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Gastric Necrosis, A Rare Disease with a Complex Diagnostic Approach

Rodríguez Correa Gustavo Iván[1], Carrillo Hernández Melissa[1], Gutiérrez Vargas Haydee Carolina[1], Macías Durón Emmanuel Salomón[2], Ponce Campos Silvia Denise[3] and Nieves Arellano Athenea[4*]

[1]Department of Internal Medicine, General Hospital ISSSTE Aguascalientes, Mexico

[2]Department of Radiology, Centenary Hospital Miguel Hidalgo Aguascalientes, Mexico

[3]Department de Pneumology, General Hospital ISSSTE Aguascalientes, Mexico

[4]Department of General Surgery, General Hospital ISSSTE Aguascalientes, Mexico

ABSTRACT

Gastric necrosis represents a rare condition and available literature is mostly in the form of case-reports being clinically under-recognized and therefore not diagnosed at an early stage. It is also a disease for which a definitive treatment has not yet been established since it can vary significantly from a conservative medical management to and aggressive surgical management, depending on the patient´s general conditions and medical history.

We describe a case of gastric necrosis presented at both departments of Internal Medicine and General Surgery. We discuss possible etiology, in this case found to be multifactorial due to angiotomography ruling out a proximal occlusive event and the outcome of the patient.

Conclusions: The diagnostic approach of patients presenting gastric necrosis is complex since etiology is not well established and is usually multifactorial. It continues to be a disease which we know very little about, and which represents a challenge to overcome to offer specialized treatment and reduce the morbidity and high mortality that it represents.

***Corresponding author**

Nieves Arellano Athenea, Department of General Surgery, General Hospital ISSSTE Aguascalientes, Mexico.

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Abbreviations

mg/dl: Milligrams/Deciliter
mmHg: Millimeters of Mercury
RR Interval: Time Elapsed Between Two Successive R Waves of The QRS Signal on the Electrocardiogram
aVR: Augmented Unipolar Right Arm Lead
CT: Computed Tomography
l/min: Liters/Minute
PCR: Polymerase Chain Reaction
ESR: Erythrocyte Sedimentation Rate.

Introduction

Gastric necrosis represents a rare entity due to the high vascularity of the stomach, given by collateral branches and by its own arteries. The arterial supply is given mainly form the celiac trunk through the left gastric artery, the right gastric artery (which comes from the common hepatic artery), left and right gastroepiploic arteries and short gastric arteries [1]. Although rare, the associated mortality is up to 30-40% when presented [2]. The etiology can be divided into two major groups in relation with alterations of gastric perfusion: systemic hypoperfusion and splanchnic hypoperfusion. Among the first causes we find shock and sepsis. In the second group we find previously case-described causes, being gastric volvulus, intrathoracic gastric hernia and massive gastric dilation due to mechanical factors, such as obstruction, pyloric stenosis or atonicity from electrolyte imbalance and anorexia nervosa [3-5]. Likewise, endoscopic interventions such as submucosal dissection and treatment with sclerotherapy; embolism, vasculitis and vascular ligation [6]. Among described risk factors are smoking, atherosclerosis, diabetes mellitus, advanced age and hypertension, which increase the risk of vasculopathy and gastropathy [2]. Any part of the stomach can be affected; however, previously described segments are the greater curvature, the posterior wall and the fundus [6]. Among clinical manifestations are abdominal pain and distension, nausea and vomiting, and bleeding form the upper digestive tract. Abdominal computed tomography may show gastric pneumatosis, perigastric venous gas, and gas present in the portal hepatic system. Endoscopy can be both diagnostic and therapeutic, and is not currently contraindicated in the presence of gastric pneumatosis. Common findings by this method are congestion, paleness, erythematous and ulceration of the mucosa [6-8]. The current treatment can either be surgical, performing

partial or total gastrectomy; or medical, with the administration of solutions being the cornerstone, placement of nasogastric tube for gastric decompression and to prevent aspiration, proton pump inhibitor therapy at higher doses and broad-spectrum antibiotics [6-8].

