IDIOPATHIC PARTIAL GASTRIC NECROSIS: A CASE REPORT

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ABSTRACT

Objective: *In the present case report, we emphasise that gastric necroses should be considered among acute abdominal syndromes, regardless of the aetiology. No clinical, radiological, or pathological finding to explain the gastric necrosis can be detected in some cases. Such cases are referred to as idiopathic gastric necrosis.*

Case: *A 62-year-old male patient who had stomach ache, nausea, and vomiting for 7 days was evaluated in the emergency ward. It was ascertained from the history of the patient that he had diabetes, hypertension, chronic obstructive pulmonary disease, and below-the-knee dry gangrene. Because standing direct abdominal radiography revealed free air under the diaphragm, and computed tomography revealed generalised intra-abdominal fluid and air, the patient was explored with a pre-diagnosis of organ perforation. A total gastric resection and Roux-en-Y oesophagojejunostomy were performed on the patient.*

Discussion: *Gastric necrosis is a rare and potentially fatal condition. Several aetiological factors have been identified. Vascular causes may include arterial embolism-thrombosis, and vasculitides and diabetes due to autoimmune diseases resulting in microcirculatory disorders. Diabetes can affect multiple systems. Especially in diabetic patients, the rate for peripheral vascular disease increases in proportion to the duration. The impaired microcirculation in diabetic patients due to microangiopathy reduces the blood supply to tissues. Tissue necrosis, the end-point of damage, is due to the reduced perfusion.*

Conclusion: *In the present case report, we emphasise that gastric necroses should be considered among acute abdominal syndromes, regardless of the aetiology, which can be recovered with early intervention, but which have a mortality of up to 90% when intervention is delayed.*

Keywords: *Stomach, necrosis, diabetes mellitus.* **Nobel Med 2017; 13(1): 70-73**

ÖZET

Amaç: Bu olgu sunumunda, gastric nekrozun akut karın sendromları arasında etyolojiye bakılmaksızın, düşünülmesi gerektiğini vurgulamak istedik. Bazı durumlarda gastrik nekrozu açıklamak için klinik, patolojik ve radyolojik bulgular olmayabilir. Böyle vakalar idiopatik gastrik nekrozis olarak belirtilir.

Olgu: Acil serviste, 62 yaşında bir erkek hasta 7 gün süren mide ağrısı, bulantı ve kusma nedeniyle değerlendirildi. Hastanın hikayesinden diyabet, hipertansiyon, kronik obstrüktif akciğer hastalığı ve diz altı kuru gangreninin olduğu saptandı. Ayakta direkt batın grafisinde diafragma altında serbest hava, tomografide batın içinde yaygın mayi ve hava olması nedeniyle organ perforasyonu ön tanısı ile ameliyat edildi. Hastaya total mide rezeksiyonu özefago-jejunostomi yapıldı.

Tartışma: Mide nekrozu nadir ve ölümcül bir durumdur. Farklı etyolojik durumlar saptanır. Mikro dolaşım bozukluğu ile sonuçlanan otoimmün hastalıkları, diyabet, vaskülitler ve arteriyel trombo-emboliler neden olabilir. Diabet bir çok sistemi etkiler. Özellikle diyabetik hastalarda, periferik vasküler hastalık oranı süreye bağlı olarak artar. Özellikle diyabetik hastalarda mikro dolaşım bozulduğu için mikroanjiopatiden dolayı kan dolaşımı azalır. Azalan dolaşımdan dolayı son nokta doku nekrozudur.

Sonuç: Biz bu olgu sunumunda, gastric nekrozun akut karın nedenleri arasında düşünülmesi gerektiğini vurgulamak istedik. Erken fark edilenler iyileşirken, geç kalındığında vakaların %90'ı ölümcül seyreder.

Anahtar kelimeler: Mide, nekroz, diabetes mellitus.
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INTRODUCTION

Total or partial gastric necrosis is a rare acute abdominal syndrome with an unknown cause. The stomach is known to be rich in vascular and collateral circulation. Gastric necrosis may occur due to vascular or mechanical factors or to the intake of infectious, toxic, or caustic agents.[1] No clinical, radiological, or pathological finding to explain the gastric necrosis can be detected in some cases. Such cases are referred to as idiopathic gastric necrosis.

