A Case of Recurrent Acute Pancreatitis Induced by Intragastric Balloon Compression

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Cite this article as: Harput ZN, Gödelek İ, Aydin MK, Bayraktar O, Altintaş E, Sezgin O. A case of recurrent acute pancreatitis induced by intragastric balloon compression. *Turk J Gastroenterol.* 2026;37(11):1196-1198.

Dear Editor,

We read with great interest the study by Yüksel et al, which reported the 6-month outcomes of endoscopic intragastric balloon (IGB) placement in a single-center prospective study. No major adverse events or significant complications were observed, except for nausea that led to balloon removal in 1.5% of patients. Based on these findings, IGB appears to be a relatively safe procedure.[1] However, although rare, life-threatening complications requiring balloon removal can occur. Given the increasing prevalence of obesity and growing use of IGB therapy, awareness of these potential complications remains important, even when they are uncommon. In this context, we present a case of recurrent pancreatitis secondary to IGB placement.

A 26-year-old woman presented with sudden-onset epigastric pain radiating to the back. Her medical and family histories were unremarkable, except for IGB placement at another center 1 month earlier. She had no history of alcohol or drug use, smoking, or surgery and had not received any medications, including glucagon like peptide 1 (GLP-1) receptor analogs. Her body mass index before IGB placement was 30 kg/m^2, and she lost 5 kg within 1 month after the procedure.

On admission, the patient was hemodynamically stable, and physical examination revealed tenderness in the epigastrium and left upper quadrant of the abdomen. Laboratory test results were normal except for elevated serum amylase (439 U/L) and lipase (1361 U/L) levels, both exceeding 3 times the upper limit of normal. Abdominal computed tomography (CT) revealed compression of the pancreatic head and body by the intragastric balloon, with mild peripancreatic inflammation. Acute pancreatitis was diagnosed based on typical abdominal pain, serum amylase and lipase levels exceeding 3 times the upper limit of normal, and CT findings (Figure 1A).

Based on the clinical history and laboratory findings (such as hypercalcemia, hypertriglyceridemia, trauma, any intervention in the bile ducts, and medications), including IgG4 levels, no alternative etiology for acute pancreatitis was identified. Abdominal ultrasonography and magnetic resonance cholangiopancreatography were also performed to evaluate for biliary sludge or microlithiasis associated with rapid weight loss. However, as Endoscopic ultrasound (EUS) was not performed, microlithiasis could not be completely excluded. As no pathological findings, including pancreatic duct dilatation, duodenal migration, or catheter displacement, were detected on these imaging modalities and IGB compression of the pancreatic head and body was confirmed (Figure 1b), acute pancreatitis secondary to IGB compression was diagnosed. Standard treatment for acute pancreatitis was initiated. According to the Revised Atlanta Classification, the patient was diagnosed with mild acute pancreatitis because there was no evidence of persistent organ failure, pancreatic or peripancreatic necrosis, or local or systemic complications. The pancreatitis resolved with symptomatic treatment within 2 days. Removal of the IGB was recommended; however, the patient's symptoms rapidly resolved without any intervention. The patient requested the balloon be retained. A decision was made to continue monitoring the IGB without removal. One month later, however, the patient presented to the emergency department with a similar presentation. Laboratory test results and imaging findings in the emergency department were nearly identical to those during the previous admission. Further

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Received: ***May 19, 2026*** *Revision Requested:* ***July 1, 2026*** *Last Revision Received:* ***August 1, 2026*** *Accepted:* ***August 17, 2026***
Publication Date: ***September 16, 2026***
DOI: ***10.5152/tjg.2026.26350***