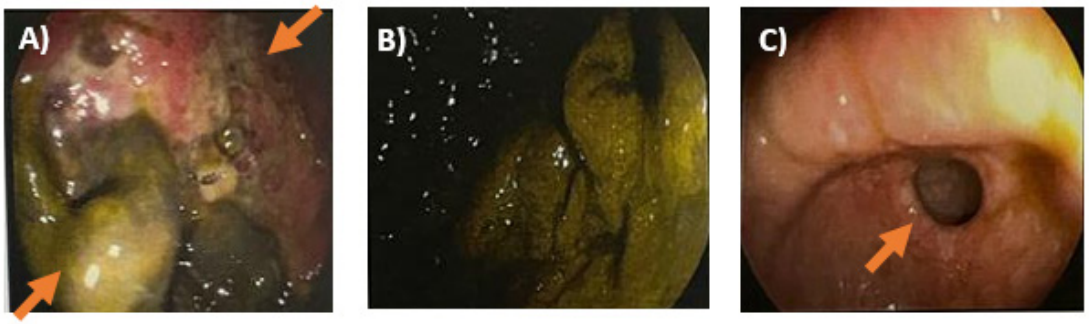
Case Presentation

An 89-year-old masculine with a medical history of long-standing systemic arterial hypertension in treatment with telmisartan, who begins his current illness the day before his hospitalization with general malaise, dyspnea and vomiting coffee grounds. He is admitted in a poor general condition, disoriented, Glasgow coma scale of 14 (eye-opening 4, verbal 5, motor 6), shallow breathing, polypnea, tachypnea, decreased thoracic excursion, decreased respiratory sounds in the bilateral subscapular region, thick rales in the bilateral interscapulovertebral region, integrating pleural effusion syndrome, saturation by pulse oximetry at 92% with 3 l/min, heart sounds with the presence of a systolic murmur in the aortic area, muscle strength 3/5 by Daniels scale, glucose test at 200 mg/dl. Endoscopy was requested due to signs of upper digestive tract bleeding, finding severe erosive gastropathy of the upper body and fundus with mucosal necrosis. Subsequent evaluation by general surgery service was performed which decides is not a candidate for surgery due to hemodynamic instability with high-dose vasopressor, not exceeding mean blood pressure of 58 mmHg. The patient required vasoactive amines for several days without improvement in blood pressure or hemodynamic status, he also had contraindication for enteral nutrition so parenteral nutrition was initiated. He developed supraventricular tachycardia with an electrocardiogram that showed a negative P wave in aVR, regular RR interval, 160 heart rate, normal axis; treated with amiodarone and verapamil without response and with high oxygen requirements. Due to the above, family members requested adequation of therapeutic effort.

Laboratory, CT scan, CT angiography and endoscopy findings are presented in attached tables and images.

Table 1

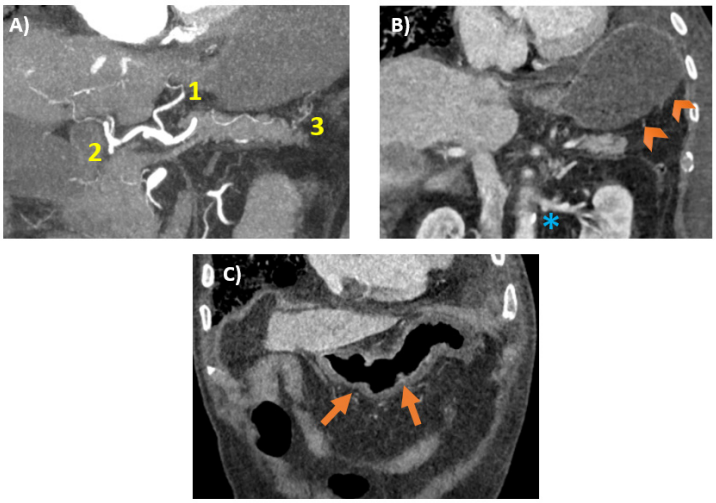
ENDOSCOPY IMAGES



Endoscopy Report
A and B. Mucosa of the entire fundus, body, both curvatures (lesser and greater) and anterior and posterior walls up to the middle body with necrosis and its border to the distal body with fibrin.
C. Duodenum without alterations.

