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Journal of Diabetes Education

To Dispel Darkness Of Diabetes

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JOURNAL OF DIABETES EDUCATION

To Dispel Darkness of Diabetes

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POLYCYSTIC OVARY SYNDROME AND DIABETES MELLITUS

A METABOLIC CONTINUUM WITH LIFELONG IMPLICATIONS

Ameya Joshi*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine disorder in women of reproductive age and is now recognized as a chronic metabolic condition rather than a purely reproductive disorder. Insulin resistance, intrinsic to PCOS, provides a mechanistic bridge to dysglycemia, gestational diabetes mellitus (GDM) and Type 2 diabetes mellitus (T2DM). Women with PCOS develop glucose abnormalities at a younger age and at lower body mass indices than the general population, particularly in South Asian cohorts. This narrative review integrates current evidence on epidemiology, pathophysiology, clinical manifestations, screening strategies and management of diabetes in PCOS, with special emphasis on the role of diabetes educators.

Polycystic ovary syndrome affects 6–20% of women depending on diagnostic criteria and ethnicity (1). Once viewed largely as a gynecological condition, PCOS is increasingly understood as an early-life manifestation of metabolic disease (2). Insulin resistance, hyperandrogenism, visceral adiposity and low-grade inflammation converge to predispose affected women to long-term cardiometabolic morbidity, including Type 2 diabetes mellitus.

PCOS often presents during adolescence or early adulthood, decades before overt diabetes develops. This temporal gap offers a critical window for preventive intervention, placing diabetes educators in a central role in risk communication, lifestyle counseling and long-term follow-up.

EPIDEMIOLOGY OF DIABETES IN PCOS

Women with PCOS have a two- to five-fold increased risk of T2DM compared with age- and BMI-matched controls (3). Impaired glucose tolerance is reported in 30–40%, while overt diabetes occurs in 8–15%, often before 40 years of age (4). Gestational diabetes mellitus is two to three times more common, conferring additional intergenerational risk.

South Asian women with PCOS exhibit greater insulin resistance and dysglycemia at lower BMI thresholds, reflecting ethnic susceptibility (5). Alarmingly, up to 70% of women with PCOS remain undiagnosed, delaying preventive strategies (6).

PATHOPHYSIOLOGY: LINKING PCOS AND DIABETES

Insulin resistance is present in 60–80% of women with PCOS and almost universally in those with obesity (2). Importantly, insulin resistance in PCOS is partly intrinsic, related to post-receptor signaling defects and not solely attributable to adiposity.

Compensatory hyperinsulinemia stimulates ovarian theca cells to increase androgen production and suppresses hepatic sex hormone–binding globulin synthesis, increasing free androgen levels. Hyperandrogenism further worsens insulin resistance, creating a vicious cycle (7).

Progression to diabetes also requires beta-cell dysfunction. Evidence suggests impaired beta-cell compensation in PCOS, especially in the presence of genetic susceptibility, obesity and chronic metabolic stress (3).

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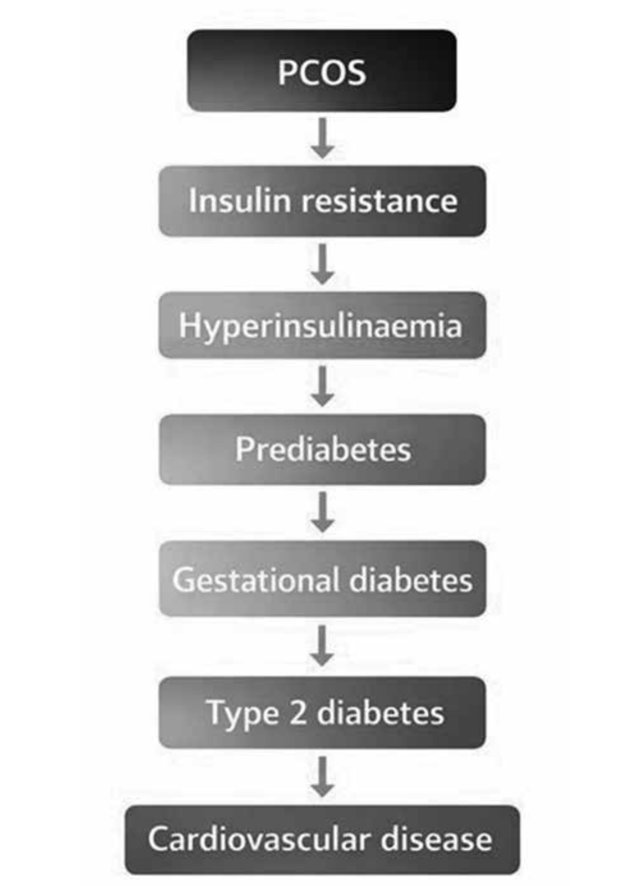
Figure 1

PCOS–Diabetes Metabolic Continuum (4)



CLINICAL SPECTRUM OF DYSGLYCEMIA

The glycemic spectrum in PCOS ranges from hyperinsulinemia and impaired fasting glucose to impaired glucose tolerance, gestational diabetes and Type 2 diabetes mellitus. Post-prandial hyperglycemia is often the earliest abnormality, making fasting glucose and HbA1c insufficient as standalone screening tools (4).

SCREENING AND DIAGNOSIS

All women with PCOS should be screened for dysglycemia regardless of BMI (8). The 75-g oral glucose tolerance test remains the preferred modality. Screening is recommended at diagnosis, every 1–3 years thereafter, preconception and early in pregnancy. Glycated hemoglobin, though useful and recommended for screening, should be done on a reliable platform. Glycated hemoglobin should not be used for screening of diabetes in anemic or pregnant females.

Table 1

DIAGNOSTIC CRITERIA FOR POLYCYSTIC OVARY SYNDROME (8)

Criteria Set	Key Features
NIH 1990	Hyperandrogenism + chronic anovulation
Rotterdam 2003	Any two: oligo/anovulation, hyperandrogenism, polycystic ovaries
AE-PCOS Society	Hyperandrogenism plus ovarian dysfunction

MANAGEMENT STRATEGIES

Lifestyle modification is first-line therapy across all phenotypes. A 5–10% weight reduction improves insulin sensitivity, androgen excess, ovulatory function and metabolic outcomes (4). Diets emphasizing on low glycemic index carbohydrate, high fibre and adequate protein are effective.

Physical activity of at least 150 minutes per week of moderate-intensity exercise, along with resistance training, improves insulin sensitivity and psychological well-being (8).

Metformin remains the most widely used insulin sensitizer, improving glucose tolerance and reducing progression to diabetes (8). GLP-1 receptor agonists show promise in obese PCOS due to weight loss and metabolic benefits and with increasing availability, efficacy and safety may find more utility in the future.

