diabetes now touches almost every patient a physician sees, whether they come to us with fever, breathlessness, a wound or a kidney problem. Meetings like DREAM keep us current, and they do it in a way that is practical and close to our daily work. The accreditation of the programme for 2 CPD credit hours by the Telangana Medical Council reflects its standard.

It was a privilege for API Telangana State Chapter to be associated with this conference, and a personal pleasure to speak on infections in diabetes. I thank Dr Ch Vasanth Kumar and the organising team for their hard work, and every delegate for their active participation.

I am sure this book will be a useful reference, and I wish DREAM many more successful editions.

A handwritten signature in blue ink, appearing to read 'Naval Chandra'.

Dr Naval Chandra MBBS, MD, FICP, FIACM

Chairman, API Telangana State Chapter

Professor and Unit Head, Department of General Medicine, NIMS

Chairman Elect, RSSDI Telangana

Organising team



ORGANISING CHAIRMAN

Dr Ch Vasanth Kumar
MD FACP

Founder and President, DAYS



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Dr Srirang Abkari MD

Consultant Physician, Udai
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Dr Khizer Hussain
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About DAYS. The Diabetes and You Society was founded in 1999 by Dr Ch Vasanth Kumar, with the aim of reaching children with diabetes from families who did not know how to manage it. DREAM, its annual conference for doctors, brings the same purpose to clinical practice.

This book is published by DAYS in association with the Association of Physicians of India, Telangana State Chapter. The session notes were prepared by Beta Cell Academy from the speakers' slides. September 2026.

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The programme

10:00 AM	Approach to Subclinical Hypothyroidism Dr Mohd Riyaz Chairpersons: Dr MN Shyam Sunder, Dr Srirang Abkari
10:20 AM	Cultivating a Preventive Mindset for Vaccine Preventable Disease in Adults of India Dr Udai Lal Chairpersons: Dr Subba Laxmi, Dr Sonika Reddy
10:40 AM	Protein Engineering for Reduced Insulin Burden: The Advent of Icodec Dr J Sreekanth Chairpersons: Dr Y Ganesh, Dr Pratibha Lakshmi
11:00 AM	From Hunger Signals to Health Outcomes: The Innovator Semaglutide Journey Dr Faraz Farista Chairpersons: Dr Ganga Kiran, Dr Mohsin Aslam
11:20 AM	When Diabetes is a Symptom: Unmasking Secondary Diabetes Dr Beatrice Anne Chairpersons: Dr B Seetharam, Dr Khizer Hussain Junaidy
11:40 AM	Infections in Diabetes Dr Naval Chandra Chairpersons: Dr A Vinaya Sekhar, Dr K Shankar
12:00 Noon	DREAM Oration: The Diabetes We Created, Can We Reverse the Epidemic? ORATION Dr M Raja Rao Chairpersons: Dr P Raghu Ramulu, Dr Mahesh Marda
12:30 PM	INAUGURAL FUNCTION
1:00 PM	LUNCH

The programme, continued

- 2:00 PM****Skin as a Clinical Dashboard for Diabetes**
Dr Sindhu Joshi
Chairpersons: Dr Swarna Deepak, Dr Sree Mukesh
- 2:20 PM****GLP-1RA for Physicians: Challenges and Solutions** **KEYNOTE**
Dr Manisha Sahay
Chairpersons: Dr M Surendra, Dr Y S N Raju
- 2:50 PM****Finerenone: A Breakthrough in Heart and Kidney Protection for CKD and T2D Patients**
Dr A Akshay Subramanian
Chairpersons: Dr Jayesh Shah, Dr Brij Mohan Subedhar
- 3:10 PM****Panel Discussion** **PANEL**
Dr Girish Narayan, Dr Sanjeev Singh Yadav, Dr K V Sree Lakshmi
Moderator: Dr Srirang Abkari
- 4:00 PM****Debate: Conventional vs Newer Drugs in Diabetes Management** **DEBATE**
Dr Yousuf Khan Zeeshan, Dr Pradnya Kamble
- 4:20 PM****Preventing and Managing Diabetic Foot Complications**
Dr Sujeeth Kumar Bashetty
Chairpersons: Dr Venkat Nani, Dr Madhu Sudhan Reddy
- 4:40 PM****Re-imagining Diagnostics for Clinics** **INDUSTRY**
Mr Meghnath Goud, Orange Health Labs
Chairpersons: Dr Khizer Hussain Junaidy
- 5:00 PM****The DREAM Quiz: 30 questions on the day's talks, played live on Kahoot**
QUIZ
Quiz master: Dr Khizer Hussain Junaidy

PRIZE DISTRIBUTION AND CLOSING REMARKS

Faculty

SPEAKERS AND PANELLISTS

Dr A Akshay Subramanian

Dr Beatrice Anne

Dr Faraz Farista

Dr Girish Narayan

Dr J Sreekanth

Dr K V Sree Lakshmi

Dr M Raja Rao

Dr Manisha Sahay

Dr Mohd Riyaz

Mr Meghnath Goud

Dr Naval Chandra

Orange Health Labs

Dr Pradnya Kamble

Dr Sanjeev Singh Yadav

Dr Sindhu Joshi

Dr Sujeeth Kumar Bashetty

Dr Udai Lal

Dr Yousuf Khan Zeeshan

CHAIRPERSONS AND MODERATOR

Dr A Vinaya Sekhar

Dr B Seetharam

Dr Brij Mohan Subedhar

Dr Ganga Kiran

Dr Jayesh Shah

Dr K Shankar

Dr Khizer Hussain Junaidy

Dr M Surendra

Dr Madhu Sudhan Reddy

Dr Mahesh Marda

Dr MN Shyam Sunder

Dr Mohsin Aslam

Dr P Raghu Ramulu

Dr Pratibha Lakshmi

Dr Sonika Reddy

Dr Sree Mukesh

Dr Srirang Abkari

Dr Subba Laxmi

Dr Swarna Deepak

Dr Venkat Nani

Dr Y Ganesh

Dr Y S N Raju

Names are printed as they appeared in the conference programme.

What was taught at DREAM 2026

Key points from each session, written from the speakers' own slides, with one practical line to take back to the clinic. These notes are a summary for revision and are not a substitute for the slides or for current guidelines.

10:00 AM to 10:20 AM

Approach to Subclinical Hypothyroidism

Dr Mohd Riyaz



A practical review of how to diagnose subclinical hypothyroidism, who to treat, what the trials show, and where treatment may do harm.

- Subclinical hypothyroidism means a raised TSH with a normal free T4. Small changes in T4 cause much larger changes in TSH, so TSH is the sensitive marker of the axis.
- It is common: 3 to 15% of the general population, 18 to 20% in older adults, commoner in women and in people with type 2 diabetes. Other risk factors are family history of thyroid disease, autoimmune disease and obesity (TSH falls with weight loss). Many cases resolve on their own: 46% of older patients after 4 years, and 52% of those with TSH below 10 mIU/L after 32 months.
- Confirm before you treat. Repeat the TSH in 3 to 6 months, check TPO antibodies (positive means autoimmune cause and a 2-fold higher risk of progression), and rule out non-thyroid causes such as ageing, non-thyroidal illness, chronic renal failure, adrenal insufficiency, assay interference, and drugs like amiodarone, lithium and checkpoint inhibitors.
- Treat when TSH is above 10 mIU/L, when hypothyroid symptoms are present, when TPO antibodies are positive, or in women of reproductive age, as per ATA and AACE. Levothyroxine start dose is 25 to 75 mcg with no cardiovascular disease, 25 mcg if there is cardiac disease, and case by case in those aged 70 and above: start low, go slow.
- Treatment in older people does not improve symptoms. The TRUST study (2017) randomised 737 adults aged 65 and above to levothyroxine 50 mcg daily or placebo for 1 year and found no difference in hypothyroid symptoms or tiredness scores. A 2017 Cochrane analysis of 350 patients in 12 trials (6 to 14 months) found no gain in symptoms, mood or quality of life, though lipid profile and some echocardiographic parameters improved. A randomised trial in 94 community-dwelling people aged 65 and above showed no cognitive benefit.
- Treatment can carry risk, and higher TSH also tracks with cardiovascular risk. In the Grossman et al case-control study of 1,558 people with subclinical hypothyroidism and TSH below 10 mIU/L, levothyroxine was linked to increased mortality, with advanced age, senile dementia, cerebrovascular disease, congestive heart failure and chronic renal failure as associated factors.

