CASE REPORT

DOI: 10.4274/nkmj.galenos.2025.99267
Nam Kem Med J 2026;14(2):222-227

Spontaneous Gastric Perforation in a Young Male with Anorexia Nervosa, Binge-eating/Purging Type: A Case Report

Tıkınırcasına Yeme/Arınma Tipi Anoreksiya Nervosa Tanılı Genç Bir Erkekte Spontan Gastrik Perforasyon: Bir Olgu Sunumu

İbrahim ALTUNDAĞ[1], Halil İbrahim EREN[2], Mehmet Taylan KOÇER[3]

[1]University of Health Sciences Türkiye, Haydarpaşa Numune Training and Research Hospital, Clinic of Emergency Medicine, İstanbul, Türkiye
[2]University of Health Sciences Türkiye, Van Training and Research Hospital, Clinic of Psychiatry, Van, Türkiye
[3]Tekirdağ Dr. İsmail Fehmi Cumalioğlu City Hospital, Clinic of Emergency Medicine, Tekirdağ, Türkiye

ABSTRACT

Bulimia nervosa (BN) is an eating disorder characterized by recurrent binge eating and compensatory behaviors. Diagnostic crossover to anorexia nervosa (AN) often occurs in chronic cases. We report a 29-year-old male with a ten-year BN history who presented with mild dyspepsia and a single episode of black-colored vomiting. Physical examination was unremarkable. However, laboratory tests revealed hypokalemia and renal dysfunction. Imaging demonstrated a 1-cm gastric perforation that required emergency surgery. Psychiatric evaluation confirmed AN-binge-eating/purging type (AN-b/p). Following surgery, combined therapy with fluoxetine and topiramate achieved clinical stabilization, promoted weight gain, and eliminated purging behaviors. This case underscores that gastric perforation, although rare, is a potentially life-threatening complication of AN-b/p. In eating disorders, altered pain perception may blunt symptom awareness, enabling serious complications to progress silently. Multidisciplinary collaboration is essential for effective treatment and recovery.

Keywords: Eating disorders, bulimia nervosa, anorexia nervosa, binge-eating/purging type, gastric dilatation, gastric perforation, male, emergency surgery

ÖZ

Bulimia nervoza (BN), tekrarlayan tıkınırcasına yeme atakları ve telafi edici davranışlarla karakterize bir yeme bozukluğudur. Kronik olgularda sıklıkla anoreksiya nervoza (AN) tanısına geçiş görülebilir. On yıllık BN öyküsü olan 29 yaş erkek hasta, hafif dispepsi ve bir kez siyah renkli kusma şikayeti ile başvurdu. Fizik muayenesi belirgin bir bulgu vermemekle birlikte, laboratuvar testlerinde hipokalemi ve böbrek fonksiyon bozukluğu saptandı. Görüntülemede 1 cm'lik mide perforasyonu tespit edilerek acil cerrahi yapıldı. Psikiyatrik değerlendirme, AN-tıkınırcasına yeme/arınma tipi (AN-b/p) tanısını doğruladı. Ameliyat sonrası fluoksetin ve topiramat tedavisi ile klinik stabilizasyon, kilo artışı ve arıtma davranışlarının sona ermesi sağlandı. Bu olgu, mide perforasyonunun nadir olmakla birlikte AN-b/p'nin potansiyel olarak hayatı tehdit eden bir komplikasyonu olduğunu vurgulamaktadır. Yeme bozukluklarındaki bozulmuş ağrı algısı, şiddetli komplikasyonların hafif veya belirsiz semptomlar altında fark edilmeden ilerlemesine yol açabilir. Etkili tedavi ve iyileşme için multidisipliner iş birliği gereklidir.