Table 2

DIABETES EDUCATOR CHECKLIST IN PCOS (8)

Domain	Key Actions
Risk assessment	Family history, BMI, ethnicity, prior GDM
Screening	OGTT, repeat every 1–3 years
Lifestyle education	Diet, exercise, sleep, stress
Medication counseling	Metformin, pregnancy safety
Psychosocial care	Screen for anxiety, depression
Life-course follow-up	Preconception, postpartum, menopause

PCOS, DIABETES AND PREGNANCY

Women with PCOS are at increased risk of GDM and adverse pregnancy outcomes. Early screening, tight glycemic control and individualized pharmacotherapy are essential. Post-partum follow-up is crucial but frequently neglected, despite high future diabetes risk.

LONG-TERM CARDIO-METABOLIC RISK

PCOS represents an early marker of future metabolic disease. When diabetes co-exists, cardio-metabolic risk is amplified and accelerated. A life-course approach emphasizing early prevention is essential.

CONCLUSION

PCOS and diabetes exist along a metabolic continuum driven by insulin resistance. Early identification and sustained lifestyle and pharmacological interventions can significantly reduce long-term metabolic burden. Diabetes educators play a pivotal role in translating this knowledge into preventive care.

REFERENCES

1. Legro RS, Kunselman AR, Dodson WC, Dunaif A. Polycystic ovary syndrome and Type 2 diabetes mellitus. Endocrine Reviews. 1999; 20:327-347.
2. Diamanti-Kandarakis E, Dunaif A. Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. Endocrine Reviews. 2012; 33:981-1030.
3. Moran LJ, Misso ML, Wild RA, Norman RJ. Impaired glucose tolerance, Type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. Human Reproduction Update. 2010; 16:347–363.
4. Teede HJ, Misso ML, Costello MF, et al. international evidence-based guideline for the assessment and management of polycystic ovary syndrome 2023. Human Reproduction. 2023; 38:2011–2048.
5. Jin JK, Kim JY, Kim JH. Obesity and polycystic ovary syndrome: implications for pathogenesis and management. Journal of Obesity & Metabolic Syndrome. 2024; 33:1-12.
6. Kalra S, Vaidya R, Verma M. Primary care screening and diagnosis of polycystic ovary syndrome. Indian Journal of Endocrinology and Metabolism. 2023; 27:105-106.
7. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. Endocrine Reviews. 1997; 18:774-800.
8. American Diabetes Association Professional Practice Committee. Classification and diagnosis of diabetes: Standards of Medical Care in Diabetes-2024. Diabetes Care. 2024;47(Suppl 1): S16-S33.

HEPATIC STEATOSIS AND FIBROSIS ASSESSMENT BY ULTRASONOGRAPHY TECHNIQUES

COMPARISON WITH OTHER METHODS

Chander P Lulla*

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterized by the presence of hepatic steatosis. Steatosis refers to excessive fat deposition in hepatocytes, while fibrosis represents the formation of scar tissue resulting from chronic liver injury. Hepatic steatosis is generally diagnosed when more than 5% of hepatocytes contain triglyceride. The global burden of MASLD has increased substantially over the past decades and it is currently estimated to affect approximately 25% of the world's population (1).

Steatotic liver disease encompasses a broad spectrum of conditions associated with fat accumulation in the liver. This spectrum ranges from simple steatosis (now referred to as MASLD) to steatosis associated with alcohol consumption, viral infections, drug exposure or certain monogenic disorders. As the disease progresses, some individuals develop steatohepatitis, a more severe stage characterized by hepatic inflammation, hepatocyte injury and varying degrees of fibrosis (2).

MASLD is strongly linked with obesity, Type 2 diabetes mellitus and metabolic syndrome and its global prevalence is increasing in parallel with these conditions (3). In the absence of timely intervention, MASLD can progress to more severe liver pathology, including cirrhosis, liver failure and hepatocellular carcinoma (4).

Early identification and precise quantification of hepatic steatosis are therefore essential to prevent disease progression and to guide appropriate therapeutic decisions. Traditionally, liver biopsy has been regarded as the reference standard for evaluating hepatic fibrosis and steatosis. Based on Brunt's classification, hepatic fat content is graded as follows: less than 5% (normal, S0); 5–33% (mild, S1); 34–66% (moderate, S2) and greater than 66% (severe, S3) (5,6,7).

However, the invasive nature of liver biopsy, along with its cost and limited suitability for repeated assessment, restricts its routine use, particularly for monitoring disease progression or treatment response. As a result, there has been a growing shift toward non-invasive approaches.

In recent years, objective, reproducible and quantitative imaging techniques have been developed to assess hepatic steatosis more accurately. These include both ultrasonography-based and magnetic resonance imaging (MRI)-based modalities, which offer promising alternatives to biopsy.

This review aims to provide a comprehensive overview of the key imaging techniques used in the detection and quantification of hepatic steatosis, highlighting their advantages, limitations and clinical utility.

ULTRASONOGRAPHY FOR STEATOSIS

Conventional B-mode Ultrasound

- Common initial tool: widely available, low-cost, safe.
- Detects steatosis based on liver echogenicity relative to kidney and decreased visibility of deep structures (8).

Performance

- Moderate-to-severe steatosis: sensitivity ~80–91% and specificity ~87–98% (8).

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- Mild steatosis: much lower sensitivity (as low as ~53%), making early detection challenging (8).

Limitations

- Subjective: interpretation depends on operator expertise.
- Lower sensitivity for early or mild steatosis.
- Cannot reliably quantify the fat percentage (8)

Ultrasound Attenuation and Elastography for Quantitative Steatosis and Fibrosis Estimation

Recently, a novel non-invasive tool that utilizes attenuation of the sound wave was developed. Ultrasound Elastography measures tissue stiffness, a proxy for fibrosis and Attenuation techniques assess steatosis.

Steatosis Assessment with Ultrasound-Based Tools

Several technologies provide improved assessment of liver fat compared with conventional B-mode ultrasonography by implementing quantitative approaches. Controlled Attenuation Parameter (CAP), Ultrasound-Guided Attenuation Parameter (UGAP), Attenuation Coefficient (ATT) and Attenuation Imaging (ATI) quantify liver fat by measuring the attenuation of Radiofrequency. In addition, Backscatter Coefficient (BSC) is a quantitative value that reflects ultrasonic pulses that are scattered back to an echo probe after passing through tissue. The number of scattered back ultrasonic pulses increases with increasing levels of liver fat. Therefore, BSC might also have the potential to detect and quantify hepatic steatosis. Finally, Quantitative Ultra Sonography (QUS) for fat-fraction estimation that quantifies liver fat using two backscatter parameters has been developed. This article discusses attributes and limitations of some of these modalities which are currently in practice.