- Rodondi et al (about 55,000 patients) showed TSH 10.0 to 19.9 mIU/L carried a coronary artery disease hazard ratio of 1.86 and cardiovascular mortality hazard ratio of 1.58, and TSH of 7.0 mIU/L or more was linked to congestive heart failure in 2,730 people aged 70 to 79 followed for 4 years. However, Gussekloo et al found higher TSH was linked to lower mortality in those aged 85 and above.
- Pregnancy is a separate situation. Different TSH thresholds apply, and mild thyroid dysfunction should be treated, especially when TPO antibody positive, because of the higher risk of adverse pregnancy and fetal outcomes (Casey BM et al, NEJM 2017).

IN CLINIC

Repeat the TSH in 3 to 6 months with TPO antibodies before starting levothyroxine, and avoid treating older patients with TSH below 10 mIU/L.

10:20 AM to 10:40 AM

Cultivating a Preventive Mindset for Vaccine Preventable Disease in Adults of India

Dr Udai Lal



A call to make adult vaccination routine in Indian practice, covering the life-course schedule, PCV20, recombinant zoster vaccine, dengue vaccine and clinic workflow.

- Adults need vaccines because immunity wanes. Immune function declines steadily after 50 (immunosenescence), chronic disease such as diabetes, COPD and heart disease raises the risk of severe outcomes, and older adults will form about 20% of India's population by 2050.
- The main adult vaccine-preventable diseases are pneumococcal disease, influenza, herpes zoster, hepatitis A and B, COVID-19, tetanus-diphtheria-pertussis, HPV, and travel or exposure-based vaccines such as typhoid and dengue.
- The API 2026 update uses a life-course model: 18 to 26 years HPV, hepatitis B, Tdap and catch-up MMR or varicella; 27 to 49 years annual influenza, hepatitis B, Tdap booster and risk-based vaccines; 50 to 64 years zoster (RZV), pneumococcal (PCV20) and influenza; 65 and above pneumococcal, zoster, influenza and RSV.
- Key schedules to remember: influenza (QIV) 1 dose every year; PCV20 a single dose for those 50 and above and at-risk adults 19 to 49; RZV 2 doses 2 to 6 months apart for all adults 50 and above and from 19 years if immunocompromised; hepatitis B 3 doses at 0, 1 and 6 months; Tdap once, then Td every 10 years. Vaccines can be given together at one visit at different anatomical sites, for example RZV with influenza, pneumococcal or Tdap, and PCV20 with influenza and COVID-19.
- PCV20 is a 20-valent conjugate vaccine using a CRM197 carrier, giving about 10% more serotype coverage than PCV13 in adults. It is a single 0.5 mL intramuscular dose in the deltoid, removes the need for PCV13 then PPSV23 sequencing, and is endorsed as a preferred option in API 2026. If PPSV23 is also planned, give PCV20 first.

- Shingrix (RZV) is a non-live recombinant vaccine (glycoprotein E with AS01B adjuvant), over 90% effective against shingles in adults 50 and above and also protecting against post-herpetic neuralgia. In ZOE-50 efficacy was 97.2% in those 50 and above, and pooled ZOE-50/70 showed 91.3% in those 70 and above and 91.2% efficacy against PHN in those 50 and above. ZOSTER-049 showed 89% efficacy up to year 10. This matters because over 90% of Indians carry latent VZV by age 40, ICMR data show about 3.3 cases per 1,000 people each year, and diabetes raises herpes zoster risk by about 38%.
- India approved its first dengue vaccine in July 2026: Qdenga (TAK-003), a live attenuated tetravalent vaccine for ages 4 to 60 years, 2 subcutaneous doses 3 months apart, with no pre-vaccination serostatus testing needed. It is supported by 7-year TIDES Phase 3 data plus a local India Phase 3 study, and it is an adjunct to, not a replacement for, vector control.
- The physician is the main driver of uptake. Use the 5 A's at every visit: Ask about vaccine history, Assess age band and risk conditions, Advise clearly on due vaccines, Administer the same day where possible, and Arrange the next dose with a reminder.

IN CLINIC

Check vaccination history at every adult visit, and offer PCV20 and 2-dose RZV to every patient aged 50 and above and every diabetic patient.

10:40 AM to 11:00 AM

Protein Engineering for Reduced Insulin Burden: The Advent of Icodec

Dr J Sreekanth



How insulin icodec was engineered for once-weekly dosing, what the ONWARDS trials showed, and how to start and titrate it in type 2 diabetes.

- The problem is poor control and poor uptake of insulin. India had 101 million people with diabetes in 2023, about 4 in 5 (83.7%) have an HbA1c of 7% or more, and only about 6 million are on insulin.
- Icodec is engineered for a long half-life: three amino acid substitutions, removal of the terminal threonine, and a C20 icosane fatty diacid side chain with an OEG spacer. These give low insulin receptor binding affinity (slow receptor-mediated clearance), strong reversible albumin binding forming an inactive depot, better stability against enzymatic degradation, and high solubility allowing a U700 formulation.
- The U700 strength is 7 times that of standard U100 insulin, so the injection volume stays the same as a once-daily insulin. Dosing falls from at least 365 injections a year to 52.
- Pharmacokinetics support weekly dosing: geometric mean half-life is 196 hours, median tmax is 16 hours, and the glucose-lowering effect is distributed close to evenly across all 7 days of the dosing interval.

- ONWARDS 1 (984 insulin-naive people with type 2 diabetes, 78 weeks, icodec versus glargine U100) showed both non-inferiority and superiority for HbA1c, with a fall of 1.55% versus 1.35%, estimated treatment difference minus 0.19% (95% CI minus 0.36 to minus 0.03, $p=0.02$). Time in range at weeks 48 to 52 was also better, 71.9 versus 66.9% (ETD 4.27, $p=0.0004$), and more patients reached HbA1c below 7% (odds ratio 1.63, $p=0.0005$).
- Hypoglycaemia was low. Overall rates of combined clinically significant or severe hypoglycaemia were under 1 event per patient-year of exposure, with no significant difference between icodec and glargine U100.
- ONWARDS 10 (412 people on basal insulin switched to once-weekly icodec or glargine U100 for 26 weeks) confirmed non-inferiority with a statistically greater HbA1c reduction: ETD minus 0.21 (95% CI minus 0.36 to minus 0.07, $p<0.0001$), end of treatment HbA1c 7.3% versus 7.4%, and again under 1 event per patient-year of clinically significant or severe hypoglycaemia.
- Dosing is simple. Start insulin-naive patients at 70 units weekly, which corresponds to the ADA-recommended 10 units of a once-daily basal insulin. When switching from a daily basal insulin, multiply the daily dose by 7 (for example 20 units daily becomes 140 units weekly). Titrate weekly on three pre-breakfast SMBG values taken 2 days before and on the day of titration: plus 20 units per week if the mean is above 130 mg/dL, no change at 80 to 130 mg/dL, and minus 20 units per week if the lowest value is below 80 mg/dL.

IN CLINIC

Consider once-weekly icodec for patients struggling with daily basal injections: start 70 units weekly if insulin-naive, or multiply the current daily basal dose by 7.

11:00 AM to 11:20 AM

From Hunger Signals to Health Outcomes: The Innovator Semaglutide Journey

Dr Faraz Farista



A review of once-weekly semaglutide 2.4 mg for weight management, covering the STEP trial programme, Asian data, safety, dosing and approved Indian indications.