Anahtar Kelimeler: Yeme bozuklukları, bulimia nervoza, anoreksiya nervoza, tıkınırcasına yeme/arınma tipi, gastrik dilatasyon, gastrik perforasyon, erkek, acil cerrahi

Address for Correspondence: İbrahim ALTUNDAĞ MD, University of Health Sciences Türkiye, Haydarpaşa Numune Training and Research Hospital, Clinic of Emergency Medicine, İstanbul, Türkiye
E-mail: dr.ibrahimaltundag@gmail.com **ORCID ID:** orcid.org/0000-0002-0880-7218
Received: 03.07.2025 **Accepted:** 12.10.2025 **Publication Date:** 16.06.2026

Cite this article as: Altundağ İ, Eren Hİ, Koçer MT. Spontaneous gastric perforation in a young male with anorexia nervosa, binge-eating/purging type: a case report. Nam Kem Med J. 2026;14(2):222-227

INTRODUCTION

Bulimia nervosa (BN) is a severe eating disorder (ED) characterized by recurrent episodes of binge eating followed by maladaptive weight-control behaviors such as self-induced vomiting, misuse of laxatives or diuretics, fasting, or excessive exercise[1,2]. BN affects approximately 1-1.5% of women and 0.5-1% of men, accounting for a substantial share of disability-related years of life lost[3]. Failure to recognize and diagnose BN in a timely manner, particularly in men, markedly increases the likelihood of medical emergencies.

BN rarely presents alone; it frequently coexists with mood and anxiety disorders, with comorbidity rates reported as high as 70%[4]. The repetitive vomiting and purging behaviors characteristic of BN substantially increase the risk of systemic complications, including electrolyte disturbances, renal impairment, cardiomyopathy, and gastrointestinal (GI) injury[5]. BN is a well-recognized cause of GI symptoms. Acute gastric dilatation, necrosis, and spontaneous perforation, though rare, are potentially fatal complications. In the past century, fewer than 40 adult cases have been reported, most commonly in association with binge-purge episodes[6]. Mortality in BN exceeds 30%, and septic hemorrhagic shock, which often results from delayed diagnosis, is the leading cause of death[6].

Diagnostic crossover between BN and anorexia nervosa (AN) has been well documented in individuals with longstanding ED[1,7]. Persistent binge eating alongside a significantly low body mass index (BMI) is classified as AN-binge-eating/purging type (AN-b/p) according to DSM-5-TR[8]. Such transitions are often underrecognized in male patients, leading to delayed diagnosis and an increased risk of medical complications[9].

This case report highlights the clinical course and significance of a rare yet serious GI complication in a patient with a ten-year history of BN who fulfills the diagnostic criteria for AN-b/p. By assessing various clinical findings and associated risks, this report aims to enhance early diagnosis and optimize medical management strategies in EDs.

CASE REPORT

History and Physical Examination

A 29-year-old male presented to the emergency department with a three-day history of dyspepsia and a single episode of black-colored vomiting. He reported frequent episodes of self-induced vomiting to a sensation of gastric fullness, which offered him temporary relief. He had no history of abdominal pain, recent surgery, trauma, or endoscopic procedures. He has had a diagnosis of BN for the past ten years. Anthropometric measurements revealed a height of 160 cm and a weight of 40 kg (BMI: 15.6 kg/m²). His psychiatric history revealed prior treatment with fluoxetine (60-80 mg/day), sertraline (100-150 mg/day), and olanzapine (2.5-5 mg/day) at different times. These treatments provided limited benefit, likely due to inconsistent adherence, premature discontinuation, and irregular clinical follow-up. For the past six months, he has been consistently taking fluoxetine at a stable dose of 40 mg/day.

On admission, his vital signs were: body temperature 36 °C, blood pressure 95/57 mmHg, heart rate 120 bpm, respiratory rate 18 breaths/min, and SpO_2 97%. Abdominal examination revealed no signs of guarding or rebound tenderness, and rectal examination showed an empty ampulla.

