# Acute Massive Gastric Dilatation: Severe Ischemia and Gastric Necrosis without Perforation

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## Abstract

Acute massive gastric dilatation is a rare event and though it can occur in a multitude of medical conditions, its pathogenesis is still debated. It leads almost invariably to gastric necrosis with or without perforation which calls for emergency surgical treatment.

We present the case of a 22 year-old male patient of normal weight with acute massive gastric dilatation due to a binge eating episode leading to gastric parietal ischemia with mucosal necrosis. Abdominal computed tomography established the diagnosis of acute massive gastric dilatation. After partial decompression of the stomach, the patient emptied his stomach by vomiting. Eight hours after gastric decompression, an upper endoscopy was performed showing ischemia with areas of necrotic gastric mucosa in the fundus and along the greater curvature. Despite presence of ischemia and gastric necrosis, conservative treatment was successful. Psychiatric assessment revealed a borderline mentally retarded young man, but no current diagnosis of a typical eating disorder.

Physicians should be aware that binge eating habits may cause acute massive gastric dilatation in patients of normal weight who are not diagnosed as having a typical eating disorder. Prompt diagnosis of acute gastric dilatation and decompression of the stomach even when gastric ischemia and mucosal necrosis is present, may avoid unnecessary laparotomy.

## Key words

Acute massive gastric dilatation - gastric ischemia - eating habits - partial decompression - conservative treatment

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## Rezumat

Dilataţia acută gastrică masivă reprezintă un eveniment rar, întâlnit totuşi în numeroase afecţiuni, şi a cărui patogeneză este încă discutată. Dilataţia acută gastrică masivă conduce aproape invariabil la necroză gastrică cu sau fără perforaţie, ceea ce necesită tratament chirurgical de urgenţă.

Prezentăm cazul unui pacient de 22 de ani de sex masculin, cu greutate normală, cu dilataţie acută gastrică masivă survenită după un acces bulimic şi care a dezvoltat o ischemie a peretelui gastric cu necroză a mucoasei. Diagnosticul de dilataţie acută masivă a fost stabilit prin examen computer tomografic. După decomprimarea parţială a stomacului, pacientul evacuează complet stomacul prin efort de vărsătură. După 8 ore, examenul endoscopic a relevat prezenţa de arii necrotice extinse la nivelul fundusului şi marii curburi gastrice. Tratamentul conservator a avut succes, deşi au fost prezente fenomene ischemice severe parietale gastrice. Evaluarea psihiatrică a stabilit profilul unui pacient la limita retardului mental, necunoscut ca având tulburări ale obiceiurilor alimentare.

Medicul trebuie să fie conştient că un episod de bulimie poate conduce la dilataţie acută gastrică masivă chiar la pacienţi cu greutate normală şi care nu sunt cunoscuţi cu tulburări de alimentaţie. Un diagnostic prompt şi decompresiunea stomacului pot conduce la evitarea unei laparotomii inutile, chiar în prezenţa ischemiei parietale şi necrozei mucoase.

## Introduction

The incidence of acute gastric dilatation (AGD) is quite rare compared with other gastric pathologies, with relatively few references in the literature, most of them as case reports. AGD is encountered most often as a postoperative complication in abdominal surgery and in a multitude of disorders, such as anorexia and bulimia nervosa, psychogenic polyphagia, trauma, diabetes mellitus etc. (1-5). The pathogenesis of AGD is still unclear, with different theories

being postulated. The gastric reservoir is well known for its rich vascular network which generally protects it from ischemia when significant gastric distension occurs, so gastric necrosis is a very rare event (5-7). When intragastric pressure from gastric distension exceeds 20 $cmH_2O$, intramural blood flow is impaired and results in gastric ischemia and necrosis (6-8).

Acute massive gastric dilatation (AMGD) represents the extreme form of AGD. However, in the literature the limit between AGD and AMGD is not clearly described. When the stomach is extremely distended occupying the abdomen from diaphragm to pelvis and from left to right, the AGD is referred to as AMGD. Most frequently AMGD requires surgical intervention to prevent or to treat gastric necrosis (3,9). Surgery is not always necessary in AMGD. An early diagnosis with a prompt gastric decompression in the phase of parietal ischemia and mucosal necrosis may avoid an unnecessary laparotomy (3,10).

We present the case of a patient with AMGD after a binge eating episode who developed gastric ischemia with mucosal necrosis and was treated successfully by conservative measures.