- Obesity is already a very large problem in India: 351 million adults live with abdominal obesity, 254 million adults with generalised obesity and 26 million children with overweight or obesity.
- Semaglutide is a GLP-1 receptor agonist with 94% homology to human GLP-1 and a half-life of about one week, which allows once-weekly subcutaneous dosing. It acts on brain areas controlling both homeostatic hunger (satiety) and hedonic drive (food cravings).
- Across the STEP phase 3 programme, about 9 in 10 participants on semaglutide 2.4 mg lost at least 5% of body weight, about 3 in 4 lost at least 10%, about 1 in 2 lost at least 15% and about 1 in 3 lost at least 20%. Weight loss began as early as 4 weeks and was sustained over 4 years.
- In adolescents aged 12 years and above (STEP TEENS), 40.5% achieved a BMI reduction of at least 20% versus 3.2% on placebo, with 75.6% achieving at least 5% reduction. CDSCO approved this indication in India on 5 May 2026 for adolescents 12 years and above with obesity and body weight above 60 kg.

- In East Asians (STEP 6), semaglutide 2.4 mg reduced abdominal visceral fat area by 40% versus 6.9% with placebo (ETD -33.2 percentage points, 95% CI -42.1 to -24.2, $p < 0.0001$). In STEP 12 DXA analysis at week 44, 75.3% of weight lost was fat mass and 24.7% was lean mass.
- Dose titration is stepwise and 4-weekly: 0.25 mg for weeks 1-4, 0.5 mg for weeks 5-8, 1.0 mg for weeks 9-12, 1.7 mg for weeks 13-16, then 2.4 mg from week 17 onwards. Titration can be delayed if gastrointestinal symptoms are significant.
- Safety in the STEP trials was in line with the GLP-1 RA class. Gastrointestinal adverse events were commonest, discontinuation was 4.3%, pancreatitis was 0.2% versus under 0.1%, cholelithiasis 1.6% versus 1.1%, and hypoglycaemia in STEP 2 was 6.2% versus 2.5%. No increased risk of malignancy or cardiovascular events was reported.
- Approved Indian indications listed were weight management in adults (BMI 30 kg/m² and above, or 27 kg/m² and above with a weight-related comorbidity), weight management in adolescents 12 years and above, reduction of major adverse cardiovascular events in adults with obesity or overweight and established cardiovascular disease, and non-cirrhotic MASH with F2 to F3 fibrosis.

IN CLINIC

Titrate semaglutide 2.4 mg in 4-weekly steps from 0.25 mg, delaying a step if gastrointestinal symptoms are significant, and use it alongside reduced-calorie diet and increased physical activity.

11:20 AM to 11:40 AM

When Diabetes is a Symptom: Unmasking Secondary Diabetes

Dr Beatrice Anne



A case-based talk on recognising secondary diabetes from endocrine, fat-distribution and drug-related causes, where treating the underlying disease improves or resolves the hyperglycaemia.

- Suspect Cushing syndrome in diabetes when discriminatory features are present: proximal myopathy, easy bruising, wide violaceous striae, facial plethora, unexplained hypokalaemia, rapidly progressive diabetes, early-onset hypertension or osteoporotic fractures. The case was a 55-year-old woman with HbA1c 10.2%, potassium 2.6 meq/L, overnight DST cortisol 40 micrograms/dL, ACTH 140 pg/mL and a 3 × 2 × 2 cm pituitary macroadenoma.
- The Cushing work-up follows a fixed sequence: screen with late-night salivary cortisol, overnight DST or 24-hour urinary free cortisol, confirm hypercortisolism, determine ACTH dependence, localise, then treat. Obesity, poorly controlled diabetes, depression and alcohol excess can all raise cortisol mildly, so true hypercortisolism needs more than one abnormal screening test. Transsphenoidal surgery is first-line, with remission rates of 70-90% for microadenomas, and insulin or oral agent doses must be reassessed frequently afterwards to avoid hypoglycaemia.
- Acromegaly can present as new diabetes or even DKA. A 37-year-old man with DKA, BP 180/110 mmHg, increasing shoe and ring size and coarse facial features had IGF-1 836 ng/mL (normal 135-220) and GH 34.4 ng/mL after 75 g OGTT. Diabetes occurs in up to 30-40% of patients with acromegaly, and colonoscopy is recommended at diagnosis.

- Screen for pheochromocytoma with plasma free or 24-hour urine metanephrines when a hypertensive patient has new or worsening diabetes, paroxysmal symptoms or unexplained weight loss. In three cases here HbA1c fell from 10.5% to 6.6%, 12.4% to 6.8% and 7.8% to 5.0% after adrenalectomy, with two coming off all diabetes medication. Impaired glucose tolerance is reported in about one-third of patients with pheochromocytoma and up to 40% of tumours are hereditary.
- Before pheochromocytoma surgery, alpha-blockade with phenoxybenzamine or doxazosin for 10-14 days comes first, then volume expansion, then beta-blockade only after alpha-blockade. Anticipate hypoglycaemia after resection and down-titrate insulin or oral agents urgently.
- Resistant hypertension with spontaneous or diuretic-induced hypokalaemia is a mandated trigger to screen for primary aldosteronism using the aldosterone-to-renin ratio. About 35-40% are unilateral adenomas treated by adrenalectomy, and about 60% are bilateral hyperplasia treated with spironolactone or eplerenone. In the case presented, fasting glucose normalised after adrenalectomy with no glucose-lowering therapy needed at 6 months.
- Think lipodystrophy in a young patient with diabetes out of proportion to BMI, very high insulin requirement, regional fat loss, acanthosis nigricans, severe hypertriglyceridaemia and fatty liver. The 29-year-old case had HbA1c 10-12% on U-500 insulin above 3 units/kg/day, triglycerides 890 mg/dL and low leptin, and needs metreleptin plus high-dose insulin, metformin and triglyceride-lowering therapy.
- In a patient on long-term antiretroviral therapy, fat redistribution with dysglycaemia and dyslipidaemia is HIV lipodystrophy, not ordinary type 2 diabetes. Stavudine and zidovudine cause mitochondrial toxicity and lipoatrophy while protease inhibitors drive insulin resistance; consider a regimen switch with the HIV physician, keep metformin first-line, and never stop effective ART to treat the metabolic syndrome.

IN CLINIC

Ask five questions before calling it type 2 diabetes: is the phenotype typical, is there a clue to another disease, a diabetogenic drug, appropriate insulin secretion, a typical course.

11:40 AM to 12:00 Noon

Infections in Diabetes

Dr Naval Chandra



A review of why people with diabetes get more infections, the main clinical syndromes and their management, plus glycaemic targets, stewardship and adult vaccination.

- Infections account for about 12% of total mortality in diabetes and often present atypically because of neuropathy and vascular disease. Risk of infection-related hospitalisation is 2.1 times higher, odds of osteomyelitis 4.4 times higher and sepsis mortality 1.8 times higher.
- Hyperglycaemia impairs neutrophil chemotaxis, adherence and phagocytosis, reduces intracellular ROS production, glycates complement C3 and C4, and suppresses IL-1, IL-6 and TNF-alpha. Angiopathy limits delivery of immune cells and drugs, and neuropathy adds trauma and autonomic stasis.