Controlled Attenuation Parameter (CAP)

Measured with *FibroScan®* is commonly used to assess steatosis in units of dB/M. CAP was the first approved method for the quantification of liver fat based on attenuation evaluation. CAP is widely used to assess hepatic steatosis and its diagnostic accuracy has been validated extensively. CAP shows better accuracy than traditional ultrasound for detecting lower levels of fat and is more objective (9).

However, it may be susceptible to multi-reflection artifacts from subcutaneous tissues as well as disruptive structures such as vessels or diaphragm since the measurement area is not guided by imaging (10).

A meta-analysis that compared histologically graded steatosis with CAP and included 19 studies (2,735 patients with chronic liver disease) found areas under the receiver operating characteristics (AUROC) of 0.82 with the CAP threshold of 248 dB/m for steatosis of >11%, 0.86 with 268 dB/m for steatosis of >33%, and 0.89 with 280 dB/m for steatosis of >66%. However, the CAP value is affected by the presence of Non-alcoholic Fatty Liver Disaese (NAFLD), diabetes mellitus and obesity (10).

Ultrasound Guided Attenuation parameter (UGAP, ATT and ATI)

One disadvantage of CAP is that it is blind to the exact location of the region of interest. UGAP is a real-time, image-guided method measuring ultrasound attenuation, integrated into ultrasound systems.

- Attenuation is measured in dB/cm/MHz or dB/M) and it is integrated into ultrasound systems.
- Its correlation with Magnetic Resonance Imaging–Proton Density Fat Fraction (MRI-PDFF): Strong correlation: $r = 0.746$ (95% CI: 0.657–0.815) in a chronic liver disease cohort (11,12,13).

In impaired glucose tolerance patients: $r = 0.890$ ($p < 0.001$) (14).

- Diagnostic Accuracy (using MRI-PDFF or CAP/histology as reference):

  AUCs:

  - ≥ S1: 0.890–0.980
  - ≥ S2: 0.906–0.955

- S3: 0.912–0.985 (15,14,11)

  In MASLD with biopsy reference: AUC = 0.828–0.779 for ≥ grade 2 steatosis; inter-observer ICC up to 0.95 (16).

- ***UGAP Performance vs Other Modalities:***

  Better than CAP for detecting severe steatosis (eg. CAP AUC lower for S3) (15,17).

  Competes well with MRIPDFF: high AUCs approaching 0.98.

- ***Reproducibility & Feasibility:***

  Excellent intra and inter-observer reliability: ICC up to 0.95 (16,18).

  100% feasibility, unaffected by probe pressure, breathing, diet (18).

Table 1

STEATOSIS ASSESSMENT - PERFORMANCE METRIC

Method	What It Measures	Sensitivity	Specificity	AUROC / Notes	Comments / Cut-off
Conventional B-mode US	Qualitative echogenicity grading	~53–76% for any steatosis	~76–93%	–	Good for moderate/severe steatosis; limited for mild (<20–30%)[19]
Ultrasound (general) vs Biopsy	Visual steatosis	60–94%	84–95%	Lower for mild disease	Subjective interpretation; obesity lowers accuracy [20]
CAP (Controlled Attenuation Parameter)	Quantitative fat attenuation	~68–82% (S≥1)	~62–95%	~0.80–0.92	AUROC ~0.92 for ≥5% steatosis in some cohorts; accuracy drops for higher grades[8] High measurement failure, Based on A mode Image
CAP vs Biopsy (Meta-analysis)	Quantitative	~78–85%	~78–84%	AUROC ~0.82–0.88	CAP good overall but cut-offs & covariates (BMI, disease) affect accuracy[8]
UGAP	Quantitative				Accuracy higher than CAP, Lower measurement failure, Based on B Mode image[15,17]
MRI-PDFF	Proton density fat fraction	~95% (≥S1)	~95%	AUROC ≥0.95	Outperforms CAP & ultrasound for all grades of steatosis [21]
MRI-PDFF vs CAP (Direct)	Quantitative	Higher than CAP	Higher than CAP	MRI-PDFF AUROC ~0.97 vs CAP ~0.78 (S>33%)	Especially in obese / bariatric surgery cohorts[22]
CT (Non-contrast)	Liver attenuation	~43–77% (moderate)	~88–100%	–	Objective but lower sensitivity than MRI; has radiation [23]
CT (Non-contrast)	Liver attenuation	~43–77% (moderate)	~88–100%	–	Objective but lower sensitivity than MRI; has radiation [23]

KEY POINTS ON STEATOSIS:

- Ultrasound is good for moderate to severe steatosis but lacks sensitivity for mild fat infiltration (19).
- CAP adds quantification to ultrasound and performs better than B-mode alone, but its accuracy is influenced by BMI, device probe, and clinical factors (8). One disadvantage of CAP is that it is blind to the exact location of the region of interest. This factor contributes to high rates of measurement failure (0–24%).
- UGAP has no measurement failures and performs better than CAP
- MRI-PDFF is currently the gold standard non-invasive tool for steatosis quantification across all grades (21) however lack of easy availability, claustrophobia and cost are some of the deterrents for it to be used as a screening tool.

TYPES OF ULTRASOUND ELASTOGRAPHY

- Transient Elastography (TE) – *FibroScan®*
- Acoustic Radiation Force Impulse (ARFI)
- Point SWE (pSWE)
- 2 D Shear Wave Elastography (SWE)

These are more quantitative and reproducible methods than conventional ultrasound (24).

Fibrosis Assessment

- Elastography methods correlate well with biopsy for significant and advanced fibrosis and cirrhosis with AUROC values often high (close to 1.0 in many studies) (24).
- AUROC ranges vary with technique and disease stage, but many elastography methods perform strongly for detecting F3–F4 fibrosis (24).
- MRE is generally more accurate than ultrasound elastography for fibrosis staging, with lower technical failure rates (eg. in obesity) (25).

Table 2

FIBROSIS ASSESSMENT - PERFORMANCE METRICS

Method	Parameter	Sensitivity	Specificity	AUROC / Notes	Comments / Cut-off
B-mode US (conventional)	Morphologic changes	Low for early fibrosis	High for advanced disease	–	Cannot reliably stage early fibrosis
Transient Elastography (TE)	Liver stiffness measurement	~70–88%	~80–91%	AUROC ~0.84–0.94	AUROC increases with severity (best for cirrhosis) [26]
Shear Wave Elastography (SWE) / ARFI	Shear wave speed	(~70–85%)	Up to ~96%	–	Operator and vendor dependent [8]
FibroScan TE (Advanced)	Detecting ≥F2	~70–85%	~80–88%	AUROC ~0.83	Advanced fibrosis detection good but less accurate for low fibrosis[26]
TE for Cirrhosis (F4)	LSM cut-off ~12.5 kPa	~84–92%	~90–98%	AUROC ~0.94	Excellent for cirrhosis; minimal invasiveness [27]
MRE (Magnetic Resonance Elastography)	Liver stiffness	≥90%	≥90%	AUROC >0.90	More accurate than US elastography for staging fibrosis

LIVER BIOPSY: THE GOLD STANDARD

- Provides micro-pathological staging of steatosis and fibrosis.
- Still considered the reference standard in research and many clinical contexts.
- Procedure is invasive, carries risk (bleeding, sampling bias) and is not ideal for serial monitoring (26).

