Case Report

Empysematous Gastritis - a case report with literature review

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Abstract

Emphysematous gastritis is a condition characterized by gas within the wall of the stomach and associated, systemic toxicity. We are reporting to our knowledge the first case of emphysematous gastritis in a 76 year old female from Islamabad, Pakistan. She was admitted with five day history of upper abdominal discomfort and vomiting. Diagnosis of emphysematous gastritis was made on CT scan. She was treated successfully with conservative management including IV antibiotics and few sessions of dialysis, and was discharged home within two weeks.

Introduction

Emphysematous gastritis is a condition characterized by gas within the wall of the stomach and associated systemic toxicity. It is a rare but lethal clinical entity. There are 41 cases of emphysematous gastritis reported so far in the English literature since 1889.[1] Diagnosis is based on clinical presentation of acute abdomen with associated features of systemic toxicity. Computed tomography (CT) is the diagnostic procedure of choice.[2-4] The mortality rate of patients with emphysematous gastritis remains high (60%-80%) despite early aggressive treatment.[5] Antimicrobial therapy with antibiotics covering gram-negative organisms and anaerobes, and surgery in appropriate cases may enhance survival.[6]

Case Report

The patient, 76 years old female, diabetic for 10 years, was transferred to our hospital from a local hospital where she was being treated for left lower lobe pneumonia for the last three days with ceftriaxone without much benefit. She presented to emergency room at Shifa International Hospital, Islamabad, with chief complaints of vomiting and upper abdominal discomfort for the last 5 days. She also had associated history of fever with chills, malaise and generalized weakness. Her vomitus contained food particles without any blood. Her abdominal pain was diffuse localized to upper abdomen, continuous, dull without any radiation, with no significant aggravating or relieving factors. There was no history of diarrhoea or malaena. She had a 40-pack year history of smoking. Rest of the clinical history was unremarkable and she had no known history suggestive of ischaemic heart disease or hypertension. She denied intake of any painkillers, alcohol, and corrosives. There was no

Table. Lab Investigations.

Complete blood count		
	Hemoglobin	17.2gm/L,
	HCT	50.30%,
	TLC	20,700/µl with 80% neutrophils having toxic granulations, 16% lymphocytes
	Platelets	50,000/µl, peripheral blood consistent with low platelet count
Biochemical Profile		
	Blood Glucose	128 mg/dl
	Serum Urea	289 mg/dl
	BUN	135 mg/dl
	Serum Creatinine	4.73 mg/dl
	Serum Sodium	142 mEq/dl
	Serum Potassium	5.1 mEq/dl
	Serum Chloride	110 mEq/dl
	Serum Biocarbonate	20 mEq/dl
	Serum Calcium	9.3 mg/dl
	Serum Posphorus	3.8 mg/dl
	ALT	67 IU/L
	AST	75 IU/L
	Alkaline Phosphatase	142 IU/L
	Total Bilirubin	1.58 mg/dl
	Direct Bilirubin	0.83 mg/dl
	PT / APTT	Normal
Urinlysis		Pus cells > 25, with 3+ leukocyte esterase
Creatinine Phosphokinase		150 IU/L

previous history of hospitalization or any psychiatric disorder.

On examination, she was slightly overweight, dehydrated, alert and in a state of distress. Her BP was 110/70 mmHg, pulse 120/min regular, temperature 98.6°F and R/R

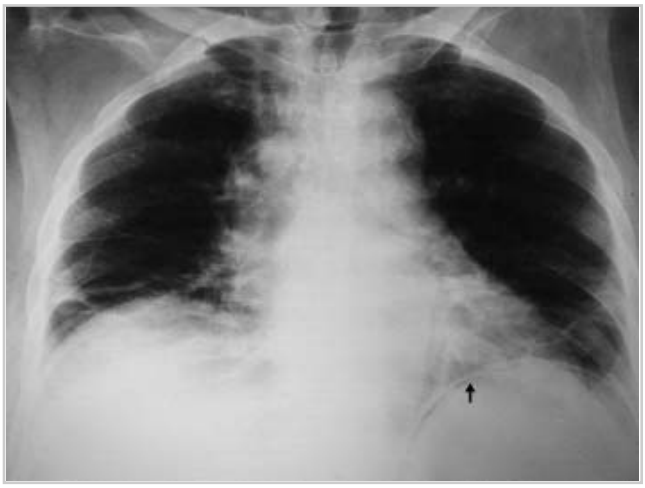

Figure 1. Gas under left hemidiaphragm (arrow).

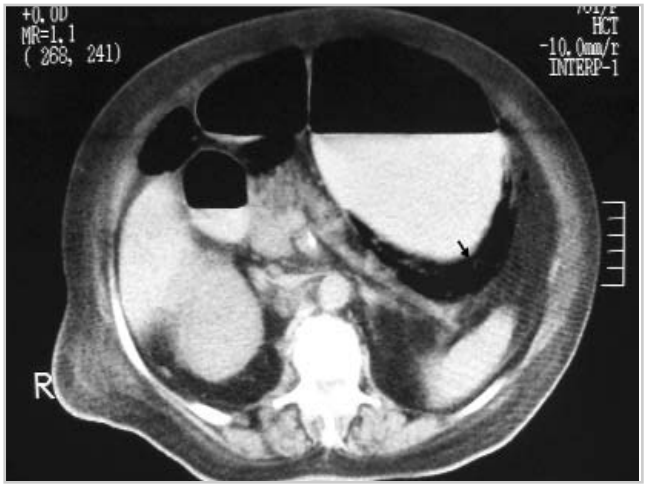

Figure 2. Gas is within the stomach wall (arrow).

of 18/min. Her abdominal examination revealed mild upper abdominal tenderness but no rebound tenderness, rigidity or guarding. Bowel sounds were normally audible. There was no palpable mass or viscera in rest of her abdominal examination. Murphy's sign was negative. Rest of the clinical examination was normal. Initial impression included acute abdomen or lower lobar pneumonia, acute coronary syndrome, and acute pancreatitis.