- Urinary tract infection is the commonest site of infection in diabetes, with asymptomatic bacteriuria in 26% of women with diabetes versus 6% of controls; diabetes alone is not an indication to screen or treat it, only pregnancy or a planned urological procedure is. Fever persisting beyond 48-72 hours mandates evaluation for obstruction, perinephric abscess or emphysematous pyelonephritis. Emphysematous pyelonephritis, 90% of which occurs in diabetes, is diagnosed on non-contrast CT and managed with early percutaneous drainage plus broad-spectrum antibiotics, reserving nephrectomy for failure.
- In diabetic foot infection, grade severity by IWGDF/IDSA classification, document perfusion with ankle-brachial index, test protective sensation with monofilament, and treat with debridement and off-loading alongside culture-directed antibiotics. Wagner grades run from 0 (at-risk foot) to 5 (extensive gangrene needing major amputation). Suspect osteomyelitis in ulcers larger than 2 cm or a positive probe-to-bone test (89% positive predictive value); MRI is the imaging gold standard, bone biopsy beats swab, and antibiotics run 6-12 weeks.
- People with diabetes are three times more likely to develop active pulmonary tuberculosis, with more lower-lobe cavitation, higher treatment failure and relapse, and MDR-TB association. Rifampicin induces CYP450 and lowers levels of sulfonylureas, pioglitazone and meglitinides, so insulin is preferred during ATT, and pyridoxine should be co-prescribed as isoniazid neuropathy mimics diabetic neuropathy.
- In sepsis, target blood glucose 140-180 mg/dL with IV insulin infusion and avoid intensive control below 110 mg/dL because of hypoglycaemia and increased mortality (NICE-SUGAR). Risk of sepsis is 60% higher in diabetes, neuropathy can mask tachycardia, and SGLT-2 inhibitors should be withheld during acute illness to prevent euglycaemic DKA.
- Mucormycosis is strongly linked to DKA and high ferritin, with black eschar on the palate or nasal turbinate as the hallmark and mortality above 70% with CNS involvement. Treatment is urgent surgical debridement plus high-dose liposomal amphotericin B, stepping down to posaconazole or isavuconazole, along with reversal of DKA and stopping unnecessary steroids.
- In the NIMS cohort of 115 hospitalised patients with diabetes, infections accounted for 74.8% of admissions and 84.4% were community-acquired. Pulmonary infection was commonest (29.1%) followed by UTI (26.7%), E. coli was the leading pathogen, and patients with sepsis or MODS had a mean HbA1c of 11.3% versus 8.4% in localised infection, with more than 95% of sepsis patients having HbA1c above 7%.

IN CLINIC

Take deep tissue, blood and urine cultures before starting antibiotics, arrange early source control, and keep inpatient glucose at 140-180 mg/dL while checking adult vaccination status at every review.

12:00 Noon to 12:30 PM

ORATION

DREAM Oration: The Diabetes We Created, Can We Reverse the Epidemic?

Dr M Raja Rao



An oration arguing that type 2 diabetes begins years before diagnosis, and that prevention during prediabetes and population metabolic health can change the epidemic's course.

- Diabetes does not begin on the day the laboratory report crosses the cut-off. The biological process may start 10 to 15 years earlier, so by the time we diagnose, we may be entering the game very late.
- The sequence is normal glucose, then insulin resistance, then compensatory hyperinsulinaemia, then progressive metabolic dysfunction, then prediabetes, then type 2 diabetes. Type 2 diabetes is insulin resistance plus progressive beta-cell failure.
- Genes alone cannot explain the rise. South Asians are susceptible to metabolic disease, but our genes did not change in a few decades, our environment did: screen-based childhood, artificial light, sleep deprivation, chronic stress, desk jobs and processed food.
- The ICMR-INDIAB national study of more than 113,000 individuals across 31 states and Union Territories estimated diabetes prevalence at 11.4% and prediabetes at 15.3% among adults. This is a population-level problem, not a clinic problem.
- Thirty minutes of morning walking does not cancel a whole day of sitting. A patient who sits eight hours at a desk, drives two hours and watches television three hours is still sedentary, so reduce prolonged sitting, use stairs and move after meals.
- Sleep and stress are neglected metabolic factors. Sleep deprivation and circadian disruption affect appetite, insulin sensitivity, hormones and body weight, and chronic stress drives poor sleep, emotional eating and reduced activity.
- Treat prediabetes as a window of opportunity, not a waiting room. Identify high-risk people early, assess family history, BMI, waist circumference, blood pressure, glucose, lipids and activity, and address cardiovascular risk alongside glucose.
- Prevention starts before birth. Maternal obesity, gestational diabetes and poor maternal nutrition influence the child's future health, so healthcare systems need three stages: diabetes care, diabetes prevention, and population metabolic health.

IN CLINIC

Tell prediabetic patients this is the stage where diabetes can still be prevented, rather than saying their sugar is only slightly high, and act on it.

2:00 PM to 2:20 PM

Skin as a Clinical Dashboard for Diabetes

Dr Sindhu Joshi



A practical review of skin signs in diabetes, showing how they flag insulin resistance, poor control, microvascular disease, infection risk and drug side effects.

- Skin involvement occurs in roughly 30 to 70% of people with diabetes and often precedes or heralds the diagnosis. The skin exam can answer five questions: insulin resistance, microvascular disease, foot risk from neuropathy, impaired immunity and wound healing, and treatment-related skin problems.
- Acanthosis nigricans, velvety hyperpigmentation of the neck, axillae, groin and abdominal folds, is a key marker of insulin resistance, driven by hyperinsulinaemia stimulating IGF receptors on keratinocytes. It is also seen in PCOS, Cushing syndrome and malignancy.

- Diabetic dermopathy, or shin spots, is the commonest cutaneous finding, seen in up to 50% of patients. The atrophic hyperpigmented pretibial macules are benign and need only reassurance, but they are significantly associated with retinopathy, nephropathy and neuropathy, so note them in the chart.
- Diabetic thick skin with limited joint mobility starts at the distal interphalangeal joints and moves proximally, giving waxy scleroderma-like skin. The hallmark is the prayer sign: the patient cannot bring both palms and fingers fully together. It occurs in both type 1 and type 2 diabetes.
- Eruptive xanthomas are crops of yellow papules on a red base over buttocks, elbows and knees, and mark severe hypertriglyceridaemia in poorly controlled diabetes from reduced lipoprotein lipase activity. Lesions resolve on their own, so treat the underlying lipid abnormality.
- Candidal intertrigo, angular cheilitis, vulvovaginitis and balanitis are often the presenting sign of undiagnosed diabetes, and tinea pedis and onychomycosis are a portal of entry for foot infection. Necrotising otitis externa from *Pseudomonas* and mucormycosis, suspected in DKA with facial pain, black eschar, periorbital swelling or necrotic palatal lesions, are emergencies.
- Know the drug-related eruptions: DPP-4 inhibitors with bullous pemphigoid and rarely Stevens-Johnson syndrome, sulfonylureas with allergic rash and photosensitivity, SGLT-2 inhibitors with genital mycotic infections and rarely Fournier's gangrene, and GLP-1 RA with altered cutaneous sensation and alopecia.
- Insulin causes lipohypertrophy, lipoatrophy and injection site reactions, and pump and CGM adhesives increasingly cause contact dermatitis. Rotate injection and infusion sites, because lipodystrophy itself makes glucose absorption erratic.

IN CLINIC

Examine the feet and skin at every diabetic visit, including monofilament testing and a visual foot exam, and screen glucose when acanthosis nigricans or recurrent candidiasis appears.

2:20 PM to 2:50 PM

KEYNOTE

GLP-1 RA for Physicians: Challenges and Solutions

Dr Manisha Sahay



A practical guide to GLP-1 receptor agonists covering organ protection trial evidence, patient selection, dosing, side effect management, monitoring and cost in Indian practice.

- Type 2 diabetes rarely occurs alone: 50% have suboptimal glycaemic control, 60% dyslipidaemia, 32% CVD, 40% CKD and 65% MASLD. GLP-1 receptors in pancreas, brain, heart, stomach, kidney, gut and blood vessels explain benefit across all these areas.
- Metformin is not always the drug of choice. Match the drug to the dominant problem: obesity favours a GLP-1 RA, diabetic CKD follows SGLT2i then GLP-1 RA then finerenone when indicated, ASCVD favours a GLP-1 RA and/or SGLT2i with proven CV benefit, and MASLD favours GLP-1 RA, tirzepatide, SGLT2i, metformin or statin.