Cut-Offs and Grading

Steatosis

- 248 dB/m for any steatosis (≥S1), Sensitivity ~69%, Specificity ~82%, AUROC ~0.82.
- Larger studies suggest varying cut-offs may be needed depending on BMI and liver disease etiology (28).
- In patients with NAFLD, the optimal CAP thresholds for detecting MRI-PDFF of ≥5% and of ≥10% were 288 dB/m and 306 dB/m, respectively, which were considerably higher than the optimal CAP thresholds obtained from a meta-analysis with multiple aetiologies of liver disease (29).

Fibrosis

- ~8–9 kPa can suggest ≥F2 (significant fibrosis) in NAFLD/MASLD contexts. *(Specific cut-offs vary by etiology and guideline consensus)*
- ~12.5 kPa strongly suggests cirrhosis (F4) - Sensitivity up to ~92%, NPV high for ruling out cirrhosis (27).
  (Exact cut-offs vary among studies and guidelines, so always interpret with clinical context)

CLINICAL TAKE AWAYS WHEN TO USE ULTRASOUND

- First-line screening for steatosis due to easy accessibility and cost.
- Useful for identifying moderate to severe steatosis and advanced fibrosis via elastography.
- Temporal screening for response to therapy

WHEN TO PREFER OTHER METHODS

- Mild steatosis: MRI-based quantification is superior.
- Discordant results: MR Elastography or biopsy may be needed.

For serial monitoring of fibrosis progression or treatment response, non-invasive ultrasound imaging and serum markers improve feasibility compared to repeated biopsy (8).

CONCLUSION

Quantifying liver fibrosis and fat with MRI and Ultrasound attenuation techniques are important clinical tools in evaluating patients with suspected MASLD and Chronic liver disease replacing liver biopsy in most patient care settings. The goal was to provide a guide for practicing physicians and diabetes educators interpreting these studies. An advantage of a non-invasive and quantitative method is that repeat measurements are easy to obtain and it can evaluate temporal changes.

REFERENCES

1. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: an international expert consensus statement. *J Hepatol.* 2020; 73:202–209.
2. European Association for the Study of the Liver, European Association for the Study of Diabetes. EASL–EASD–EASO clinical practice guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *Obes Facts.* 2024; 17:374–444.
3. Eslam M, Sanyal AJ, George J, et al. MAFLD: a consensus-driven proposed nomenclature for metabolic associated fatty liver disease. *Gastroenterology.* 2020; 158:1999–2014.
4. Angulo P. Nonalcoholic fatty liver disease. *N Engl J Med.* 2002; 346:1221–1231.
5. Kleiner DE, Brunt EM, Van Natta M, Behling C, Contos MJ, Cummings OW, et al. Design and validation of a histological scoring system for nonalcoholic fatty liver disease. *Hepatology.* 2005; 41:1313–1321.
6. Ratziu V, Charlotte F, Heurtier A, Gombert S, Giral P, Bruckert E, et al. Sampling variability of liver biopsy in nonalcoholic fatty liver disease. *Gastroenterology.* 2005; 128:1898–1906.
7. Brunt EM, Kleiner DE, et al. The NAS and the histopathologic diagnosis in NAFLD: distinct clinicopathologic meanings. *Hepatology.* 2011; 53:810–820.
8. Grąt K, Grąt M, Rowiński O. Usefulness of different imaging modalities in evaluation of patients with non-alcoholic fatty liver disease. *Biomedicines.* 2020;8:298.

9. Jun BG, Park WY, Park EJ, Jang JY, Jeong SW, Lee SH, et al. A prospective comparative assessment of the accuracy of FibroScan in evaluating liver steatosis. *PLoS One.* 2017;12: e0182784.

10. Karlas T, Petroff D, Sasso M, Fan JG, Mi YQ, de Lédinghen V, et al. Individual patient data meta-analysis of controlled attenuation parameter (CAP) technology for assessing steatosis. *J Hepatol.* 2017; 66:1022–1030.

11. Tada T, Kumada T, Toyoda H, Kobayashi N, Sone Y, Oguri T, et al. Utility of attenuation coefficient measurement using an ultrasound-guided attenuation parameter for evaluation of hepatic steatosis: comparison with MRI-determined proton density fat fraction. *AJR Am J Roentgenol.* 2019; 212:332–341.

12. Suzuki Y, Yasuda S, Toyota H, Imajo K, Nakajima A, Sugimoto K, et al. Evaluation of hepatic steatosis in chronic liver disease by UGAP: comparison with MRI-PDFF. *Jpn J Med Ultrasonics.* 2021;48(Suppl): S275.

13. Imajo K, Toyoda H, Yasuda S, et al. Utility of ultrasound-guided attenuation parameter for grading steatosis with reference to MRI-PDFF in a large cohort. *Clin Gastroenterol Hepatol.* 2022; 20:2533–2541.

14. Gao R, Han J, Song D, Wang P, Chen H, Shen H, et al. Quantitative assessment of hepatic steatosis by ultrasound-guided attenuation parameter in patients with impaired glucose tolerance. *Insights Imaging.* 2025; 16:247.

15. Kuroda H, Abe T, Fujiwara Y, Nagasawa T, Takikawa Y. Diagnostic accuracy of ultrasound-guided attenuation parameter as a noninvasive test for steatosis in nonalcoholic fatty liver disease. *J Med Ultrasonics.* 2021. doi:10.1007/s10396-021-01123-0

16. Cannella R, Agnello F, Porrello G, Spinello AU, Infantino G, Pennisi G, et al. Performance of ultrasound-guided attenuation parameter and 2D shear wave elastography in patients with metabolic dysfunction-associated steatotic liver disease. *Eur Radiol.* 2025; 35:2339–2350.

17. Byenfeldt M, Grönlund C, Nasr P, Lindam A, Ekstedt M, Lundberg P, et al. A comparative study between ultrasound-guided attenuation parameter (UGAP), controlled attenuation parameter (CAP), and proton density fat fraction (PDFF) for assessment of hepatic steatosis. *Scand J Gastroenterol.* 2026; 61:124–132.

18. Zhao Y, Jia M, Zhang C, Feng X, Chen J, Li Q. Reproducibility of ultrasound-guided attenuation parameter (UGAP) in noninvasive evaluation of hepatic steatosis. *Sci Rep.* 2021;11:18274.