Laboratory and Radiologic Findings

Laboratory investigations revealed leukocytosis (white blood cell: 12.52×10^3/μL), mild anemia (hemoglobin: 11.3 g/dL), and elevated inflammatory markers (C-reactive protein: 9 mg/L). Renal function tests showed markedly elevated urea (133 mg/dL) and creatinine (3.02 mg/dL) levels. Electrolyte analysis indicated hyponatremia (Na: 132 mEq/L) and significant hypokalemia (K: 2.57 mEq/L). Arterial blood gas analysis demonstrated alkalemia (pH: 7.51) with metabolic alkalosis (bicarbonate: 59 mEq/L) and a mildly elevated lactate level (2.2 mmol/L). Urinalysis was positive for ketones. The patient's laboratory results are presented in Table 1. Abdominal radiography revealed diffuse radiolucent air densities in the left upper and lower quadrants, while chest radiography was unremarkable with no evidence of subdiaphragmatic free air (Figure 1). Contrast-enhanced abdominal computed tomography demonstrated gastric dilatation on the coronal plane (Figure 2) and an approximately 1 cm perforation on the posterior wall of the gastric fundus, accompanied by contrast extravasation on the axial plane (Figure 3).

Mental Status Examination

On mental status examination, the patient's attention was intact, mood was mildly depressive with an appropriate and reactive affect, and no psychotic features or suicidal ideation were observed. Sleep was disturbed, while psychomotor activity remained within normal limits. The patient demonstrated a tendency to rationalize purging behavior, and insight was partially preserved. The clinical presentation was considered consistent with AN-b/p, according to DSM-5-TR criteria[1].

Psychiatric evaluation revealed that the patient was actively engaging in maladaptive eating behaviors. Despite prior therapeutic interventions, the patient had not adhered to consistent follow-up or pharmacotherapy, leading to a chronic pattern of disordered behavior. During the stabilization period, fluoxetine 40 mg/day was continued, and trazodone 25 mg at bedtime was introduced for sleep, with good tolerance.

Table 1. The patient's laboratory results at admission

Laboratory parameters	Results	Normal range
Hemogram		
Leukocytes (10^3/µL)	12.52	4-10
Neutrophils (10^3/µL)	9	2-7
Lymphocytes (10^3/µL)	2.12	0.4-7
Hemoglobin (10^3/g/dL)	11.3	12-16
Biochemistry		
Alanine aminotransferase (U/L)	18	0-50
Aspartate aminotransferase (U/L)	25	10-50
Urea (mg/dL)	133	12-43
Creatinine (mg/dL)	3.02	0.7-1.2
C-reactive protein (mg/L)	9	0-5
Sodium (mEq/L)	132	134-145
Potassium (mEq/L)	2.57	3.3-5.2
Chloride (mEq/L)	67	98-107
Calcium (mg/dL)	8.91	8.4-10.2
Albumin (g/L)	37	35-52
Amylase (U/L)	61	29-100
Lipase (U/L)	34	13-60
International normalized ratio	0.9	0.8-1.2
Blood gas		
pH	7.51	7.35-7.45
Bicarbonate (mmol/L)	59.6	22-26
Lactate (mmol/L)	2.2	0.5-2
Urinalysis		
Leukocytes	1	0-5
Erythrocytes	1	0-2
Ketones	++	Negative

Follow-up and Clinical Outcome

The patient underwent emergency laparotomy, and the gastric perforation was repaired primarily. His postoperative course remained stable, and he was discharged on the ninth day with complete surgical resolution. The patient was assessed as being at moderate risk for refeeding syndrome, and nutritional management was coordinated by the dietitian and internal medicine team with close monitoring of electrolyte levels.

Postoperatively, no recurrence of purging behaviors was observed, and treatment adherence was good. The fluoxetine dose was revised to 60 mg/day, and topiramate was added and titrated to 50 mg/day. Following the initiation of topiramate, the patient demonstrated a notable decrease in vomiting urges and a more regular pattern of food intake. The patient's BMI reached 19.3 kg/m², and he returned to work.