- Semaglutide trial results by indication: SUSTAIN gave 1.8% A1c and 7 kg weight reduction at 26 weeks, PIONEER 1.5% A1c and 5 kg at 26 weeks, STEP 17% weight reduction at 68 weeks, SELECT 20% MACE reduction over 4 years, SUSTAIN-6 26% MACE reduction over 2 years, FLOW 24% reduction in major kidney outcome over 3 years, and ESSENCE 63% MASH resolution and 37% fibrosis improvement at 72 weeks.
- CV risk reduction differs across the class: SUSTAIN-6 semaglutide SC 26%, AMPLITUDE-O efpeglenatide 27%, HARMONY albiglutide 22%, PIONEER-6 oral semaglutide 21%, LEADER liraglutide 13%, REWIND dulaglutide 12%, EXSCEL exenatide ER 9%, and ELIXA lixisenatide showed no reduction. FLOW also showed 20% lower all-cause mortality.
- Titrate slowly. Oral semaglutide goes 3 mg then 7 mg then 14 mg once daily at 4-week steps, and injectable semaglutide goes 0.25, 0.5, 1 mg once weekly for diabetes and up to 1.7 and 2.4 mg for obesity, each step 4 weeks. GI side effects occur in about 20%, are mostly mild to moderate, appear early, and are managed by slow up-titration. For a missed injectable dose, give it immediately if fewer than 5 days have passed, and if 5 days or more have passed skip it and inject the next dose on the originally scheduled day.
- Use GLP-1 RA for organ protection in type 2 diabetes, obesity, or BMI above 27 with comorbidity such as CKD, CAD or MASLD. They are not indicated in normal weight non-diabetic people and are not useful in non-albuminuric disease.
- Around surgery, individualise. Stop the drug only in patients with severe GI symptoms, those in the dose escalation phase, or those with previous aspiration. For emergency surgery, treat the patient as a full stomach and use aspiration precautions, following the American Society of Anesthesiologists guidance.
- Muscle data are reassuring. In the STEP 1 DXA substudy at week 68 lean mass fell proportionally less than fat mass, and in the SEMALEAN 12-month study fat mass fell steadily while lean mass stayed essentially stable and grip strength improved. Counsel resistance training, protein intake as per CKD stage, and monitor grip strength, with sarcopenia cut-offs below 28 kg in men and 18 kg in women.

IN CLINIC

Choose the drug by the dominant organ problem rather than reaching for metformin first, and titrate GLP-1 RA slowly over 4-week steps while monitoring grip strength and GI symptoms.

2:50 PM to 3:10 PM

Finerenone: A Breakthrough in Heart and Kidney Protection for CKD and T2D Patients

Dr A Akshay Subramanian



How finerenone blocks mineralocorticoid receptor overactivation and protects the heart and kidney in CKD with type 2 diabetes, with trial data and practical dosing.

- Stage CKD in type 2 diabetes using both eGFR and UACR, not eGFR alone. A patient with eGFR 62 ml/min/1.73 m² looks low risk, but a UACR of 350 mg/g puts him at severely increased risk of progression. ADA, KDIGO and AACE advise testing both at least annually in all patients with T2D.

- Albuminuria matters even at low levels. A UACR as low as 30 mg/g is linked to higher all-cause and CV mortality in CKD. In people with CKD and urinary albumin 300 mg/g or more, a fall of 30% or greater is recommended to slow progression.
- Finerenone works on a driver that RAS inhibitors and SGLT-2 inhibitors do not cover: mineralocorticoid receptor overactivation, which causes inflammation and fibrosis in the kidney and heart. It is a bulky, non-steroidal and selective MR antagonist, with high MR selectivity, a short half-life of 2 to 3 hours, no active metabolites and little effect on BP.
- FIDELITY, the pooled analysis of FIDELIO-DKD and FIGARO-DKD (n=13,026, CKD stage 1 to 4, median follow-up 3 years), showed reduced kidney and CV risk versus placebo. Finerenone cut the risk of ESKD by about one fifth (HR 0.80, 95% CI 0.64 to 0.99) and hospitalisation for heart failure by more than one fifth (HR 0.78, 95% CI 0.66 to 0.92). Almost all patients were already on an ACE inhibitor or ARB (99.8%).
- Benefit was consistent whether or not the patient was already on an SGLT-2 inhibitor. In the FIDELITY subgroup analysis, UACR fell by 37% with finerenone in those on a baseline SGLT-2 inhibitor and by 31% in those not on one.
- The CONFIDENCE study (n=818, phase II, published 2025) tested starting finerenone and empagliflozin 10 mg together in T2D with eGFR 30 to 90 ml/min/1.73 m² and UACR 100 to under 5000 mg/g. Combination gave an additive UACR reduction of up to 52% at day 180, a fall of over 30% was seen within 14 days, and 70% of patients reached the ADA target of more than 30% UACR reduction.
- Hyperkalaemia was manageable in CONFIDENCE. Serum potassium above 6.0 mmol/L was uncommon and was numerically lower with combination therapy than with finerenone alone. No case was serious, none led to hospitalisation or death, and discontinuation for hyperkalaemia was under 1% in all arms. Serum potassium above 4.8 mmol/L was an exclusion at entry.
- In heart failure with LVEF 40% or more, FINEARTS-HF (n=6001, median follow-up 32 months, finerenone 10, 20 or 40 mg once daily by eGFR) showed a 16% relative risk reduction in CV death plus total HF events (RR 0.84, 95% CI 0.74 to 0.95, p=0.007), with separation from placebo by day 28 and benefit in all 17 subgroups. Finerenone now has DCGI approval in India for adults with HFmrEF or HFpEF.

IN CLINIC

Check UACR along with eGFR at least yearly in every T2D patient, and add finerenone to RAS inhibitor and SGLT-2 inhibitor when albuminuria persists and potassium allows.

3:10 PM to 4:00 PM

PANEL

Panel Discussion

Dr Girish Narayan, Dr Sanjeev Singh Yadav and Dr K V Sree
Lakshmi. Moderator: Dr Srirang Abkari



4:00 PM to 4:20 PM

DEBATE

Debate: Conventional vs Newer Drugs, the case for conventional

Dr Yousuf Khan Zeeshan



The case for the older agents: long evidence, hard outcome data and a price most Indian patients can actually keep paying.

- "Conventional" was defined as what is tried, tested and commonly used rather than new. The argument was not that newer drugs are bad, but that the older ones already do the job for most patients.
- Each conventional oral agent has a long record behind it. Metformin dates to 1922, acarbose to 1970, repaglinide to 1983, pioglitazone to 1990, glimepiride to 1995 and sitagliptin to 1999.
- India's problem is control, not choice of molecule. 101 million adults had diabetes in 2023, expected to reach 124 million by 2030, about 1 in 10 adults. Among Indians with diabetes, about 74% do not reach an HbA1c below 7%, about 80% do not reach a BP below 130/80 mmHg, and about 83% do not reach an LDL below 70 mg/dL.
- Affordability is the binding constraint. About 62% of people with diabetes in India cannot afford the full cost of treatment. Per capita income is about Rs 2,27,000 a year, roughly Rs 18,900 a month, and only Rs 4,012 crore, 3.9% of the Rs 1,03,462 crore Union health budget for 2026-27, goes to non-communicable diseases.
- A 1% fall in HbA1c is worth a great deal on its own: 14% relative risk reduction in myocardial infarction, 12% in stroke, 16% in heart failure, 18% in cardiovascular death, 37% in retinopathy, 21% in nephropathy, 43% in neuropathy and 12% in amputation. Sulphonylureas give about 1.0 to 1.5%, GLP-1 RA about 1.0 to 1.5% and SGLT2 inhibitors about 0.7 to 1.0%.
- Sulphonylureas have outcome data, not only glucose data. UKPDS (1998) showed a 33% reduction in microvascular complications ($p=0.0004$). ADVANCE (2008), on a gliclazide-based regimen, reduced major macrovascular and microvascular events (HR 0.94, 95% CI 0.88 to 1.00) with no increase in cardiovascular mortality, and cut nephropathy by 21% (HR 0.79, 95% CI 0.66 to 0.93). CAROLINA (2019) found glimepiride and linagliptin gave similar 3-point MACE (HR 0.98, 95% CI 0.84 to 1.14) and no difference in the renal composite (HR 1.04, 95% CI 0.83 to 1.31).
- Alpha-glucosidase inhibitors remain a cheap option for post-meal control. Voglibose and acarbose each lower HbA1c by about 0.5 to 1.0%. Through the Jan Aushadhi (PMBJP) network, voglibose 0.2 mg costs about Rs 10 for ten tablets, 0.3 mg about Rs 12.75, and acarbose 50 mg about Rs 55.90.