19. Petzold G. Role of ultrasound methods for the assessment of NAFLD. *J Clin Med.* 2022; 11:4581.

20. Karanjia RN, Crossey MM, Cox IJ, Fye HKS, Njie R, Goldin RD, et al. Hepatic steatosis and fibrosis: non-invasive assessment. *World J Gastroenterol.* 2016;22:9880–9897.

21. Wu S, Pan J, Song M, Zhao YC, Chen W, Huang H, et al. Performance of magnetic resonance imaging and ultrasound for identifying different degrees of hepatic steatosis: a systematic review and meta-analysis.

22. Garteiser P, Castera L, Coupaye M, Doblas S, Calabrese D, Burgio MD, et al. Prospective comparison of transient elastography, MRI and serum scores for grading steatosis and detecting nonalcoholic steatohepatitis in bariatric surgery candidates. *JHEP Rep.* 2021;3:100381.

23. Dag N, Kutlu R. A to Z of imaging modalities for the diagnosis and quantification of hepatic steatosis. *J Inonu Liver Transpl Inst.* 2025;3(Suppl 1): S71–S77.

24. Pozowski P, Bilski M, Bedrylo M, Sitny P, Zaleska-Dorobisz U. Modern ultrasound techniques for diagnosing liver steatosis and fibrosis: a systematic review with a focus on biopsy comparison. *World J Hepatol.* 2025;17:100033.

25. Wang J, Qin T, Sun J, Li S, Cao L, Lu X. Non-invasive methods to evaluate liver fibrosis in patients with nonalcoholic fatty liver disease. *Front Physiol.* 2022; 13:1046497.

26. Kazi IN, Kuo L, Tsai E. Noninvasive methods for assessing liver fibrosis and steatosis. *Gastroenterol Hepatol.* 2024;20(1):21–29.

27. Damjanovska S, Karb DB, Tripathi A, Asirwatham J, Delozier S, et al. Accuracy of ultrasound elastography and fibrosis-4 index (FIB-4) in ruling out cirrhosis in obese NAFLD patients. *Cureus.* 2022;14: e29445.

28. Karlas T, Petroff D, Sasso M, Fan JG, Mi YQ, de Lédinghen V, et al. Individual patient data meta-analysis of controlled attenuation parameter (CAP) technology for assessing steatosis. *J Hepatol.* 2017;66:1022–1030.

29. Caussy C, et al. Optimal threshold of controlled attenuation parameter with MRI-PDFF as the gold standard for detection of hepatic steatosis. *Hepatology.* 2018; 67:1348–1359.

GUT MOTILITY PROBLEMS IN DIABETES MELLITUS

AN INDIAN CLINICAL PRACTICE PERSPECTIVE FOR DIABETES EDUCATORS

Ajay Jhaveri*

INTRODUCTION

India is often referred to as the "diabetes capital of the world," with a rapidly rising burden of Type 2 diabetes mellitus affecting both urban and rural populations. Alongside well-recognized complications such as cardiovascular disease, nephropathy, retinopathy and neuropathy, gastrointestinal (GI) complications due to altered gut motility are common but frequently overlooked in routine diabetes care.

In Indian clinical practice, gut motility problems are often underreported due to social stigma, normalization of symptoms (eg. constipation or bloating), lack of awareness and limited consultation time. These disorders significantly affect dietary tolerance, medication adherence, glycemic variability, nutritional status and quality of life. For diabetes educators in India who are often the most accessible healthcare professionals; early recognition and education are critical (1).

This article focuses on the Indian scenario, including dietary habits, healthcare access, cultural factors and common therapeutic challenges. This is expected to help diabetes educators effectively address gut motility problems in people with diabetes.

UNDERSTANDING GUT MOTILITY AND DIABETES

Gut motility refers to the coordinated movement of food through the gastrointestinal tract, regulated by:

- Enteric nervous system
- Autonomic nervous system
- Smooth muscle function
- Gut pacemaker cells (Interstitial cells of Cajal)
- Gastrointestinal hormones

In diabetes mellitus, chronic hyperglycemia leads to diabetic autonomic neuropathy, which disrupts this coordination. Over time, this results in delayed, rapid, or irregular movement of food through different segments of the gut (2).

PATHOPHYSIOLOGY: RELEVANCE TO THE INDIAN PATIENT

Chronic Hyperglycemia and Late Diagnosis

Many Indian patients are diagnosed late, often after several years of undetected hyperglycemia. This increases the risk of autonomic neuropathy and GI complications even at first presentation (3).

Autonomic Neuropathy

Damage to vagal and sympathetic nerves affects esophageal peristalsis, gastric emptying, intestinal transit and anorectal function. This neuropathy often coexists with peripheral neuropathy, a common finding in Indian patients (2).

Nutritional and Dietary Factors

Indian diets are typically high in carbohydrates and rich in fiber due to the regular consumption of whole grains, pulses and vegetables, with fat intake varying depending on regional cooking practices. While such diets are generally healthy and beneficial for overall metabolic health, the high fiber content in traditional meals may worsen symptoms in patients with gastroparesis or those experiencing severe bloating and dietary modifications may be required in such cases. (4).

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Recurrent Infections and SIBO

Poor gut motility increases the risk of small intestinal bacterial overgrowth (SIBO), which is common but underdiagnosed in India due to limited testing facilities (2).

COMMON GUT MOTILITY DISORDERS IN DIABETES (INDIAN CONTEXT)

Esophageal Dysmotility

Esophageal dysmotility in people with diabetes commonly presents with difficulty in swallowing, particularly solids and a sensation of food sticking in the chest. Some patients may also experience persistent heartburn that does not respond adequately to antacids. In routine practice, these symptoms are frequently attributed to simple "acidity" and are often self-managed with over-the-counter medications. This tendency toward self-treatment can delay proper evaluation and diagnosis, allowing the underlying motility disorder to remain unrecognized (2).

Diabetic Gastroparesis: Clinical Importance and Glycemic Impact

In Indian gastroparesis is increasingly getting recognized among individuals with long-standing diabetes. It is particularly suspected in insulin-treated patients with Type 2 diabetes, those who demonstrate persistently erratic HbA1c levels despite good treatment adherence and among women and elderly patients. Clinically, patients commonly report early satiety, an inability to finish normal meals, postprandial bloating and episodes of nausea or vomiting, sometimes containing undigested food consumed several hours earlier. A key concern is the impact on glycemic control. Delayed gastric emptying leads to a mismatch between insulin action and glucose absorption, resulting in early post-meal hypoglycemia followed by late post-meal hyperglycemia. These unexplained glucose fluctuations are often misinterpreted as poor compliance or dietary indiscretion, which may delay appropriate recognition and management of the underlying motility disorder (5).