DISCUSSION

This case report presents the clinical course and management of gastric perforation in a patient with AN-b/p. The patient exhibited a diagnostic transition from BN to AN. Although EDs are primarily psychiatric conditions, they are often accompanied by severe medical complications. Our case involved a young male patient who presented with only mild dyspeptic symptoms. He exhibited no abdominal pain or signs of guarding that would suggest an acute abdomen. The unexpected finding of a fundic perforation underscores the importance of heightened clinical vigilance for rare but potentially life-threatening complications of AN-b/p. Gastric perforation can occur in AN—especially the AN-b/p type—due to chronic gastric hypomotility and delayed emptying, even without obvious symptoms[10,11].

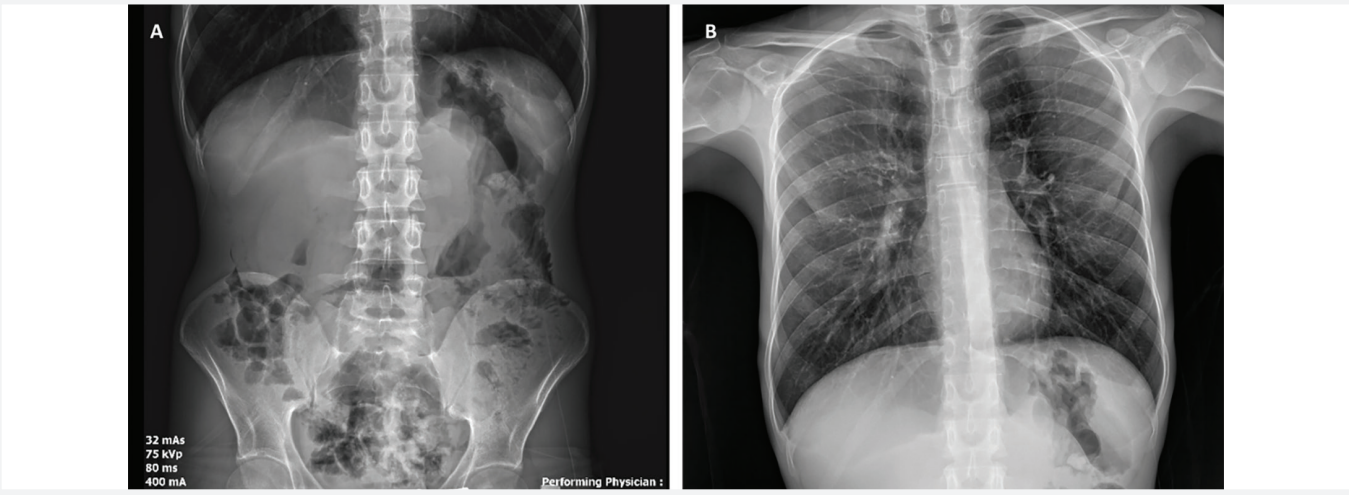

Figure 1. Radiographic findings: (A) Abdominal radiograph demonstrates diffuse radiolucent air densities in the left upper and lower quadrants, (B) Normal chest radiograph

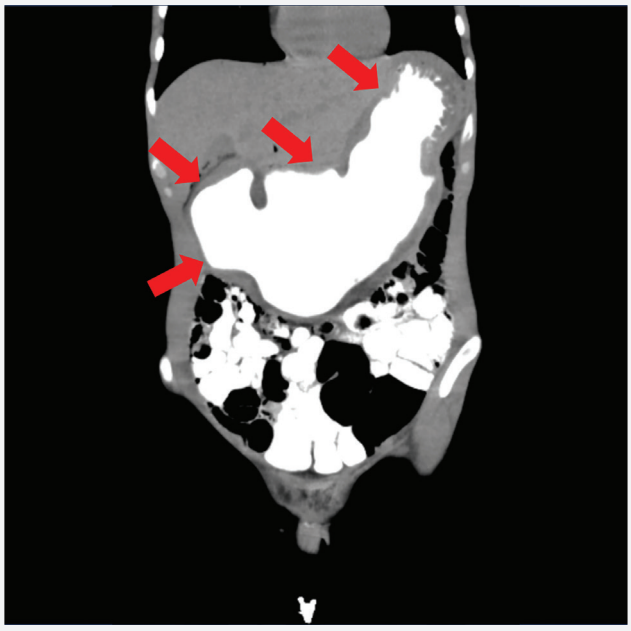

Figure 2. Abdominal computed tomography demonstrates gastric dilatation in the coronal plane (arrow)