CASE

A 62-year-old male patient who had stomach ache, nausea, and vomiting for 7 days was evaluated in the emergency ward. It was ascertained from the history of the patient that he had diabetes, hypertension, chronic obstructive pulmonary disease, and below-the-knee dry gangrene. From an examination of the patient, who had no history of caustic agent intake, drug intoxication, or previous abdominal or cardiac surgery, the overall condition of the patient was moderate, cooperative, and oriented, and the abdominal examination revealed generalised sensitivity. His arterial blood pressure was 90/60 mmHg and his pulse was 112 beats/min. From laboratory tests, the white blood cell count was 32,000 K/µL and haemoglobin was 8.4 g/dL. Because plain abdominal x-ray revealed free air under the diaphragm, and computed tomography revealed generalised intra-abdominal fluid and air, the patient was explored with a pre-diagnosis of organ perforation and areas of necrosis were detected between the fundus and antrum along the greater curvature on the anterior surface of the stomach (Figure 1,2). When the posterior surface of the stomach was examined by opening the gastrocolic ligament, a portion of the fundus and antrum had full-thickness necrosis and the presence of generalised intra-abdominal necrotic fluid was observed (Figure 3). There was no volvulus or internal herniation due to diaphragm defects, omental adhesions, or obvious cause of necrosis. Pulsation was obtained from the proximal branches of the celiac truncus and superior mesenteric artery. A total gastric resection and Roux-en-Y oesophagojejunostomy was performed on the patient. The patient, who was followed-up in intensive care with no surgical problem, was initiated on enteral feeding through a nasogastric tube on postoperative day 7. The histopathological results showed acute necrotising gastritis. The patient, who was intubated, was followed in intensive care due to pulmonary problems, and was lost on postoperative day 18 due to cardiopulmonary arrest.

DISCUSSION

Gastric necrosis is a rare and potentially fatal condition.[1,2] Several aetiological factors have been identified. The mechanical factors include gastric volvulus, intrathoracic herniation of the stomach due to diaphragmatic pathologies, and acute gastric dilatation. Acute gastric dilatation may be due to many reasons, such as eating disorders, Prader-Willi syndrome (PWS), resuscitation trauma, hiatal hernia, volvulus, electrolyte disorders, superior mesenteric artery syndrome, muscular dystrophy, psychogenic polyphagia, and anorexia nervosa.[1,3,4] Clinically, the primary complaint in most cases is vomiting, which is accompanied by abdominal