Diabetic Enteropathy

Diabetic enteropathy is a manifestation of autonomic neuropathy affecting intestinal motility and function. One of its common presentations is diabetic diarrhea, which is typically painless, often occurs at night and may cause significant social embarrassment for patients. On the other hand, constipation is also frequently seen, particularly among Indian patients and is often chronic and severe. Many individuals do not report these symptoms, either due to embarrassment or because they consider it routine or diet-related. Some patients experience alternating episodes of diarrhea and constipation, a pattern that suggests underlying autonomic neuropathy and may also indicate the presence of small intestinal bacterial overgrowth (SIBO) (6).

Small Intestinal Bacterial Overgrowth (SIBO)

Small intestinal bacterial overgrowth (SIBO) is an important but often underrecognized complication in people with diabetes and is particularly relevant in the Indian context. Impaired gut motility due to autonomic neuropathy promotes bacterial stasis and overgrowth in the small intestine. In addition, factors such as variable sanitation conditions in some areas and frequent or repeated antibiotic exposure may further increase the risk. Clinically, SIBO commonly presents with excessive gas, abdominal bloating and distension, chronic diarrhea and features of malabsorption, including weight loss, nutritional deficiencies or unexplained worsening of glycemic control (7).

Ano-rectal Dysfunction

Ano-rectal dysfunction is another manifestation of diabetic autonomic neuropathy and may present with symptoms such as excessive straining during defecation and a persistent sensation of incomplete evacuation. In some cases, patients may also experience fecal incontinence, although this is rarely reported. In the Indian setting, cultural factors and social embarrassment often prevent patients from openly discussing bowel control problems. As a result, these symptoms may remain unrecognized unless healthcare providers ask about them in a sensitive manner (6).

DIAGNOSTIC CHALLENGES IN INDIAN SETTINGS

Advanced diagnostic tests may not always be available.

Diabetes educators should advise prompt medical evaluation when certain gastrointestinal symptoms suggest a potentially serious underlying problem. Patients should be referred if they report persistent or recurrent vomiting, significant unintentional weight loss or frequent episodes of unexplained hypoglycemia. Medical assessment is also necessary in cases of chronic diarrhea lasting more than two to three weeks, as well as severe constipation that does not improve despite appropriate lifestyle and dietary measures. Early referral helps ensure timely diagnosis and prevents worsening nutritional, metabolic and glycemic complications (4).

MANAGEMENT PRINCIPLES IN INDIAN CLINICAL PRACTICE

Glycemic Control: The Foundation

Glycemic control is the foundation of prevention and slowing the progression of autonomic neuropathy, as sustained and stable blood glucose levels help reduce further nerve damage. Diabetes educators play a crucial role in supporting this process by encouraging regular self-monitoring of blood glucose (SMBG) or the use of continuous glucose monitoring (CGM) wherever available. They also help patients identify postprandial glucose patterns and provide guidance on appropriate insulin timing in relation to meals to achieve optimal glycemic control (8).

Dietary Management (Indian Diet Adaptations)

For Gastroparesis

For patients with gastroparesis, dietary modification plays an important role in symptom management. Small, frequent meals (5–6 times a day) are recommended to reduce gastric burden and improve tolerance. Soft and easily digestible foods such as khichdi, curd rice and dal water are preferable. Intake of insoluble fiber, including raw vegetables and salads, should be reduced as it may delay gastric emptying. Fried, oily and spicy foods should be avoided and the consumption of carbonated beverages as well as tea or coffee immediately after meals should be limited (4).

For Diarrhea

For patients experiencing diarrhea, maintaining adequate hydration is essential and oral sugarfree rehydration solution (ORS) may be used when needed. Lactose intake should be temporarily reduced, as it may worsen symptoms and patients should avoid street food and high-fat foods to prevent further gastrointestinal irritation (4).

For Constipation

In cases of constipation, dietary fiber should be increased gradually rather than suddenly to avoid discomfort and bloating. Adequate water intake is important to support bowel function, along with maintaining regular meal timings to help establish a consistent bowel routine (9).

Physical Activity

Mild physical activity plays an important role in improving gastrointestinal function. Light walking for 10–15 minutes after meals can help enhance gastric emptying and reduce symptoms. Patients should be advised to avoid lying down immediately after eating (4).

Medication Awareness (Educator's Perspective)

From an educator's perspective, medication awareness is also essential, as some commonly used drugs in India can affect gastrointestinal motility. Metformin may cause diarrhea and bloating, GLP-1 receptor agonists can worsen gastroparesis and anticholinergic medications may aggravate constipation. Patients should be clearly advised not to stop or modify their medications without consulting their doctor (4).

Addressing Psycho-social Barriers

Addressing psychosocial barriers is equally important. Many patients tend to normalize gastrointestinal symptoms as "gas" or "weak digestion," may feel embarrassed discussing bowel habits, or may prefer home remedies instead of seeking medical advice. Diabetes educators should therefore ask open-ended and non-judgmental questions, normalize gastrointestinal symptoms as a possible part of diabetes and encourage early reporting so that timely management can be initiated (10).

ROLE OF DIABETES EDUCATORS IN INDIA

Diabetes educators are central to managing gut motility problems due to frequent patient contact. Diabetes educators play a central role in the identification and management of gut motility problems, as they often have the most frequent and sustained contact with patients. Their responsibilities include routine screening for gastrointestinal symptoms during follow-up visits and providing dietary counselling that is practical and aligned with Indian food habits and cultural preferences. Educators are also well positioned to recognize patterns of glycemic variability that may suggest underlying motility disorders. An important aspect of their role is patient empowerment through education about symptom recognition, self-care and the importance of timely reporting. In addition, diabetes educators should facilitate early referral to physicians or gastroenterologists when warning signs are present and coordinate care with dietitians and the broader healthcare team to ensure comprehensive management (11).

Case Example

A 60-year-old man with 15 years of Type 2 diabetes on basal-bolus insulin reports frequent bloating and erratic sugars. On questioning, he admits to early fullness and occasional vomiting after heavy meals. The diabetes educator identifies probable gastroparesis, suggests meal modification (smaller portions, soft foods), walking after meals and alerts the physician. Insulin timing is adjusted, resulting in improved glucose control and symptom relief.

Public Health and Community Considerations

In India, a large proportion of people with Type 2 diabetes are managed at the primary care level and diabetes educators often serve patients from semi-urban and rural communities where access to specialist care may be limited. In such settings, simple, practical education can have a significant impact on preventing gastrointestinal complications and improving overall outcomes. Guidance on maintaining regular meal timing, ensuring adequate hydration, promoting healthy bowel habits and discouraging unsupervised self-medication can help patients manage symptoms early and reduce the risk of worsening motility problems and glycemic instability (12).