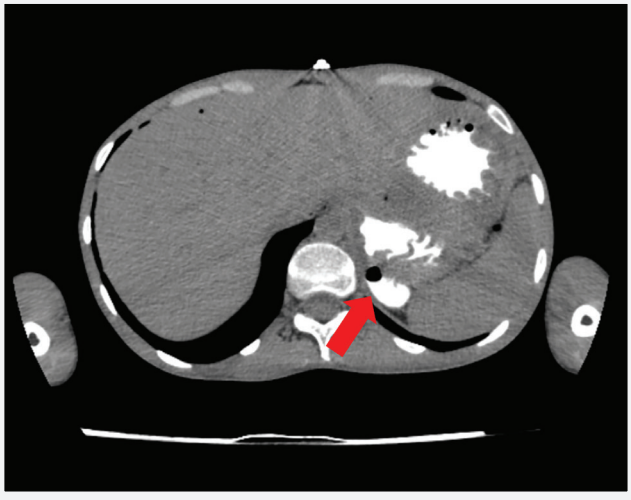

Figure 3. Abdominal computed tomography demonstrates a perforation in the posterior wall of the gastric fundus, with associated contrast extravasation and free intraperitoneal air in the axial plane (arrow)

EDs have been associated with a wide range of GI symptoms. Postprandial fullness and abdominal bloating are reported in the majority of individuals with EDs. Additionally, over half experience symptoms such as abdominal pain, gastric distension, early satiety, and nausea[12]. GI manifestations of EDs include esophageal symptoms, delayed gastric motility, functional dyspepsia, constipation, irritable bowel syndrome, rectal prolapse, hepatic failure, and even superior mesenteric artery syndrome[13]. BN also leads to complications specific to binge-purge behaviors, including recurrent vomiting and laxative misuse. Compensatory behaviors such as repeated vomiting and laxative abuse have been shown to cause electrolyte imbalances (e.g., hypokalemia, hypochloremic alkalosis), GI mucosal injury, oral pathologies, and hormonal-metabolic dysfunctions. These behaviors have also been associated with severe outcomes including esophageal rupture, gastric perforation, parotid gland hypertrophy, and even esophageal cancer[13]. In individuals with AN and low BMI, even isolated purging behaviors may provoke severe GI complications and significantly increase mortality[14]. In EDs, severe complications may develop silently, even in the absence of overt clinical symptoms. Impaired pain perception allows such complications to progress unnoticed. The pathophysiology of AN has been proposed to involve deficits in interoception, reflecting a reduced capacity to perceive and regulate internal physiological states[15]. Supporting this notion, empirical studies have shown that individuals with AN exhibit altered subjective responses to interoceptive cues, including pain[16]. Additionally, patients with AN often present with elevated levels of alexithymia, which is associated with difficulties in identifying and articulating emotions[17].

Acute gastric dilatation, a potentially fatal complication, has been reported in individuals engaging in binge-eating behavior, particularly when surgical management is delayed[18]. It has been shown that BN can rarely result in gastric perforation[6,19]. Gastric dilatation resulting from binge eating, combined with elevated intragastric pressure during vomiting, can lead to vascular insufficiency, dilation, and mucosal necrosis in the gastric fundus[20]. These factors are considered to predispose to gastric perforation. In our case, the sensation of bloating and dyspepsia preceding the vomiting episode may indicate acute gastric dilatation and act as a trigger for the compulsive purging behavior frequently observed in BN. The black-colored vomitus may further support the presence of chronic purging behavior and possible mucosal injury. Prompt recognition and timely intervention are essential to prevent severe complications. Acute gastric dilatation and rupture have also been reported in AN patients without prior binge episodes, potentially resulting from altered gastric compliance and ischemia[21].