## Case report

A 22 year-old male patient presented to the emergency department reporting acute-onset abdominal pain and progressively distended abdomen for the past 6 hours. The pain was described as continuous and significant in intensity. He had persistent nausea, but was unable to vomit, although he had attempted to several times. He explained that he had not had a meal for the last 40 hours. Seven hours prior to presentation, he had attempted a binge-eating episode. One hour later, the symptoms appeared. The patient had a negative past medical history. He was not taking any medication and had no known allergies. He was a smoker (15-20 cigarettes per day) and an occasional alcohol drinker.

His vital signs were normal and he had a body temperature of 38.2°C. The patient was in obvious discomfort and distress, particularly from his inability to vomit. His mental status was initially evaluated as normal. On physical examination, the abdomen was massively distended, with diffuse tenderness to palpation. A generalized tympany was elicited and bowel sounds were absent. Rectal examination revealed a distended Douglas pouch but no pain. Normal stool was present in the rectal ampulla with no blood traces.

In the emergency room, a complete blood count revealed a leucocytosis of $12.5x10^3/mm^3$, haemoglobin 15.8 g/dL, and a normal platelet count. The C reactive protein was slightly elevated - 29 mg/L. Other laboratory values were within the normal range.

Intravenous fluid replacement was immediately started and a nasogastric tube was placed without difficulty. Only 200cc of greyish semisolid material returned. A plain abdominal film showed an image consistent with a massive gastric dilatation (Fig.1). No free air in the peritoneal cavity and no bowel fluid levels were identified. Because of the uncertainty of diagnosis, an abdominal computed tomography (CT) was immediately obtained. This revealed a massive gastric dilatation with the stomach occupying the abdominal cavity entirely from diaphragm to pelvis (Fig.2). The duodenum was not dilated but presented a thickened wall. A relatively small distance between the aorta and the superior mesenteric vessels was noticed. The vessels were slightly shifted towards right. A right colonic loop was distended and small bowel loops were reduced to the pelvic area and right lower abdominal quadrant. No other lesions were detected.

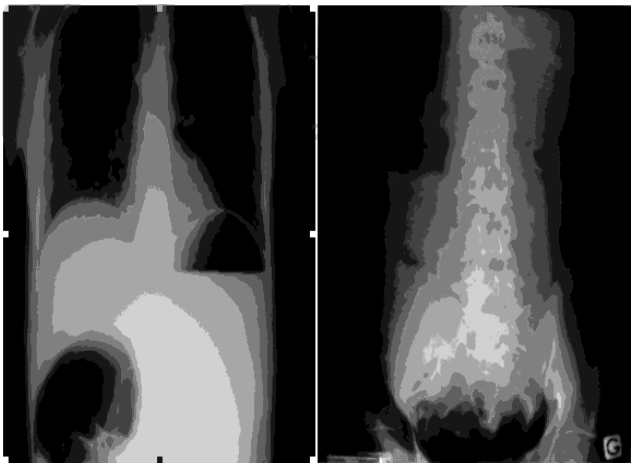

**Fig.1** Plain abdominal film suggesting massive acute gastric dilatation

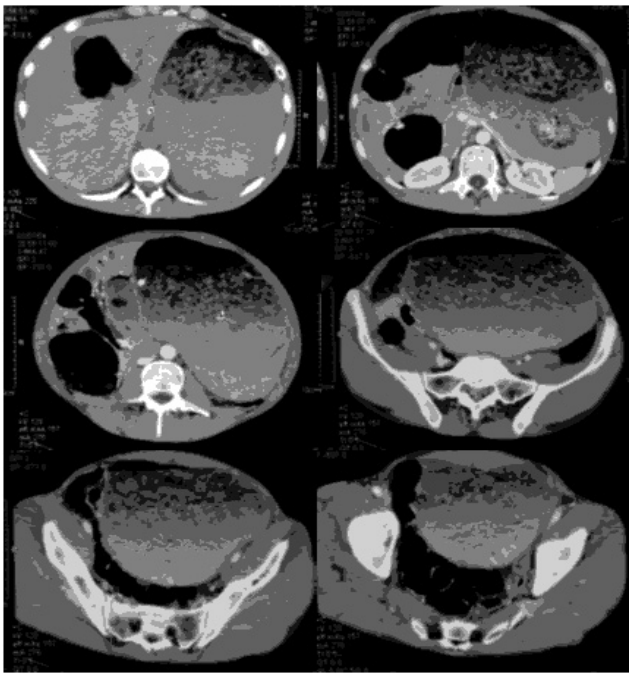

**Fig.2** Computed tomography of the abdomen shows massively dilated stomach occupying the abdominal cavity entirely from diaphragm to pelvis. The duodenum is not dilated. There is a relatively small distance between the aorta and superior mesenteric vessels. Also noted are a distended right colonic loop and small bowel loops reduced to the pelvic area and right lower abdominal quadrant.