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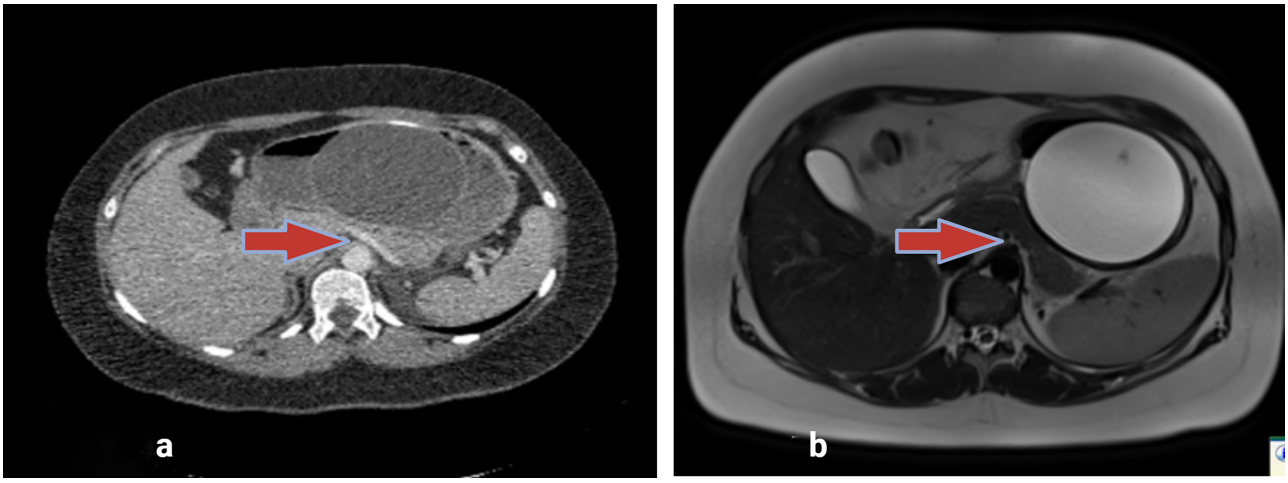

Figure 1. Pancreatic head and body compression by the IGB, with mild peripancreatic inflammation, normal biliary ducts, no local complications, and no evidence of duodenal migration on abdominal CT (A) and MRCP (B). CT, computed tomography; IGB, intragastric balloon; MRCP, magnetic resonance cholangiopancreatography.

investigations did not identify an alternative etiology for acute pancreatitis. The second episode was also classified as mild acute pancreatitis according to the Revised Atlanta Classification, as there was no evidence of persistent organ failure, pancreatic or peripancreatic necrosis, or local or systemic complications. Removal of the IGB was recommended. Following IGB removal, no recurrence of pancreatitis was observed during the 1-year follow-up period. Laboratory and radiological findings from both admissions are summarized in Supplementary Table 1. Written informed consent was obtained from the patient for publication of this case and accompanying images. PaperPal AI was used for language editing during the preparation of the manuscript.

IGB therapy has been used for nearly 30 years as an effective and minimally invasive treatment for obesity. Although the available literature on IGB-associated acute pancreatitis consists mainly of case reports and small case series, a 7-year retrospective cohort study reported an incidence of 5.56%. This is theoretically explained by the pressure of the pancreatic duct or body caused by balloon distension and migration of the catheter into the duodenum. Standard management of pancreatitis and IGB removal were required in 84% of patients. In addition, the type and brand of balloon may influence the risk of pancreatitis.[2] However, because the procedure was performed at another center, detailed information about the IGB was unavailable. Although the prognosis is favorable in most reported cases, a case of IGB-related necrotizing pancreatitis has recently been reported.[3,4]

Although no specific interval has been established between IGB placement and the development of pancreatitis, cases have been reported 1 to 11 months after the procedure. The diagnosis and treatment of acute pancreatitis following IGB placement are similar to those for acute pancreatitis of other etiologies. In a 7-year retrospective, single-center cohort study, all cases of IGB-associated pancreatitis were classified as mild according to the Revised Atlanta Classification, with a median hospital stay of 2 days. Similarly, both episodes in our case were classified as mild acute pancreatitis, with a hospital stay of 2 days.[2]

The primary clinical decision point is the timing of IGB removal, for which no clear consensus exists regarding the optimal duration. In addition to clinical status, patient and physician preferences should play an important role in decision-making. Although conservative management without balloon removal may be considered in carefully selected patients through shared decision-making, our experience highlights the need for close clinical monitoring. Given the recurrence observed in our patient, conservative follow-up without balloon removal should be approached with extreme caution.[5] Our patient was fortunate that both episodes of pancreatitis were mild. Although most reported cases in the literature are mild, patients should be counseled carefully about the potential for recurrent episodes if the IGB is not removed.