Table 2

CT ANGIOGRAPHY IMAGES



CT Angiography
A. Coronal plane in arterial phase showing permeable left gastric artery (1), gastroduodenal artery (2) and gastro omental artery (3).
B and C. Coronal plane in venous phase showing a calcified atheromatous plaque in the abdominal aorta (asterisk), thinning and lack of enhancement in the fundus and gastric body wall (arrowheads in B) compared to the antrum and pylorus (arrows in C). No gastric or portal pneumatosis is observed.

Table 3

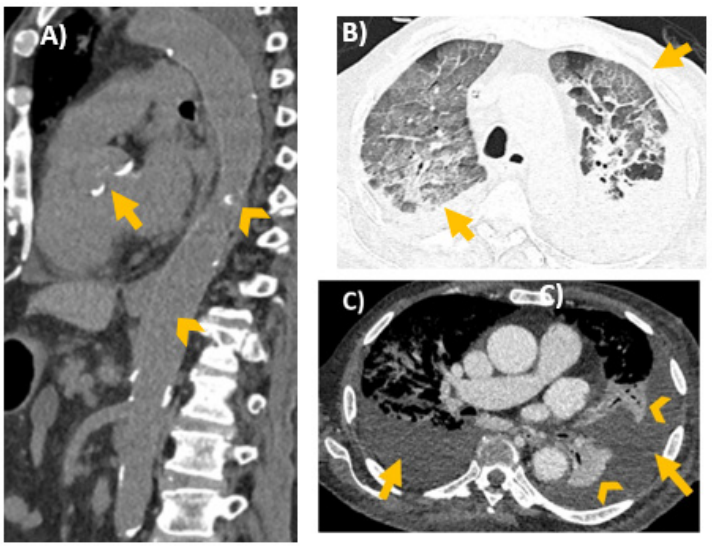
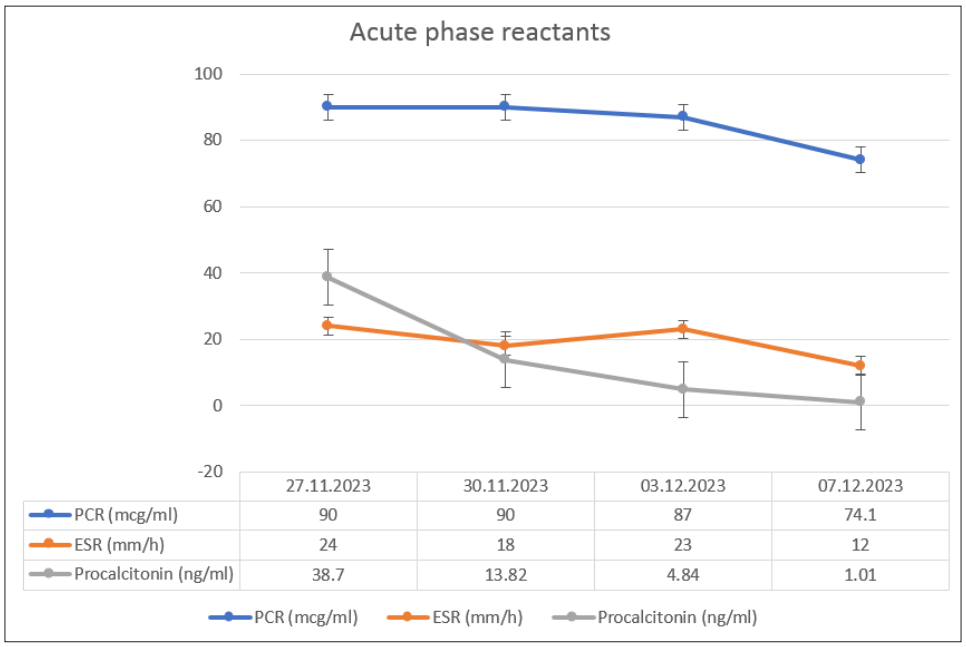
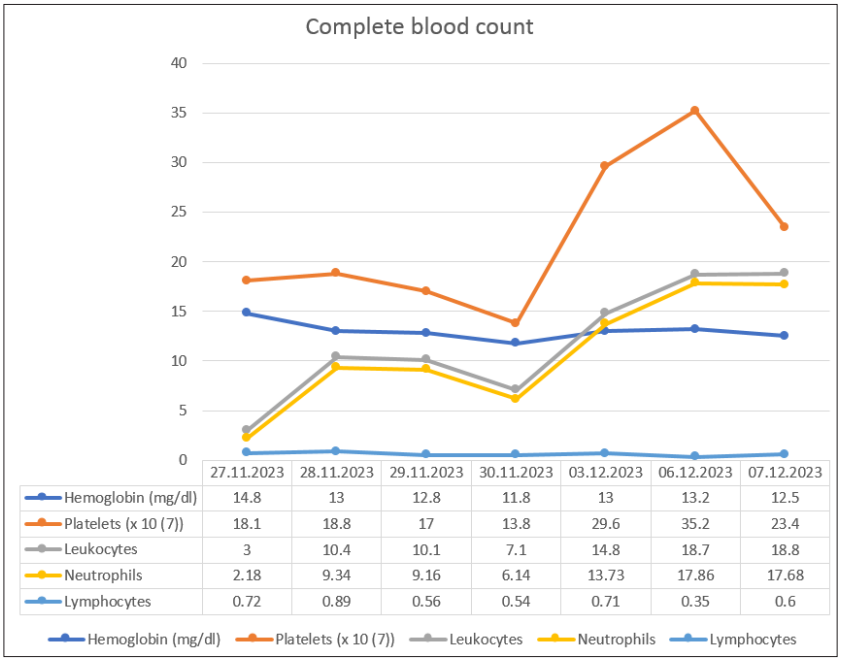
CT IMAGES