- The monthly cost gap is the argument in one line. Sulphonylureas Rs 20 to 50, metformin Rs 30 to 60, voglibose Rs 60 to 100 and pioglitazone Rs 100 to 200, against DPP-4 inhibitors Rs 500 to 1,200, SGLT2 inhibitors Rs 800 to 1,500, insulin Rs 800 to 2,500 and GLP-1 receptor agonists Rs 1,000 to 18,000.

IN CLINIC

Weigh what the patient can afford to keep taking for years before reaching for the newest agent, because an affordable drug actually continued still delivers the 1% HbA1c fall.

4:00 PM to 4:20 PM

DEBATE

Debate: Conventional vs Newer Drugs, the case for newer modalities

Dr Pradnya Kamble



The newer-modalities side of the debate, arguing that organ protection, weight and outcomes, not HbA1c alone, should drive treatment in high-risk diabetes.

- The core argument: diabetes is a multi-organ disease, so HbA1c is necessary but not sufficient. CV and renal risk persists despite good glycaemic control, and care must move from glucose-centric to outcome-centric.
- SGLT2 inhibitors make the strongest case in CKD and heart failure. They lower HbA1c by about 0.5 to 1%, reduce weight and BP, and give cardio-renal protection (EMPA-REG, DAPA-CKD). Watch for genital infections and dehydration. Examples: empagliflozin, dapagliflozin, canagliflozin.
- GLP-1 receptor agonists lower HbA1c by about 1 to 1.5%, give clinically meaningful weight loss and appetite suppression, and reduce CV risk (LEADER, SUSTAIN-6). Main adverse effects are nausea and GI upset. Examples: liraglutide, semaglutide, dulaglutide.
- Combining an SGLT2 inhibitor with a GLP-1 RA uses complementary mechanisms, improves CV and renal outcomes and reduces insulin requirement, which makes it suited to the high-risk patient. Dual GIP/GLP-1 agonists add greater weight-loss potential and strong glycaemic efficacy in severe insulin resistance and obesity.
- The old criticism of insulin, weight gain and hypoglycaemia, is answered by newer insulins. Once-weekly basal insulin icodec (Awiqli) cuts injection burden from about 30 shots a month to 4, and insulin efsitora alfa (QWINT-1) is the other once-weekly option. Also available are faster-acting insulin aspart (Fiasp), inhaled insulin (Afrezza) and the fixed combination IcoSema, which puts insulin icodec and weekly semaglutide together.
- CGM picks up variability that HbA1c misses, reveals hypoglycaemia and post-prandial spikes, and supports personalised titration and patient engagement. Insulin pumps and artificial pancreas systems extend this to data-driven precision care.
- Use a risk-based treatment model: CKD means SGLT2 inhibitor first; ASCVD or obesity means GLP-1 RA or a dual incretin; variable glucose means CGM; a mixed phenotype means a combination strategy.
- Closing case for this side: newer modalities reduce organ failure and not only glucose, they match contemporary patient phenotypes better, and they align with current standards of care, so they should take centre stage in high-risk patients.

IN CLINIC

Choose the next drug by the patient's organ risk, CKD, ASCVD or obesity, rather than by HbA1c alone, and use CGM when glucose readings are variable.

4:20 PM to 4:40 PM

Preventing and Managing Diabetic Foot Complications

Dr Sujeeth Kumar Bashetty



A surgeon's case for the diabetic foot as a stageable, preventable condition: screen it in sixty seconds, classify it in numbers, and refer it before the window closes.

- A limb is estimated to be lost to diabetes every 20 seconds somewhere in the world, and more than 80% of those amputations begin as a foot ulcer that experts consider preventable (IDF Diabetes Atlas estimate; Armstrong DG et al., JAMA 2023).
- India carries 101 million people with diabetes (ICMR-INDIAB 2023), up to 1 in 4 of whom develop a foot ulcer in their lifetime. The speaker put annual amputations at 40,000 to 100,000, stating plainly that this is an estimate because India has no amputation registry, and that about 75% are considered preventable. The Indian pattern is distinct: late presentation, polymicrobial gram-negative infection and high multidrug resistance.
- Five-year mortality after a major diabetic amputation exceeds that of breast cancer and the pooled figure for all cancers (Armstrong DG et al., J Foot Ankle Res 2020). The argument made from this was direct: we stage cancers, so we should stage feet.
- The foot fails in three ways at once. Neuropathy removes pain, claws the toes and dries the skin. Ischaemia in diabetes is infra-popliteal, heavily calcified, silent because neuropathy masks claudication, and present in up to half of ulcers, so a normal or high ABI can mislead and toe pressure or TcPO2 should be used. Immunopathy means about half of limb-threatening infections present with no fever and no leukocytosis, so normal temperature and WBC never rule out a foot that is being lost.
- The 60-second foot exam, at every visit for every patient with diabetes: look at colour, callus, fissures, deformity and between the toes; feel skin temperature with the back of the hand, where a difference of more than 2 degrees C means inflammation, and palpate both pulses; test with a 10 g monofilament at 3 to 4 plantar sites plus a 128 Hz tuning fork at the hallux; examine the footwear, which is often the weapon; and teach daily inspection, never barefoot and no hot soaks.
- Then write the risk category in the chart, using IWGDF 2023: risk 0 reviewed yearly, risk 1 (loss of protective sensation or PAD) at 6 to 12 months, risk 2 (both, or either with deformity) at 3 to 6 months, and risk 3 (either plus prior ulcer, amputation or dialysis) at 1 to 3 months. A documented category books its own follow-up.
- A healed ulcer is remission, not cure: recurrence is 40% at 1 year and 65% at 5 years. Custom insoles and protective footwear roughly halve recurrence, but the speaker was careful to say the benefit was confined to patients who actually wore them (26% versus 48% in adherent users; Bus 2013), and he advised negotiating indoor chappals rather than lecturing, given how common barefoot walking at home and in temples is in Telangana.

- Charcot foot is the trap. A unilateral red, hot, swollen foot in a neuropathic patient is acute Charcot until proven otherwise. MRI early, because it shows stage-0 disease while the X-ray is still normal; immobilise in a total contact cast and keep it 4 to 6 weeks after the foot cools; and do not label it cellulitis, gout or DVT and send the patient home.
- Classify, do not describe. SINBAD (site, ischaemia, neuropathy, bacterial infection, area, depth, 0 to 6) needs no instruments and is the IWGDF 2023 first choice; the IDSA grade drives tonight's two decisions, oral versus IV antibiotics and home versus admission; Wifl is what the vascular team adds. A referral reading "SINBAD 4, IDSA 3, no palpable pulses" tells the surgeon exactly how fast to move.
- Three numbers to carry away. An ulcer not 50% smaller at 4 weeks despite good care is ischaemic until proven otherwise, so reassess perfusion rather than changing the dressing brand. Area reduction at 4 weeks predicts healing, so measure or photograph at every dressing change. Probe-to-bone has about 90% positive predictive value for osteomyelitis in a deep infected ulcer, and a negative probe in a low-risk clinic wound almost excludes it.
- Indian microbiology differs from the textbook. In a Mumbai tertiary centre series (Saseedharan 2018, n=261, aerobic isolates), 55% of infections were polymicrobial, 24% of *S. aureus* was MRSA, 1 in 5 *Pseudomonas* was carbapenem-resistant and 1 in 3 gram-negatives were ESBL or carbapenemase producers. So culture deep tissue after debridement rather than swabbing the surface, and note that IWGDF/IDSA 2023 allows empirical *Pseudomonas* cover in moderate to severe infection in tropical settings when it has recently been isolated from that foot.
- Antibiotics by IDSA grade: uninfected wounds get none, only wound care and offloading; mild infection gets oral amoxicillin-clavulanate or cephalexin for 1 to 2 weeks; moderate infection gets admission with piperacillin-tazobactam or ceftriaxone plus metronidazole for 2 to 4 weeks; severe infection gets resuscitation, theatre and meropenem with vancomycin or linezolid, de-escalated on cultures at 48 to 72 hours. The speaker's warning was that antibiotics buy time but do not drain pus or remove dead bone.
- Sharp debridement to bleeding tissue is the single most effective wound intervention, because it resets a wound stalled in chronic inflammation and un-roofs the pockets where pus hides. It is a programme and not one event, the surrounding callus must go too, and perfusion must be checked first, since debriding a critically ischaemic foot without a revascularisation plan only enlarges a wound that cannot heal.
- Offloading was called the most under-prescribed treatment in the field. A non-removable knee-high device, the total contact cast, is first choice and heals about 90% of plantar ulcers at 12 weeks against about 60% in removable devices, because patients wear removable walkers for only a minority of their steps. Where casting is not possible, a removable walker made non-removable with a cable tie is evidence-based and costs a few rupees.
- No blood means no healing. Below a TcPO₂ or toe pressure of 30 mmHg the priority is flow, not a better dressing. BEST-CLI (2022) favoured open bypass where a good vein is available and BASIL-2 (2023) favoured endovascular-first in infra-popliteal disease, but the practical message for physicians was that both trials show revascularisation works and the modality is the vascular team's call. What the referring doctor controls is latency: an ulcer referred at week 2 has options a week-16 referral has lost.