Recent epidemiological studies estimate the prevalence of BN in men to be approximately 0.5-1%, highlighting that a substantial proportion of cases—especially among young adult males—remain undiagnosed or untreated[3].

From a psychiatric standpoint, the patient's history includes multiple trials of antidepressants and antipsychotics. However, inconsistent follow-up and poor treatment adherence have resulted in a chronic behavioral pattern. Factors including

irregular treatment attendance, premature discontinuation, and comorbid impulsivity have been reported to reduce remission rates in EDs[4]. At admission, the patient was consistently taking fluoxetine, providing stability for psychopharmacological management. During the postoperative period, pharmacological treatment for AN-b/p included a combination of fluoxetine (60 mg/day) and topiramate (50 mg/day). The use of fluoxetine in AN is off-label, with some studies suggesting potential benefits, particularly after weight restoration[22]. Topiramate is effective in controlling purging behaviors and impulsivity. A randomized, double-blind, placebo-controlled trial showed it significantly reduces binge eating and self-induced vomiting in BN patients[23]. Pharmacological management in AN-b/p requires caution because of malnutrition-related risks. Although selective serotonin reuptake inhibitors and topiramate are commonly employed, their use must be carefully monitored and individualized for each patient[10].

This case underscores the crucial need for a high index of suspicion for severe GI complications in patients with EDs, especially those presenting with the AN-b/p subtype. Despite presenting without overt abdominal symptoms, our patient developed gastric perforation—a life-threatening condition likely triggered by chronic purging and underlying gastric hypomotility. This case underscores the need to remain vigilant for severe somatic complications, even in patients with EDs who present with only mild or non-specific GI symptoms. Furthermore, the case exemplifies the complexity of managing EDs with psychiatric and medical comorbidities, emphasizing the importance of a multidisciplinary approach. Early recognition, timely surgical intervention, and coordinated psychiatric follow-up including evidence-based pharmacological treatment are essential for reducing morbidity and preventing recurrence. Clinicians should be aware that EDs can present with atypical features and carry the risk of abrupt, fatal complications even in young patients who may remain undiagnosed or undertreated.

CONCLUSION

This case additionally emphasizes the value of detailed case reporting for rare but potentially fatal complications in EDs, particularly among underrepresented groups such as male patients. By documenting the clinical course, diagnostic challenges, and successful multidisciplinary management, this report provides a reference for early recognition and intervention strategies. It reinforces the need for heightened awareness among clinicians, not only for timely surgical management but also for coordinated psychiatric care, thereby contributing to improved patient outcomes and guiding future clinical practice.

Ethics

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report and any accompanying data.