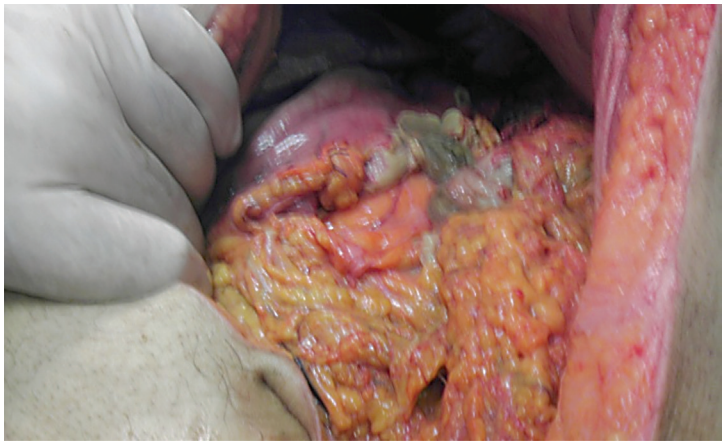

Figure 1. Greater curvature and gastrocolic ligament

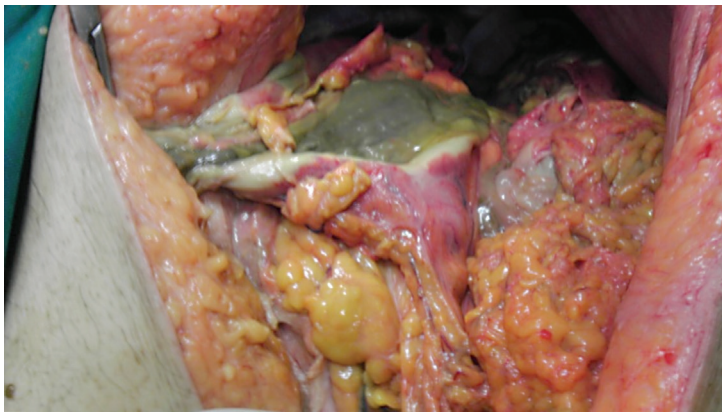

Figure 2. Greater curvature

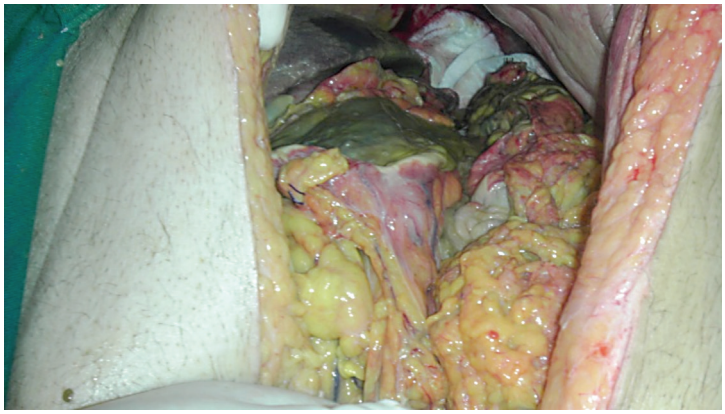

Figure 3. Gastric corpus posterior surface

distension and pain.[5] If not diagnosed early, this may result in gastric perforation, bleeding, and other serious complications.[1] Mortality is 80-100% when ischaemia and perforation develop as a result of gastric dilatation.[6] A normal stomach may hold ~4 L of fluid. The gastric volume may reach up to 15 L in people with some eating disorders, psychogenic polyphagia, bulimia, and PWS.[1] The increased pressure of the stomach leads to ischaemia. Rapidly developing acute gastric distension reduces the return of blood supply associated with vena cava compression. As a result, hypotension occurs.[7] Gastric dilatation was reported to occur as a result of direct neurogenic paralysis associated with malnutrition in psychological patients, such as those with anorexia nervosa.[3]

Many reports demonstrate that ischaemic modifications due to gastric dilatation occur mostly in the greater curvature of the stomach and less frequently in the pyloric region as generalised mucosal infarcts and multifocal transmural necrosis.[8] Ischaemia may occur in the gastric wall in necrotising gastritis, which emerges as a serious outcome of infectious gastritis. The stomach is very oedematous and there is generally diffuse submucosal involvement.[9]

The aetiology and the mechanism may be partially explained by the intake of a caustic agent with the intent of committing suicide in the partially intrathoracic localised stomach due to diaphragmatic herniation, necrosis of the remnant stomach in patients who have had gastric surgery in gastric necrosis due to analgesic abuse, or necroses following operations such as intramucosal sclerotherapy, percutaneous endoscopic gastrostomy, and laparoscopic gastric banding, due to stomach-based gastrointestinal bleeding.[2,3]

Vascular causes may include arterial embolism-thrombosis, and vasculitides and diabetes due to autoimmune diseases resulting in microcirculatory disorders. The aetiology of vasculitides is highly variable. It is important to determine whether the disease is systemic. There can be involvement of the medium and large vessels in polyarteritis nodosa (PAN), Kawasaki disease, temporal arteritis, and Takayasu disease. Henoch-Schönlein purpura primarily affects the small vessels of the skin, gastrointestinal system, and organs, such as the kidneys. Diabetes can affect multiple systems. Especially in diabetic patients, the rate for peripheral vascular disease increases in proportion to the duration. Generally, 15% of patients experience occlusive problems within 10 years following the diagnosis. These problems are experienced by almost 50% of patients within 20 years.[7] The impaired microcirculation in diabetic patients due to microangiopathy reduces the blood supply to tissues. Tissue necrosis, the end-point of damage, is due to the reduced perfusion. This occurs secondarily to the progressive development of obstructive arterial disease in medium and small arteries. In the past, the vascular pathology in diabetic patients has been considered to be at the microvascular level. However, this opinion has changed. There is obstruction in the major vessels as well. The main cause originates from atherosclerosis.[7] Small intestinal ischaemia and colonic ischaemia or necrosis are common in diabetic patients. It is known

that the arterial and collateral blood supply of the stomach always protects the organ. Thus, a very severe obstruction is required in the arterial collateral flow for the occurrence of gastric necrosis.[10] In our case, vascular pathology was not observed and as result of our tests and during the operation. As a histopathologic result, it was reported that there was no finding of embolism, thrombosis, or vasculitis beyond finding of gastric necrosis.

Although the lack of polyphagia and polydipsia in the anamnesis and no history of drug use, except an antidiabetics, led us to consider infectious. Diffuse and mucosa-based necrosis being at the forefront in necrotising gastritis and the presence of areas of partial and full-thickness necrosis in our case, the non-growth in the culture antibiogram resulted in excluding infectious causes and the case was diagnosed as partial gastric necrosis.

CONCLUSION

Total or partial gastric necrosis is a rare cause of acute abdomen. It may cause acute abdomen due to chemical reasons, secondary to vascular or mechanical reasons, or to the intake of infectious, toxic, or caustic agents; however, there may not be an underlying cause. In the present case report, we emphasise that gastric necroses should be considered among acute abdominal syndromes, regardless of the aetiology, which can be recovered with early intervention, but which have a mortality of up to 90% when intervention is delayed.

*The authors declare that there are no conflicts of interest.

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