- Amputation done well is reconstruction, not defeat. Take the lowest level that will reliably heal and leave a walkable foot: toe or ray excision, trans-metatarsal with a healthy heel pad, below-knee for ascending sepsis or non-reconstructable ischaemia, and above-knee only as a rescue. A timely ray amputation that preserves walking is a limb-salvage success; repeated heroic attempts to save a toe often end higher.
- Organising the care beats any single innovation. Structured multidisciplinary foot teams, the surgeon or podiatrist working with the vascular surgeon and the diabetologist, are associated with roughly 50% fewer major amputations (pooled OR 0.40; reduction seen in 31 of 33 studies), and a Singapore inpatient pathway took major amputations from 18% to 5% over 17 years. The speaker noted the evidence is observational rather than randomised.
- Pick up the phone the same day if any one of these is true: an ulcer with absent pedal pulses; cellulitis beyond 2 cm or any systemic sign; gas, crepitus or foul necrosis; a probe that reaches bone or visible tendon or bone; an acute hot swollen foot; new pain in a previously painless foot; a wound not 50% smaller after 4 weeks of good care; or sudden unexplained glycaemic deterioration.

IN CLINIC

Do the 60-second foot exam at every visit, write the IWGDF risk category in the chart so it books its own follow-up, and refer the same day on any one of the red flags rather than reviewing in two weeks.

4:40 PM to 5:00 PM

INDUSTRY SESSION

Re-imagining Diagnostics for Clinics

Mr Meghnath Goud, Orange Health Labs



An industry presentation by a diagnostics company on clinic diagnostics workflow: sample collection, report turnaround and in-clinic testing models.

- The service model offers home sample collection on demand within 30 minutes, available from 6 am to 10 pm, with reports stated at 6 hours. This matters mainly for how quickly you can expect results back when you order tests from clinic.
- Two models are offered for clinics: in-clinic diagnostics, where the sample is collected at your clinic and the lab does the testing, and e-clinics, where the lab deploys phlebotomists for end-to-end diagnostics management.
- Quality and support points relevant to a clinic: 100% NABL accreditation, tele-pathology with full auditability, standardised phlebotomy, 16/7 doctor support and future order scheduling.
- A patient-facing digital record was presented, holding longitudinal trends of markers and complete health history, which can be useful when you need a patient's earlier results at follow-up.

The clinic checklist

One action from each talk, in the speakers' own words from the session notes. Tick them off, or photocopy this spread for your consulting room.



AT EVERY VISIT

- ☐ Check vaccination history at every adult visit, and offer PCV20 and 2-dose RZV to every patient aged 50 and above and every diabetic patient.
Dr Udai Lal, adult vaccination

- ☐ Examine the feet and skin at every diabetic visit, including monofilament testing and a visual foot exam, and screen glucose when acanthosis nigricans or recurrent candidiasis appears.
Dr Sindhu Joshi, skin in diabetes

- ☐ Do the 60-second foot exam at every visit, write the IWGDF risk category in the chart so it books its own follow-up, and refer the same day on any one of the red flags rather than reviewing in two weeks.
Dr Sujeeth Kumar Bashetty, diabetic foot

- ☐ Tell prediabetic patients this is the stage where diabetes can still be prevented, rather than saying their sugar is only slightly high, and act on it.
Dr M Raja Rao, DREAM Oration

BEFORE YOU DIAGNOSE

- ☐ Ask five questions before calling it type 2 diabetes: is the phenotype typical, is there a clue to another disease, a diabetogenic drug, appropriate insulin secretion, a typical course.
Dr Beatrice Anne, secondary diabetes

- ☐ Repeat the TSH in 3 to 6 months with TPO antibodies before starting levothyroxine, and avoid treating older patients with TSH below 10 mIU/L.
Dr Mohd Riyaz, subclinical hypothyroidism

Diabetic foot: refer the same day if any one is present

- An ulcer with absent pedal pulses
- Cellulitis beyond 2 cm, or any systemic sign
- Gas, crepitus or foul necrosis
- A probe that reaches bone, or visible tendon or bone
- An acute hot swollen foot
- New pain in a previously painless foot
- A wound not 50% smaller after 4 weeks of good care
- Sudden unexplained glycaemic deterioration

THE CLINIC CHECKLIST, CONTINUED

CHOOSING AND STARTING TREATMENT

- ☐ Choose the drug by the dominant organ problem rather than reaching for metformin first, and titrate GLP-1 RA slowly over 4-week steps while monitoring grip strength and GI symptoms.

Dr Manisha Sahay, GLP-1 RA

- ☐ Choose the next drug by the patient's organ risk, CKD, ASCVD or obesity, rather than by HbA1c alone, and use CGM when glucose readings are variable.

Dr Pradnya Kamble, the case for newer drugs

- ☐ Weigh what the patient can afford to keep taking for years before reaching for the newest agent, because an affordable drug actually continued still delivers the 1% HbA1c fall.

Dr Yousuf Khan Zeeshan, the case for conventional drugs

- ☐ Consider once-weekly icodec for patients struggling with daily basal injections: start 70 units weekly if insulin-naïve, or multiply the current daily basal dose by 7.

Dr J Sreekanth, insulin icodec

- ☐ Titrate semaglutide 2.4 mg in 4-weekly steps from 0.25 mg, delaying a step if gastrointestinal symptoms are significant, and use it alongside reduced-calorie diet and increased physical activity.

Dr Faraz Farista, semaglutide

KIDNEY AND INFECTION

- ☐ Check UACR along with eGFR at least yearly in every T2D patient, and add finerenone to RAS inhibitor and SGLT-2 inhibitor when albuminuria persists and potassium allows.

Dr A Akshay Subramanian, finerenone

- ☐ Take deep tissue, blood and urine cultures before starting antibiotics, arrange early source control, and keep inpatient glucose at 140-180 mg/dL while checking adult vaccination status at every review.

Dr Naval Chandra, infections in diabetes

The numbers from the day

Doses, schedules and cut-offs quoted by the speakers at DREAM 2026. Check the current prescribing information and guidelines before you act on them.