Her investigations are shown in Table. Chest x-ray showed suspicion of gas under Left hemi-diaphragm (Figure 1). Two sets of blood cultures prior to the start of antibiotics were negative. Clinical impression was sepsis, urinary tract infection, and acute abdomen with acute renal failure and rule out free air in the abdomen. Her CT Scan abdomen with oral contrast only showed intramural gas in the stomach wall predominantly in the fundus (Figure 2). Gas was also seen in the portal venous system in the liver. Rest of the CT was unremarkable.

Diagnosis of Empysematous Gastritis with sepsis, urinary tract infection and acute renal failure was made. Patient was admitted in monitored care settings and made nil per oral. Surgical, nephrology and infectious diseases services were consulted. Patient was started on Inj. Piperacillin/Tazobactum with IV Levofloxacin in renal adjusted doses. She was managed on the lines of pre-renal failure with IV fluids and later on three sessions of dialysis.

After 72 hours in the hospital, her general condition improved but total leukocyte count increased to 59,000/µl. Surgical services were again consulted and they ordered repeat CT Scan abdomen with the suspicion of visceral perforation or abdominal collection / abscess. Her repeat CT Scan showed resolution of the intra-mural gas in the stomach wall. There was no collection. Antibiotics were changed to IV Imipenum cilastatin in renal adjusted dose. Her renal function improved with three sessions of dialysis and creatinine decreased to 1.5 mg/dl.

On 10th day of admission, she was feeling much better with no abdominal pain or fever. Her leukocyte count declined to 14,000/µl. She was discharged with follow up in nephrology, surgical and internal medicine clinics.

Discussion

The presence of gas within the stomach wall can be either secondary to benign or pathologic entity. Besides infection with gas-forming bacteria, other possible sources include bland tissue infarction with necrosis; gas should be differentiated from atmospheric air introduced at recent instrumentation or secondary to pulmonary causes, like emphysema or positive pressure ventilation. Other clinical factors that contribute to the increased production or slowed removal of gas include a depressed cell-mediated immune response, local tissue necrosis, and the presence of arteriosclerosis.[7,8]

Of all the hollow viscera, the stomach is the least commonly affected by gas-forming infections.[5] Of the 41 reported cases in the English literature, caustic ingestion (37%) and alcohol abuse (22%) were found to be the most common causes. Other predisposing conditions include recent gastroduodenal surgery, trauma, gastric infarction, diabetes mellitus, end stage renal disease, multiple sclerosis, mucormycosis, aplastic anaemia, polycystic disease, empyema thoracis, acute gastric dilatation, immunocompromised state, NSAID/ ETOH overdosage, Coca Cola intake, infarction closure of coeliac vessel, pancreatitis, acute appendicitis, malnutrition, disseminated stronglyloidiasis, vasopression infusion, alkali intake, lye intake, acid intake, leukaemia, phytobezoar and adenocarcinoma stomach.[1,5,9,10] In our patient, the causative factor was diabetes mellitus, which is an uncommon cause, as reported in the literature.

Clinical manifestation may be dramatic, ranging from acute sepsis to gastric haemorrhage and, rarely, vomiting of the necrotic stomach cast.[2,3,5] Early endoscopic evaluation will reveal a "cobblestone" appearance of the gastric mucosa, a finding that represents submucosal blebs of air. Common offending bacteria include E coli, C welchii, and mixed infections with Staphylococcus aureus.[5]

Characteristic findings may be seen at conventional radiography within the first few days of illness and may persist up to 4 weeks.[10] Innumerable bubbles are seen to outline the stomach in a mottled distribution. The gastric folds are thickened due to underlying mucosal oedema, which may be diffuse or localized to the greater curvature of the stomach. CT scan is considered the modality of choice for detection of intramural gas and evaluation for the presence of pneumoperitoneum or portal venous gas. CT may also demonstrate irregular mucosal fold thickening and may be used to monitor response to treatment or disease.

An important differential diagnosis to consider is benign gastric emphysema or Gas collections within the gastric wall without associated infection by gas-forming organisms.[5] Gas may enter the wall from the lumen, peritoneal surface, or oesophageal or duodenal connection and is usually associated with violent coughing, vomiting, or severe obstructive pulmonary disease. Gastric fold inflammation and thickening are not present, and the patient is usually asymptomatic with spontaneous resolution expected. The characteristic radiographic features of benign gastric emphysema include thin, linear streaks of air running parallel to the border of the stomach that do not change with patient position.[5] CT will help confirm this linear distribution of mural air and, when performed with enteric contrast material, will demonstrate a normal gastric mucosal thickness of 3 mm or less.[2]

Treatment of emphysematous gastritis involves vigorous fluid support, correction of acid-base and electrolyte abnormalities, and coverage with intravenous broad-spectrum antimicrobial therapy. Unless perforation has occurred, surgery is best delayed until the fulminant sepsis can be better controlled. Even delayed surgeries have significant complications, including anastomotic breakdown, development of fistulae, and chronic stricture formation involving the distal oesophagus and nonresected portions of the stomach.[2,10] The mortality rate of patients with emphysematous gastritis remains high (60%-80%) despite early aggressive treatment.[5]

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