The nasogastric tube, which was left in place, continued to be unproductive. Nine hours after the onset of symptoms, the patient was taken to the operative theatre and under anaesthesiologist supervision, a large Faucher tube was inserted with the intent to evacuate and to lavage the stomach. Despite these efforts, only 1000cc of the same greyish semisolid material and a few food particles were evacuated. The Faucher tube was removed and a standard nasogastric tube was returned in place. The abdomen remained distended, but the patient had less pain and felt some relief after the lavage manoeuvre.

Because the patient was stable and showed no signs of peritonitis, he was admitted to the ward with the intent to repeat the gastric lavage. One hour later, the patient started vomiting. More than 8000cc of foul smelling food particles were evacuated. After that, the abdomen became soft and the patient had less pain, but he continued to complain of persistent nausea and abdominal discomfort.

Eight hours after the emetic episode, an upper endoscopy was performed. Multiple areas of mucosal necrosis (the largest area was 3.5 cm in diameter) were found along the greater curvature and gastric fundus. The rest of the mucosa was pale. We believe that this represented mucosal necrosis and severe ischemia of the remaining gastric layers, but not gangrene. Conservative care was continued, which included nasogastric drainage, total parenteral nutrition, and intravenous antibiotics.

His condition gradually improved and an upper gastrointestinal study performed with Gastrografin® 72 hours after admission showed free passage through the stomach and duodenum without extravasation. The stomach appeared slightly enlarged, the duodenum was not dilated, nor were there signs of a superior mesenteric artery syndrome. A liquid diet was started and the patient was discharged from the hospital. A psychiatric evaluation one month later revealed the patient to have the mental capacity of a poorly functioning, mentally-challenged individual. The psychiatric interview also revealed two previous bulimic episodes that were spontaneously resolved after vomiting.

## Discussion

Acute gastric dilatation was first described by Duplay in 1833 (4). Although it may occur in a multitude of medical conditions, most of the reported cases are postoperative complications (1,2). Other causes include: anorexia nervosa and bulimia, psychogenic polyphagia, diabetes mellitus, trauma, electrolyte disturbances, gastric volvulus, spinal conditions etc. (3-9). The pathogenesis of AGD is still debated as several theories have been postulated. Due to its frequency as a postoperative complication, Morris et al. claimed that anesthesia and debilitation may be predisposing factors. These factors can cause relaxation of the upper esophageal sphincter with aerophagia leading to gastric distention (1,2,9).

The atonic theory was introduced in 1859 by Brinton and sustained by others (9). In patients with eating disorders, the stomach undergoes atony and muscular atrophy during a period of starvation, so that a sudden ingestion of food overtaxes an already weakened stomach. A mechanical theory, commonly known as superior mesenteric artery syndrome (SMAS), was proposed by von Rokitansky in 1861 in which AGD follows vascular compression of the third segment of the duodenum, between superior mesenteric artery, aorta, and vertebral column (3). Other authors suggest that the AGD may be a functional entity secondary to regional diseases, such as pancreatitis, peptic ulcer, gallbladder disease, appendicitis etc. (11,12).

Acute gastric dilatation in eating disorders is reported as occurring in patients with binge eating and drinking habits associated with anorexia nervosa and bulimia or during refeeding when body mass index is low (3). Psychogenic disturbances, specifically those related to abnormal eating habits, have been also stressed as important etiological factors in precipitating AGD (13,14). Five cases of AGD in patients with eating disorders and normal body weight were reported in the literature (10,14-17). All patients reported recurrent vomiting prior to the AGD, two were anorectic, and three were bulimic. Our patient had a normal body weight, he was not anorectic, and did not have a diagnosis of an eating disorder. Only one case of AGD due to abnormal binge-eating habits in a subject who does not have a diagnosis of a typical eating disorder has been reported (10), this being to the best of our knowledge, the second case. Probably, borderline mental retardation was the primary cause of the binge eating episode in our patient. The 40 hour starvation period induced atony of the stomach which was overtaxed by the rapidly ingested large quantity of food and this resulted in AMGD.