CT Scan A. Sagittal plane at midline. Multiple calcified atheromatous plaques in the aorta (arrowheads) and aortic valve with calcifications (arrow). B. Axial plane in pulmonary window. Bilateral diffuse ¨crazy paving¨ pattern (arrows) predominantly in the axial interstice, respecting the subpleural interstice. C. Contrasted in venous phase, axial plane in mediastinal window. Bilateral pleural effusion (arrows) associated with passive atelectasis (arrowheads).

Table 4: Depicts acute phase reactants evolution. An increase above reference values is observed in acute phase reactants upon admission and a progressive decrease in them after the beginning of antibiotic treatment due to septic shock.



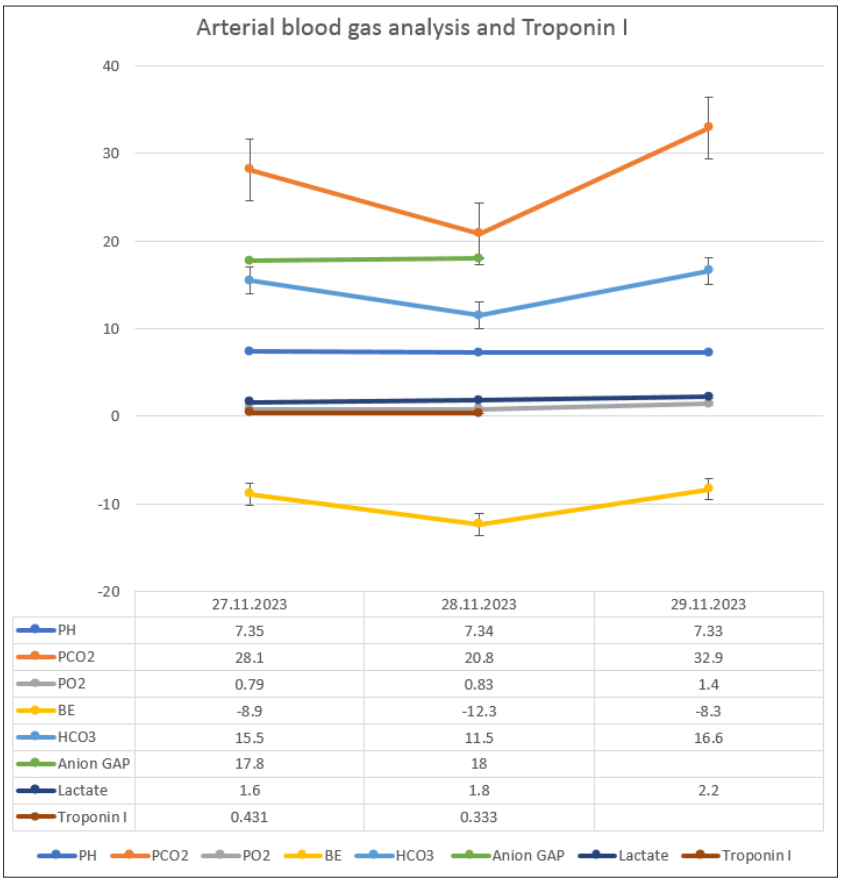
	27.11.2023	30.11.2023	03.12.2023	07.12.2023
PCR (mcg/ml)	90	90	87	74.1
ESR (mm/h)	24	18	23	12
Procalcitonin (ng/ml)	38.7	13.82	4.84	1.01