THYROID

Treat subclinical hypothyroidism	TSH above 10 mIU/L, or symptoms, TPO antibodies positive, or a woman of reproductive age
Confirm before treating	Repeat TSH in 3 to 6 months, with TPO antibodies
Levothyroxine start	25 to 75 mcg; 25 mcg if cardiac disease

ADULT VACCINES

PCV20	1 dose, 0.5 mL IM; age 50+ and at-risk adults 19 to 49
RZV (Shingrix)	2 doses, 2 to 6 months apart; age 50+, or 19+ if immunocompromised
Influenza	1 dose every year
Hepatitis B	3 doses at 0, 1 and 6 months
Tdap	Once, then Td every 10 years
Qdenga (dengue)	2 subcutaneous doses 3 months apart; age 4 to 60

INSULIN AND GLP-1 RA

Icodec, insulin-naïve	70 units once weekly
Icodec, switching	Daily basal dose x 7 (20 units daily = 140 weekly)
Semaglutide 2.4 mg (obesity)	0.25, 0.5, 1.0, 1.7, then 2.4 mg; 4 weeks per step
Oral semaglutide	3 mg, then 7 mg, then 14 mg daily; 4 weeks per step
Missed weekly GLP-1 RA dose	Fewer than 5 days late: give it straight away

KIDNEY

Screen	UACR and eGFR together, at least yearly
Albuminuria	UACR 30 mg/g or more is abnormal
Target	If UACR is 300 mg/g or more, aim for a fall of 30% or more
Finerenone start	Serum potassium 5.0 mmol/L or less

INFECTION

Sepsis glucose target	140 to 180 mg/dL; avoid below 110 mg/dL
SGLT-2 inhibitors	Withhold during acute illness

FOOT (IWGDF 2023)

Review interval	Risk 0 yearly; 1 at 6 to 12 months; 2 at 3 to 6 months; 3 at 1 to 3 months
Monofilament	10 g, at 3 to 4 plantar sites
Skin temperature	A difference over 2 degrees C means inflammation
Probe-to-bone test	Positive predictive value 89%
Healing check	Not 50% smaller at 4 weeks: treat as ischaemic until proven otherwise

The DREAM Quiz

The day closed with a live quiz of 30 questions, every one of them drawn from a talk delivered that day, played by the delegates on their phones on Kahoot.

24

delegates played

30

questions

63%

of all answers correct

27/30

top score



2

SECOND PLACE

Dr Jeet

22,759 points · 25 of 30 correct



1

FIRST PLACE

Dr Uma Mahesh

23,740 points · 27 of 30 correct



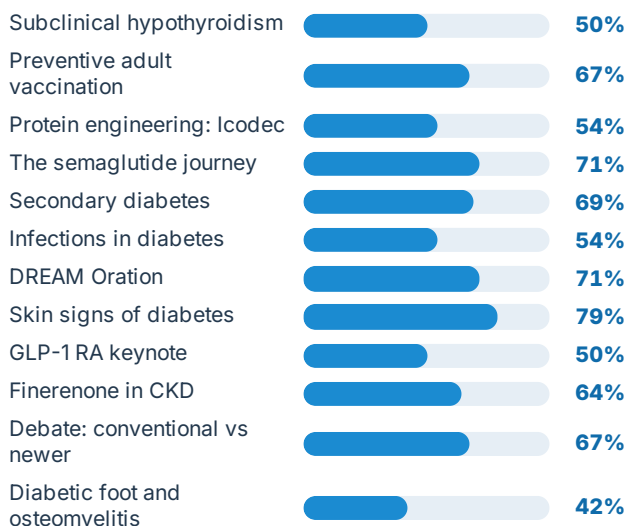
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THIRD PLACE

Dr Rafath Unnisa begum

21,235 points · 25 of 30 correct

HOW THE HALL SCORED, TALK BY TALK



Share of all answers that were correct, for the questions drawn from each talk.

THE QUESTIONS THAT STUMPED THE HALL

12%

In the TRUST study, what did levothyroxine 50 mcg do for adults aged 65 and above at 1 year?

Answer: No difference in symptoms or tiredness

12%

What share of emphysematous pyelonephritis cases occur in people with diabetes?

Answer: About 90%

42%

What is the positive predictive value of the probe-to-bone test in a diabetic foot ulcer?

Answer: 89%

Most answered correctly (92%): in the DREAM Oration, prediabetes was described as a window of opportunity, not a waiting room.

Congratulations to our three winners, and thank you to every delegate who stayed on to play. Quiz master: Dr Khizer Hussain Junaidy, Diabetologist & Bariatric Physician.

Answer key

All 30 questions from the quiz, with the correct answers. Try them yourself before you look.

1. Subclinical hypothyroidism is defined by which laboratory pattern?

Answer: **Raised TSH with a normal free T4**

2. In the TRUST study, levothyroxine 50 mcg in adults aged 65+ gave what result at 1 year?

Answer: **No difference in symptoms or tiredness**

3. Which is a clear indication to treat subclinical hypothyroidism?

Answer: **TSH above 10 mIU/L**

4. What is the Shingrix (RZV) schedule for adults aged 50 and above?

Answer: **2 doses, 2 to 6 months apart**

5. Why does the API 2026 schedule prefer PCV20 in adults?

Answer: **20 serotypes in one dose, no PCV13 then PPSV23 sequence**

6. India's first approved dengue vaccine, Qdenga (TAK-003), is given how?

Answer: **2 subcutaneous doses, 3 months apart**

7. Insulin icodec (Awiqli): what starting dose is recommended for an insulin-naïve patient?

Answer: **70 units once weekly**

8. Switching a patient from daily basal insulin to icodec: how is the weekly dose worked out?

Answer: **Multiply the daily dose by 7**

9. How is semaglutide 2.4 mg brought up to its maintenance dose?

Answer: **Stepwise, every 4 weeks, from 0.25 mg**

10. Across the STEP trials, how many adults on semaglutide 2.4 mg lost 20% or more of body weight?

Answer: **About 1 in 3**

11. In a person with diabetes, which finding points hardest to Cushing syndrome?

Answer: **Proximal myopathy with wide purple striae**

12. Before removing a pheochromocytoma, what must come first?

Answer: **Alpha blockade, then beta blockade**

13. Resistant hypertension with spontaneous hypokalaemia and new dysglycaemia. Screen for what?

Answer: **Primary aldosteronism**

14. What share of emphysematous pyelonephritis cases occur in people with diabetes?

Answer: **About 90%**

15. Surviving Sepsis: what blood glucose range is targeted in the septic patient?

Answer: **140 to 180 mg/dL**

16. Which antitubercular drug induces CYP450 and lowers oral antidiabetic drug levels?

Answer: **Rifampicin**

17. ICMR-INDIAB: what is the adult prevalence of diabetes and prediabetes in India?

Answer: **11.4% diabetes, 15.3% prediabetes**

18. In the DREAM Oration, prediabetes was described as what?

Answer: **A window of opportunity, not a waiting room**

19. Acanthosis nigricans is chiefly a clinical marker of what?

Answer: **Insulin resistance**

20. The prayer sign, failure to bring the palms fully together, is the hallmark of what?

Answer: **Diabetic thick skin with limited joint mobility**

21. Which is the most common cutaneous finding in diabetes, seen in up to half of patients?

Answer: **Diabetic dermopathy, the shin spots**

22. Which is the landmark kidney outcomes trial of semaglutide in CKD?

Answer: **FLOW**

23. A patient misses a weekly GLP-1 RA dose by fewer than 5 days. What should be done?

Answer: **Give the missed dose straight away**

24. How does finerenone differ from spironolactone and eplerenone?

Answer: **It is a bulky, non-steroidal, selective MR antagonist**

25. CONFIDENCE: starting finerenone and an SGLT2 inhibitor together cut UACR by up to how much?

Answer: **52%**

26. At what serum potassium level may finerenone be started?

Answer: **5.0 mmol/L or less**

27. In the debate on conventional drugs, which agent dates back to 1922?

Answer: **Metformin**

28. Every 1% fall in HbA1c cuts the relative risk of diabetic retinopathy by roughly how much?

Answer: **37%**

29. For the high-risk patient with CKD, which class does the newer-modalities case put first?

Answer: **An SGLT2 inhibitor**

30. In a diabetic foot ulcer, what is the positive predictive value of the probe-to-bone test?

Answer: **89%**

Moments from the day



Faculty and guests on the dais at the inaugural function



Delegates at API Hall



Felicitations of the faculty

In the hall



The panel discussion



A talk in progress



A question from the floor

The close of the day



Faculty and delegates at the close of DREAM 2026



The DREAM Quiz under way



After the closing session

Acknowledgements

DAYS thanks every speaker, panellist, chairperson and moderator who gave their time to DREAM 2026, and the delegates whose questions made the day.

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We are grateful to our sponsors, whose support made the conference possible. They are listed in alphabetical order.



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DREAM2026



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