Letter to the Editor (Correspondence) on the Article Entitled "Gastric Emptying Patterns in Type 2 Diabetes Mellitus Patients with Symptoms of Gastroparesis and the Impact of Levosulpiride on These Patterns"

Nitin Chintaman Gawari[1], Sujata Prakash Shingare[2], AV Tilak[3], Krupa H Vasani[4]

[1,2]Assistant Professor, [3]Professor and HOD, [4]Junior Resident, Department of Pharmacology, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, India

Dear Editor,

We read with interest the expert consensus by Kant et al. on levosulpiride for the management of symptoms of gastroparesis in type 2 diabetes mellitus (T2DM) patients. The article is timely and informative, particularly in view of the increasing complexity of treatment in Indian diabetes care. The debate around the novel mechanism of levosulpiride—addressing the relationship both gastric motility patterns and therapeutic outcomes—is well articulated and is in line with the increasing emphasis on both.[1]

The recommendation on the use of levosulpiride in patients with symptoms of gastroparesis in T2DM is practical and relevant. Levosulpiride, in particular, has been shown to effectively alleviate symptoms such as nausea, vomiting, and early satiety, likely due to its action on the chemoreceptor trigger zone.[2]

However, we would like to make a few comments:

- The authors have clearly established the global and Indian burden of diabetes, which is strong. However, the introduction could flow more smoothly from general (diabetes mellitus burden) to specific (gastroparesis) to research gap. The research gap ("limited research on the correlation between scintigraphic patterns and symptoms") is mentioned at the end but could be more explicit and earlier in the text. The rationale for using levosulpiride is stated but not linked directly to the research gap.[3]
- Data transparency: A flow diagram of participant recruitment and attrition was not provided. Adding a participant flow diagram showing numbers at each stage will be better (screened, eligible, included, followed up, analyzed). There is no accounting for missing data, and it is unclear whether all 27 participants completed follow-up. The number of participants who did not improve or who worsened was not mentioned.
- Confounding variables (glycemic control, concomitant medications, and diet) are not discussed. The article does not connect to the local (North India) study context, which is important for external validity. There is also a lack of discussion on generalizability to other populations.

Orcid

Nitin Chintaman Gawari https://orcid.org/0009-0006-4241-8909
Sujata Prakash Shingare https://orcid.org/0009-0002-5260-364X

References

1. Kant R, Kavya NP, Mittal R, et al. Gastric emptying patterns in type 2 diabetes mellitus patients with symptoms of gastroparesis and the impact of levosulpiride on these patterns. J Assoc Phys India 2025;73(10):15–19.
2. Mearin F, Rodrigo L, Pérez-Mota A, et al. Levosulpiride and cisapride in the treatment of dysmotility-like functional dyspepsia: a randomized, double-masked trial. Clin Gastroenterol Hepatol 2004;2(4):301–308.
3. Abraham R, Foong D, Ho V. Combined gastric alimetry and gastric emptying scintigraphy testing increases clinician certainty in the diagnosis and management of suspected gastroparesis. J Neurogastroenterol Motil 2026;32(1):61–70.

Gonadotropin-releasing Hormone Agonist-induced Autoimmune Thyroiditis in a 49-year-old Woman

Sruthi Yalamanchili[1], Nikhil Gupta[2], Tanvi Batra[3], Atul Kakar[4]

[1,2]DNB trainee; [3]Associate Consultant; [4]Chairman and Senior Consultant, Department of Internal Medicine, Sir Ganga Ram Hospital, New Delhi, India

Dear Editor,

Goserelin is a synthetic gonadotropin-releasing hormone (GnRH) agonist that initially stimulates and then profoundly suppresses pituitary gonadotropin secretion through receptor desensitization. It is commonly prescribed in the management of hormone-dependent conditions such as prostate cancer, breast cancer, endometriosis, and adenomyosis.[1] Typical side effects of goserelin are attributable to the resulting hormonal deprivation, including vasomotor symptoms, bone mineral loss, mood disturbances, and injection site reactions. However, rare endocrine complications beyond expected hypoestrogenism, such as thyroid dysfunction, have also been described.[2,3] Here we discuss the case of a 49-year-old woman, a known case of prediabetes (glycosylated hemoglobin: 6.2%), hypertension, and dyslipidemia, who presented to the gynecology outpatient department with complaints of menorrhagia and infertility. Laboratory investigations revealed normal blood counts and normal kidney and liver functions, with a standard thyroid function test [free T4/thyroid-stimulating hormone (TSH): 11.1 pmol/L and 2.10 mU/L, respectively]. Hormonal workup, including luteinizing hormone, follicle-stimulating hormone, and serum anti-Mullerian hormone, was normal. CA-125 was normal. A Pap smear showed no evidence of malignancy, and mammography showed breast imaging-reporting and data system (BI-RADS) 1. Ultrasound of the pelvis revealed a markedly bulky uterus (172 mL) and no significant fibroids. Evaluation revealed abnormal uterine bleeding associated with adenomyosis (AUB-A). Considering this to be a mechanical barrier to successful embryo implantation, she was initiated on GnRH agonist therapy. She was administered a monthly dose of goserelin (Zoladex) 3.6 mg subcutaneously for a total of 6 months. Treatment successfully induced a hypoestrogenic menopausal-like state, leading to amenorrhea. A follow-up pelvic ultrasound after completion of therapy showed a significant reduction in uterine volume from 172 to 93.4 mL, confirming a good anatomical response to GnRH therapy. No thyroid-related symptoms were reported during the treatment course. Six weeks after her last dose of GnRH agonist, she presented to us with complaints of palpitations, episodic tremors, heat intolerance, anxiety, and insomnia. There was no history of neck pain or visual disturbances. On examination, she had fine tremors with tachycardia (pulse rate: 102/minute, regular). The rest of the hemodynamic and systemic examination was normal. Physical examination of the eyes and thyroid gland revealed no abnormalities. Initial investigations revealed a normal complete blood count and liver and kidney function tests. Thyroid function tests revealed overt thyrotoxicosis [Low TSH (0.004 µIU/mL), low fT3 (1.098 pg/dL) and high fT4 (3.6 ng/dL)]. Thyroid antibody testing revealed markedly elevated antithyroid peroxidase (anti-TPO) and antithyroglobulin (anti-Tg) levels, 764