Studies of gastric volume in bodies placed in a sitting position have found the regular occurrence of tears in the mucous membranes along the lesser curvature after the administration of 4L (16). Revilloid demonstrated also, that 4L is the capacity of the stomach before perforation occurs (13). It is well known that the stomach is very resistant to ischemia due to its rich blood supply and its extensive intramural anastomoses. Experimental studies have shown that both arterial and venous circulation must be interrupted before gastric ischemia and necrosis can occur (1,2,9,14,18). Increased intragastric pressure over 20 $cmH_2O$, which exceeds venous pressure, results in mucosal necrosis. In cases of AMGD, intragastric pressure usually exceeds 30 $cmH_2O$ and produces a dramatic decrease of intramural blood flow, with necrosis and perforation usually following (19). In the majority of the cases, the necrosis that occurs along the greater curvature and gastric fundus is significant and requires emergent treatment (13). Ischemia generally occurs before perforation and mucosal necrosis before full-thickness gastric necrosis. If the diagnosis is established in an early stage, surgery may be avoided. What is interesting in our case is that the stomach was massively distended with over 9000 cc for about 12 hours; however, no full-

thickness necrosis or perforation occured. We were able to find only two cases reported in the literature in which 7000 cc of gastric fluid were aspirated without rupture of the stomach (9,20).

Clinically, emesis is a symptom present in more than 90% of cases of AGD (21). Another sign reported in the literature is the inability to vomit, which was present in our case. Why these patients are not able to vomit is not fully understood. It has been suggested that this may be due to the occlusion of the gastroesophageal junction by the distended fundus, which angulates the esophagus against the right crus of the diaphragm, producing a one-way valve (22). Significant, diffuse abdominal distension accompanied by abdominal pain is common. However, pain is sometimes mild in intensity in contrast with the massively distended abdomen (21)

On physical examination, a diffuse tympany on palpation, a splash on percussion, and a distended Douglas pouch may be found (2,21). An accurate history is important in such patients, but may be difficult when mental problems are present, so the physician must rely only on physical examination.

Plain abdominal films may differentiate a diagnosis when a fluid level in a markedly distended stomach is present (3, 21). The most useful diagnostic investigation is an abdominal CT scan that can clearly demonstrate gastric distension (3). CT scan may reveal also a cause such as SMAS or associated abdominal pathologies. A small distance between the aorta and superior mesenteric vessels as well as a distended proximal duodenum are in favor of a SMAS (3,23). The association between SMAS and psychogenic disturbances was emphasized by many authors (3,24,25). A SMAS may be precipitated by a binge-eating episode leading to an AGD. Symptoms may be regarded as "psychogenic" and part of the mental disorder so that the diagnosis of SMAS is not entertained. This is why SMAS must be searched in all patients with mental disorders who develop AGD. Upper contrast gastrointestinal examination must be performed carefully with a water soluble material as perforation is always a possibility in AMGD. Endoscopy is frequently necessary to rule out mechanical causes of obstruction such as tumors, webs, or peptic ulcer disease (26). Endoscopy is extremely important because it can show the general status of the gastric mucosa.

First line treatment for AMGD as for AGD consists of nasogastric decompression and fluid resuscitation. A normal size nasogastric tube may prove to be inefficient, as was the case with our patient. A large Faucher or Edlich tube under anaesthesiologist supervision in the operating room must be placed to ensure adequate gastric empting. Sometimes, when semisolid material is present in the stomach, even a large tube may be inefficient. If total gastric empting is not possible, a partial decompression may help because it can decrease the intragastric pressure and reduce the risk of necrosis and perforation. It also may allow vomiting as it may free the gastroesophageal junction obstruction, as was the case for our patient.

If conservative measures fail or gastric infarction with or without perforation is suspected, immediate surgical intervention is mandatory (9). If gastric necrosis or perforation is not recognized and the treatment is delayed, an 80% mortality has been reported (2,27). When endoscopy reveals areas of necrosis and no signs of peritonitis are present, a conservative treatment may be attempted. For the majority of cases of AMGD reported in the literature, the treatment consisted of a laparotomy, and according to the findings, surgical decompression, partial, or total resection of the stomach was performed (4,5,10,14,23). In our case, the decision was made to continue non-operative treatment, which included nasogastric aspiration, intravenous fluids including total parenteral nutrition, intravenous antibiotics, and meticulous monitoring of the patient, to detect as early as possible signs suggestive of perforation or sepsis.

When the acute phase subsides, we advise a psychological and psychiatric examination of all patients, especially when an organic cause of gastric dilatation has not been identified.

To the best of our knowledge, we are the first to present a case of AMGD due to abnormal binge-eating habits with significant gastric ischemia and mucosal necrosis, as demonstrated by endoscopy, and treated successfully by conservative measures. Physicians should be aware that binge eating habits may cause AMGD in patients of normal weight who are not diagnosed as having a typical eating disorder. A high index of suspicion is necessary to diagnose this rare, rapidly progressive and potentially fatal condition. CT scan examination is the diagnostic modality of choice. Severe ischemia with extensive mucosal necrosis in AMGD is not always an indication for surgery. Prompt treatment with even partial decompression, fluid resuscitation, intravenous antibiotics, and close monitoring may avoid unnecessary surgery.

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