CONCLUSION

Gut motility disorders are common, disabling and under-recognized complications of diabetes in India. Cultural factors, dietary habits, delayed diagnosis and limited awareness contribute to underreporting and inaction to resolve these issues.

Diabetes educators in India are uniquely positioned to identify early symptoms, provide culturally appropriate dietary guidance, improve glycemic outcomes and advocate timely care. Integrating gut health into routine diabetes education can significantly enhance quality of life and overall diabetes management.

REFERENCES

1. Bytzer P, Talley NJ, Leemon M, et al. Prevalence of gastrointestinal symptoms in diabetes mellitus. *Am J Gastroenterol.* 2001; 96:604–611.
2. Bharucha AE, Kudva YC, Prichard DO. Diabetic gastroenteropathy: clinical features and pathophysiology. *Gastroenterology.* 2015; 149:1657–1666.
3. Anjana RM, Deepa M, Pradeepa R, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. *Lancet Diabetes Endocrinol.* 2023; 11:98–110.
4. Camilleri M, Parkman HP, Shafi MA, et al. Clinical management of gastroparesis. *Am J Gastroenterol.* 2013; 108:18–37.
5. Kashyap P, Farrugia G. Diabetic gastroparesis: what we have learned and had to unlearn. *Gut.* 2010; 59:1716–1726.
6. Vinik AI, Maser RE, Mitchell BD, et al. Diabetic autonomic neuropathy. *Diabetes Care.* 2003; 26:1553–1579.
7. Camilleri M, Bharucha AE, Farrugia G. Diabetic gastrointestinal disorders. *Gastroenterology.* 2011;140 :1775–1785.
8. Pop-Busui R, Boulton AJM, Feldman EL, et al. Diabetic neuropathy: a position statement by the American Diabetes Association. *Diabetes Care.* 2017;40 :136–154.
9. American Gastroenterological Association (AGA). Medical position statement on constipation. *Gastroenterology. 2013;*

144:211–217.

10. Young-Hyman D, de Groot M, Hill-Briggs F, et al. Psychosocial care for people with diabetes: a position statement of the American Diabetes Association. *Diabetes Care.* 2016; 39:2126–2140.
11. Powers MA, Bardsley J, Cypress M, et al. Diabetes self-management education and support in adults with type 2 diabetes: a consensus report of the ADA, AADE, and the Academy of Nutrition and Dietetics. *Diabetes Care.* 2020; 43:1636–1649.
12. India State-Level Disease Burden Initiative; Diabetes Collaborators. The increasing burden of diabetes and variations among the states of India. *Lancet Glob Health.* 2018;6: e1352–e1362.

QUESTIONS AND ANSWERS

Q: What is the difference between plant protein and animal protein for renal patients?

A. In patients with chronic kidney disease (CKD), especially those with Diabetic Kidney Disease (DKD), the type and quantity of dietary protein play an important role in disease progression and metabolic management in kidney disease. Diabetic kidney disease is one of the most common causes of CKD and is characterized by significant proteinuria and accelerated decline in kidney function. Persistent hyperglycemia, insulin resistance and metabolic disturbances promote oxidative stress and the formation of advanced glycation end products leading to endothelial dysfunction, podocyte injury and mesangial and tubular abnormalities. Another key mechanism involved in the development of diabetic nephropathy is activation of the renin angiotensin aldosterone system, which increases intraglomerular pressure and accelerates kidney damage.

Dietary interventions such as low-protein diets (LPDs) are often recommended in CKD to reduce glomerular hyperfiltration and slow disease progression. Evidence suggests that LPDs may reduce intraglomerular pressure, partly through modulation of the renin–angiotensin–aldosterone system, resulting in reduced proteinuria and delayed progression to kidney replacement therapy.

Plant proteins have lower bioavailability and produce less renal hyperfiltration compared to animal protein. Owing to the lower bioavailability, plant-based diets may attenuate the increase in intraglomerular pressure that typically occurs after a high protein intake. Plant proteins also have lower phosphorus bioavailability and generate a lower dietary acid load, which help reduce metabolic acidosis and improve mineral metabolism in CKD. Additionally, plant-based foods are rich in dietary fiber and antioxidants, which may improve gut microbiota and reduce production of uremic toxins.

On the other hand, animal proteins have a higher biological value and provide all essential amino acids in adequate proportions. This can be beneficial for maintaining muscle mass and preventing protein-energy wasting, particularly in patients with advanced CKD or those undergoing dialysis. However, animal proteins are associated with higher phosphorus absorption, increased acid load, and greater stimulation of glomerular hyperfiltration, which may contribute to a speedier kidney function decline.

For these reasons, many dietary approaches in CKD emphasize on recommending a plant-dominant low-protein diet, typically providing about 0.6–0.8 g/kg/day of protein. Vegetarian dietary patterns are being increasingly used in CKD patients to facilitate adherence to these recommendations, while still providing adequate nutrition. Overall, a balanced approach that prioritizes plant-based proteins with sufficient essential amino acids may slow CKD progression, reduce proteinuria and improve metabolic outcomes in renal patients.

Q. What would occur if electrolytes are high and it is not corrected by diet and medications?

A. If electrolytes, particularly potassium remain high in a patient with kidney disease and is not controlled with diet or medications, it can lead to an exacerbated condition of chronic kidney disease. The kidneys help maintain the balance of electrolytes but impairment in kidney function causes electrolytes to build up in the blood.

High potassium levels can affect the normal rhythm of the heart, leading to irregular heartbeat, muscle weakness and in severe cases sometimes cardiac arrest. Similarly, excess sodium and phosphorus may accelerate fluid retention, high blood pressure, bone problems and sometimes confusion or other neurological symptoms. If these imbalances persist and are not treated timely, they may become life-threatening leading to the patient requiring dialysis to restore hemodynamic balance.

RECIPES

MILLET VEGETABLE KHICHDI

INGREDIENTS

50 gm Foxtail millet (Kangni)

50 gm split yellow Moong dal

100 gm mixed vegetables (carrot, beans, peas, bottle gourd, capsicum)

50 gm small Onion

50 gm Tomato

5 gm grated Ginger

1 green Chilli

½ tsp Turmeric powder

1 tsp Cumin seeds

1 tsp Groundnut oil

Salt to taste

Few fresh coriander leaves

METHOD

Wash millet and moong dal.

Heat oil in a pressure cooker and add cumin seeds.

Add vegetables and sauté for 2–3 minutes.

Add millet, dal, turmeric, salt and water.

Pressure cooks for 3 whistles.

Garnish with coriander leaves and serve warm.