Table 5: Depicts the evolution of the components of various blood counts. No decrease in hemoglobin levels was observed upon admission despite initial data of gastrointestinal bleeding and there was no significant decrease in the following days. Leukocytosis with progressive neutrophilia was observed while clinical manifestations of septic shock developed. Platelet count was maintained at reference values.



	27.11.2023	28.11.2023	29.11.2023	30.11.2023	03.12.2023	06.12.2023	07.12.2023
Hemoglobin (mg/dl)	14.8	13	12.8	11.8	13	13.2	12.5
Platelets (x 10 (7))	18.1	18.8	17	13.8	29.6	35.2	23.4
Leukocytes	3	10.4	10.1	7.1	14.8	18.7	18.8
Neutrophils	2.18	9.34	9.16	6.14	13.73	17.86	17.68
Lymphocytes	0.72	0.89	0.56	0.54	0.71	0.35	0.6

Table 6: Depicts the evolution of the components of various arterial blood gas analysis and troponin I. A high anion gap metabolic acidosis was established upon admission associated with progressive increase in lactate secondary to septic shock. Troponin I levels were found above reference but with progressive decrease despite the episode of supraventricular tachycardia.



	27.11.2023	28.11.2023	29.11.2023
PH	7.35	7.34	7.33
PCO2	28.1	20.8	32.9
PO2	0.79	0.83	1.4
BE	-8.9	-12.3	-8.3
HCO3	15.5	11.5	16.6
Anion GAP	17.8	18	
Lactate	1.6	1.8	2.2
Troponin I	0.431	0.333	

Table 6: Depicts the evolution of the components of various arterial blood gas analysis and troponin I. A high anion gap metabolic acidosis was established upon admission associated with progressive increase in lactate secondary to septic shock. Troponin I levels were found above reference but with progressive decrease despite the episode of supraventricular tachycardia.

Discussion

The diagnostic approach of patients presenting gastric necrosis is complex since etiology is not well established and is usually multifactorial as in the case that we present because the patient had multiple risk factors such as advanced age, long-standing arterial hypertension, atherosclerotic disease evidenced in tomography images. Clinically, the patient presented data compatible with upper gastrointestinal bleeding and shock perpetuating the state of ischemia due to decreased blood flow to the gastric circulation, leading to the presence of frank necrosis. Although angiotomography ruled out a proximal occlusive event as well as the presence of gastric pneumatosis (important data for image documentation), absence of enhancement and thinning of the gastric wall were identified. By endoscopy, the presence of necrosis was verified, mainly affecting the lesser and greater curvatures, fundus and anterior and posterior wall of the gastric body, respecting the pylorus and duodenum, as described in literature. The patient´s acute presentation did not allow us to perform a surgical approach, since he was in poor general conditions with the support of vasopressors, opting for a complete medical management with palliative guidance at the request of his family. From the above, we can conclude that this type of disease continues to not be considered in the presence of abdominal pain, coupled with bleeding form the upper gastrointestinal tract, which is why it tends to be under-diagnosed. A high diagnostic suspicion must be had before complications such as shock occur, which perpetuates the reduction of general blood flow and specifically to the splanchnic circulation and opt for more aggressive strategies for its diagnosis and treatment. In diagnostic imaging, tomography and angiotomography are important; where, in addition to looking for gastric pneumatosis, changes in the wall and decreased enhancement must be analyzed. From all the above, it can be concluded that gastric necrosis continues to be a disease which we know very little about and its study and diffusion is essential to understand its pathophysiology and identify its possible causes, which represents a challenge to overcome to offer specialized treatments and reduce the morbidity and high mortality that it represents.

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