Footnotes

Authorship Contributions

Concept: İ.A., H.İ.E., M.T.K., Design: İ.A., M.T.K., Data Collection or Processing: H.İ.E., M.T.K., Analysis or Interpretation: İ.A., H.İ.E., Literature Search: H.İ.E., M.T.K., Writing: İ.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 5th ed., text revision (DSM-5-TR). American Psychiatric Association Publishing; 2022.
2. Fairburn CG, Harrison PJ. Eating disorders. Lancet. 2003;361:407-16.
3. Santomauro DF, Melen S, Mitchison D, Vos T, Whiteford H, Ferrari AJ. The hidden burden of eating disorders: an extension of estimates from the Global Burden of Disease Study 2019. Lancet Psychiatry. 2021;8:320-8.
4. Udo T, Grilo CM. Prevalence and correlates of DSM-5-defined eating disorders in a nationally representative sample of U.S. adults. Biol Psychiatry. 2018;84:345-54.
5. Westmoreland P, Krantz MJ, Mehler PS. Medical complications of anorexia nervosa and bulimia. Am J Med. 2016;129:30-7.
6. Jano F, Behr C, Almajali F, Moran V. Acute gastric dilatation complicated by necrosis and perforation following a binge eating episode. Cureus. 2022;14:e31727.
7. Eddy KT, Dorer DJ, Franko DL, Tahilani K, Thompson-Brenner H, Herzog DB. Diagnostic crossover in anorexia nervosa and bulimia nervosa: implications for DSM-V. Am J Psychiatry. 2008;165:245-50.
8. Crow SJ, Peterson CB, Swanson SA, Raymond NC, Specker S, Eckert ED, et al. Increased mortality in bulimia nervosa and other eating disorders. Am J Psychiatry. 2009;166:1342-6.
9. Nilsson K, Sundbom E, Hägglöf B. A longitudinal study of perfectionism in adolescent onset anorexia nervosa-restricting type. Eur Eat Disord Rev. 2008;16:386-94.
10. Gibson D, Workman C, Mehler PS. Medical complications of anorexia nervosa and bulimia nervosa. Psychiatr Clin North Am. 2019;42:263-74.
11. Hassan A, Lawlis S, Landmann A, Ruiz-Elizalde A, Middleman A. Not just an upset stomach: gastric perforation in a patient with anorexia nervosa. Eat Weight Disord. 2022;27:1907-11.
12. Santonicola A, Gagliardi M, Guarino MPL, Siniscalchi M, Ciacci C, Iovino P. Eating disorders and gastrointestinal diseases. Nutrients. 2019;11:3038.
13. Sato Y, Fukudo S. Gastrointestinal symptoms and disorders in patients with eating disorders. Clin J Gastroenterol. 2015;8:255-63.
14. Zipfel S, Giel KE, Bulik CM, Hay P, Schmidt U. Anorexia nervosa: aetiology, assessment, and treatment. Lancet Psychiatry. 2015;2:1099-111.
15. Pollatos O, Kurz AL, Albrecht J, Schreder T, Kleemann AM, Schöpf V, et al. Reduced perception of bodily signals in anorexia nervosa. Eat Behav. 2008;9:381-8.

16. de Zwaan M, Biener D, Bach M, Wiesnagrotzki S, Stacher G. Pain sensitivity, alexithymia, and depression in patients with eating disorders: are they related? J Psychosom Res. 1996;41:65-70.

17. Strigo IA, Matthews SC, Simmons AN, Oberndorfer T, Klabunde M, Reinhardt LE, et al. Altered insula activation during pain anticipation in individuals recovered from anorexia nervosa: evidence of interoceptive dysregulation. Int J Eat Disord. 2013;46:23-33.

18. Dincel O, Goksu M. Acute gastric dilatation due to binge eating may be fatal. North Clin Istanb. 2016;4:199-202.

19. Han YJ, Roy S, Siau AMPL, Majid A. Binge-eating and sodium bicarbonate: a potent combination for gastric rupture in adults-two case reports and a review of literature. J Eat Disord. 2022;10:157.

20. Lee JD, Prabhakaran S, Wilkie BD, Peng C, Thomson BNJ. Massive gastric distension due to bulimia nervosa: a hotpot emergency. J Surg Case Rep. 2022;2022:rjac193.

21. Panyko A, Vician M, Dubovský M. Massive acute gastric dilatation in a patient with anorexia nervosa. J Gastrointest Surg. 2021;25:856-8.

22. Marvanova M, Gramith K. Role of antidepressants in the treatment of adults with anorexia nervosa. Ment Health Clin. 2018;8:127-37.

23. Nickel C, Tritt K, Muehlbacher M, Pedrosa Gil F, Mitterlehner FO, Kaplan P, et al. Topiramate treatment in bulimia nervosa patients: a randomized, double-blind, placebo-controlled trial. Int J Eat Disord. 2005;38:295-300.