PROVIDES 3 SERVINGS

Nutritional Information Per Serving

Energy (kcals)	Protein (grams)	Carbohydrate (grams)	Fat (grams)	GI
150	5	20	2	Low

SPECIAL FEATURES

- Fibre rich
- Low Glycemic Index

METHI PANEER MILLET WRAP

INGREDIENTS

60 gm Bajra or Jowar flour

50 gm finely chopped fresh Methi leaves

60 gm skim Paneer (crumbled)

50 gm grated Carrot and Capsicum

1 tsp Groundnut oil

20 gm hung Curd

Salt, pepper and roasted cumin powder according to taste

METHOD

Prepare a soft dough using millet flour, methi leaves, salt, and water.

Roll into thin rotis and cook on a pan.

Mix paneer, vegetables, hung curd, and spices to make filling.

Place filling on the roti and roll it like a wrap.

PROVIDES 2 SERVINGS

Nutritional Information Per Serving

Energy (kcals)	Protein (grams)	Carbohydrate (grams)	Fat (grams)	GI
120	10	20	4	Low

SPECIAL FEATURES

- High Protein
- Low Glycemic Index

HOW KNOWLEDGEABLE ARE YOU?

1. Myocardial ischemia in diabetes presents silently, without symptoms because of:

A. Hyperlipidemia

B. Autonomic neuropathy

C. Hypertension

D. Obesity

2. Which of the following is a macrovascular complication of diabetes?

A. Retinopathy

B. Neuropathy

C. Coronary artery disease

D. Nephropathy

3. Loss of sweating in the feet in diabetes is due to:

A. Peripheral vascular disease

B. Autonomic neuropathy

C. Infection

D. Electrolyte imbalance

4. Which of the following is the most common microvascular complication of diabetes?

A. Coronary artery disease

B. Diabetic retinopathy

C. Stroke

D. Peripheral arterial disease

5. Diabetic nephropathy is usually first detected by:

A. Elevated serum creatinine

B. Hematuria

C. Microalbuminuria

D. Proteinuria >3.5 g/day

6. Which nerve fibers are primarily affected in diabetic autonomic neuropathy?

A. Motor nerves

B. Sensory nerves

C. Sympathetic and parasympathetic nerves

D. Cranial nerves

7. Postural hypotension in diabetes is due to:

A. Volume depletion

B. Excessive dose of diuretics

C. Autonomic neuropathy

D. All of above

B. Erectile dysfunction in men with diabetes is mainly due to:

A. Hormonal deficiency

B. Psychological causes

C. Autonomic neuropathy and vascular disease

D. Infection

C. Diabetic gastroparesis may present with:

A. Early satiety

B. Nausea and vomiting

C. Bloating after meals

D. All of the above

10. The earliest clinical feature of diabetic peripheral neuropathy is usually:

A. Muscle weakness

B. Burning sensation in feet

C. Paralysis

D. Loss of reflexes

ANSWERS:

1. B 2. C 3. B
4. B 5. C 6. C
7. D 8. C 9. D
10. B

MYTHS AND FACTS

Myth: Continuous glucose monitors (CGM) are only for Type 1 diabetes.

Fact: A Continuous Glucose Monitor (CGM) is not only used in the management of glucose in Type 1 diabetes, but it is also now increasingly used in people with Type 2 diabetes. CGM devices continuously measure glucose levels in the interstitial fluid throughout the day and night, providing the real-time information about glucose trends in contrast to a single reading like a finger-stick test. This assists patients and diabetes educators in understanding how meals, physical activity, stress and medications affect blood glucose levels.

In Type 2 diabetes, CGM can give insight into detecting hidden hyperglycemia as well as episodes of hypoglycemia, especially in patients on insulin or multiple glucose-lowering medications. It also improves patient awareness and encourages better lifestyle choices, as individuals can immediately see how diet and/or exercise influences blood glucose levels. In addition, the CGM data allows clinicians to adjust medications more precisely and improve overall glycemic control. CGM has emerged as an important tool not only in Type 1 diabetes but also for better monitoring and management of Type 2 diabetes.

Myth: Intermittent fasting is safe for everyone with diabetes.

Fact: Intermittent fasting may help many people with diabetes improve weight and glucose control, but it is not suitable for everyone. People with diabetes who are taking insulin or sulphonylureas or glinides may be at risk of hypoglycemia (low blood sugar) if they skip meals or fast for long periods or indulge into a severe bout of exercise. Fasting can also lead to large fluctuations in blood glucose levels, dehydration or overeating during non-fasting periods.

Therefore, intermittent fasting should only be considered under medical supervision with proper adjustment of medications, careful monitoring of blood glucose levels and appropriate meal planning. Individual factors such as type of diabetes, medications, age and overall health status should always be evaluated before recommending fasting as part of diabetes management.

Myth: GLP-1 injections are a quick fix for rapid weight loss.

Fact: GLP-1 receptor agonists such as Liraglutide, Semaglutide and Tirzepatide lead to gradual and sustained weight loss rather than rapid results as demonstrated in large clinical trials. For example, in the STEP-1 trial, participants achieved an average weight loss of about 15% over 68 weeks, highlighting that meaningful outcomes occur over many months and not quickly. Longer-term studies show that this weight loss can be maintained with continued therapy but weight regain is common after stopping the medication, reinforcing that these drugs are part of chronic weight management rather than a one-time solution. Therefore, GLP-1 injections are effective tools but they work best when combined with long-term lifestyle changes and ongoing medical supervision.

DIABETES EDUCATOR CERTIFICATE COURSE

Dr Chandalia's DENMARC in association with Help Defeat Diabetes Trust (HDDT) presents to you a Certificate course for Diabetes Educators (CDE)!

Help Defeat Diabetes Trust (HDDT) is a registered, non-profit public trust, having amongst its many objectives, the main objective of promoting education and awareness about diabetes among people from different fields.

- **Open to** graduates in Nutrition and professionals in Medicine, Nursing, Pharmacy, Occupational Therapy and Physiotherapy.
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- **For more details,** visit our website or email us at helpdefeatdiabetesinfo@gmail.com.

MEMBERSHIP FORM

Association of Diabetes Educators (ADE)

(For eligibility criteria: Check Website www.diabeteseducatorsindia.com)
(Kindly print, duly fill, scan and upload)

Name ………………..………………………………………………………Age:……………………Gender:………………

Address ……

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E-mail id: ……………………………………………………………………...

Educational Qualifications:……………………………………..........................…………………………………………

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Work Experience: ……...…………………………………………………………………………………………………

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- Update my knowledge and skills
- As a faculty in ADE's Educational Activities
- Organizational Activities as Office Bearer

Please pay the membership fees (Rs.2000/-) through NEFT / RTGS/online to the following bank account. The details are as follows:

Account name: Association of Diabetes Educators

Account type: Savings Account

Name of the bank: Bank of India

Account number: 006610110001734

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