Although acute pancreatitis secondary to IGB placement appears to be a rare complication, maintaining a high level

of clinical awareness is crucial. This is particularly important because the hallmark symptoms of acute pancreatitis, such as nausea, vomiting, and abdominal pain, frequently overlap with the expected, transient side effects commonly observed after IGB placement. Acute pancreatitis can be easily underrecognized or misattributed to routine balloon accommodation, delaying timely diagnosis in mild cases. Given the increasing prevalence of obesity and the growing use of IGB therapy, clinicians should remain aware of this potential complication when evaluating patients with abdominal pain after the procedure.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that PaperPal AI was used for language editing in the preparation of the manuscript.

Informed Consent: Written informed consent was obtained from the patient who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – Z.N.H.; Design – Z.N.H., I.G.; Supervision – M.K.A., O.S.; Resources – Z.N.H., I.G.; Materials – O.B., E.A.; Data Collection and/or Processing – Z.N.H., I.G., O.B.; Analysis and/or Interpretation – Z.N.H., I.G.; Literature Search – Z.N.H., I.G.; Writing – Z.N.H., I.G.; Critical Review – O.S.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declare that this study received no financial support.

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Supplementary Table 1. Clinical, laboratory, and imaging findings during both episodes of acute pancreatitis

	First admission	First admission	Second admission	Second admission
	Hour 0	Hour 48	Hour 0	Hour 48
		(before discharge)	(1 month after the first admission)	(before discharge)
White Blood Cell (4.5-11 x10^3/μL)	9.49	8.49	9.5	7.2
Neutrophil (1.5-6.7x10^3/μL)	4.81	3.81	3.23	2.99
Lymphocyte (1.5-4 x10^3/μL)	4.02	4.02	5.35	3.31
Hemoglobin (11-17.4 g/dL)	13	11.2	13	11.4
Platelet (150-400x10^3/μL)	318	271	247	202
Blood urea nitrogen (6-24 mg/dL)	15.4	9.8	11.2	9.81
Serum Creatinine (0.7-1.2 mg/dL)	0.7	0.62	0.71	0.65
Glucose (74-100 mg/dL)	126	87	118	76
Alanine Aminotransferase(<50 U/L)	12	11	18	13
Aspartate Aminotransferase (<50 U/L)	13	10	21	14
Alkaline Phosphatase (40-150 U/L)	70	58	72	62
Gamma-glutamyl transferase (<55 U/L)	10	9	10	9
Plasma Total Bilirubin (<1.2 mg/dL)	0.51	0.83	0.43	1.29
Plasma Direct Bilirubin (<0.2 mg/dL)	0.1	0.16	0.1	0.19
Serum amylase (28-125 U/L)	439	118	662	58
Serum lipase (8-78 U/L)	1369	102	2449	28
Lactate dehydrogenase (< 248 U/L)	201	141	186	148
Serum Sodium (132-146 mEq/L)	141	140	138	135
Serum Potassium (3.7-5.4 mEq/L)	3.7	3.9	4.2	3.78
Serum Calcium (8.5-10.7 mg/dL)	9.6	8.73	8.93	8.92
Albumin (35-52 g/L)	43.5	36.94	42	36.67
CRP (0-5 mg/L)	3.6	2.4	1	4.4
Plasma Triglyceride (44-249 mg/dL)	83			
IgG4 (<135 mg/dL)	1.47			
Abdominal ultrasonography	-	-	Compression of the pancreatic head and body by the gastric balloon with mild peripancreatic inflammation and normal biliary ducts	-
Computed Tomography (Figure 1a)	Compression of the pancreatic head and body by the gastric balloon with mild peripancreatic inflammation, no evidence of duodenal migration or catheter displacement	-	-	-
MRCP (Figure 1b)	Compression of the pancreatic head and body by the gastric balloon with mild peripancreatic inflammation and normal biliary ducts	-	-	-

MRCP, magnetic resonance cholangiopancreatography.