GERONTOLOGIA POLSKA 2023; 31; 130-133 OPIS PRZPADKU / CASE REPORT
zgłoszono 12.02.2023 – zaakceptowano 15.03.2023

Emphysematous gastritis – case report

Rozedmowe zapalenie błony śluzowej żołądka – opis przypadku

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Abstract

We report the case of a 71-year old man admitted urgently to the geriatric ward due to epigastric pain, nausea, and general weakness. Emphysematous gastritis was diagnosed based on elevated inflammatory markers and typical computed tomography scan with gastric distension, thickened mucosal folds, and intramural gas. The patient successfully underwent conservative treatment with intravenous broad-spectrum antibiotics, fluid and electrolyte imbalance correction, and nutritional support. The abdominal CT scan performed on the first day of hospitalization was decisive for making the correct diagnosis, and later treatment this rare disease. (Gerontol Pol 2023; 31; 130-133) doi: 10.53139/GP.20233109

Keywords: *emphysematous gastritis, stomach wall air, epigastric pain*

Streszczenie

Przedstawiamy przypadek 71-letniego mężczyzny przyjętego do oddziału geriatrii w trybie pilnym z powodu bóli nadbrzusza, nudności i ogólnego osłabienia. Na podstawie podwyższonych markerów stanu zapalnego oraz typowego obrazu tomografii komputerowej, w którym uwidoczniono rozdęcie żołądka, pogrubienie fałdów błony śluzowej i obecność powietrza w jego ścianie rozpoznano rozedmowe zapalenie błony śluzowej żołądka. Chorego z powodzeniem leczono zachowawczo stosując antybiotyki o szerokim spektrum działania, wyrównując zaburzenia wodno-elektrolitowe oraz prowadząc leczenie żywieniowe. Wykonane w pierwszej dobie hospitalizacji badanie tomografii komputerowej jamy brzusznej było decydujące dla postawienia rozpoznania i prowadzenia właściwego leczenia tej rzadkiej choroby. . (Gerontol Pol 2023; 31; 130-133) doi: 10.53139/GP.20233109

Słowa kluczowe: *rozedmowe zapalenie błony śluzowej żołądka, powietrze w ścianie żołądka, ból w nadbrzuszu*

Background

Emphysematous gastritis is a rare disease caused by gas-producing microorganism invading the stomach wall. It is a severe, life-threatening form of phlegmonous gastritis that may arise locally through the mucosa or spread via hematogenous. Abundant blood supply, effective mucosal barrier, and an acidic pH make that we see this type of gastritis a very uncommon. Predisposing factors are diabetes mellitus, renal failure, recent abdominal surgery, long-term corticosteroid and nonsteroidal anti-inflammatory drug use, alcohol abuse [1-4]. There are many microorganisms associated with this type gastritis: Streptococcus sp, Enterobacter sp, Clostridium sp, Escherichia coli, Pseudomonas aeruginosa, Staphylococcus aureus, Candida sp, Mucor sp [4,5]. The mortality in its course is estimated at 50-60%. Treatment consists of bowel rest, hydration, and intravenous administration of broad-spectrum antibiotics. Surgical intervention is not recommended during acute infection. It is reserved for patients who have failed optimal medical management, with perforations, peritonitis, strictures, and uncontrolled sepsis [5]. The most important thing is to make an early diagnosis and differentiate emphysematous gastritis from gastric emphysema because of the significant difference in management and prognosis.

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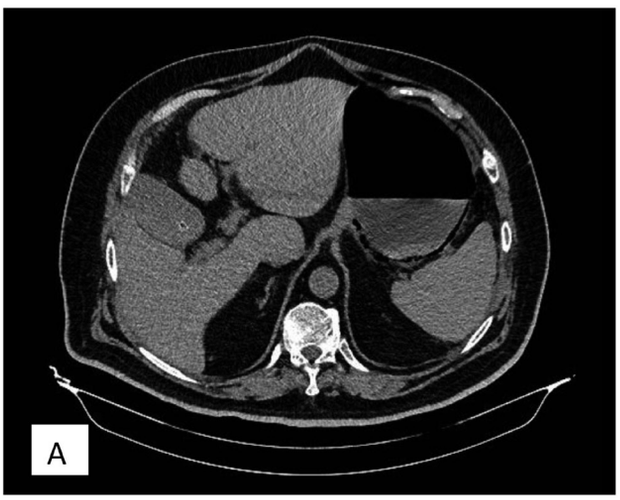
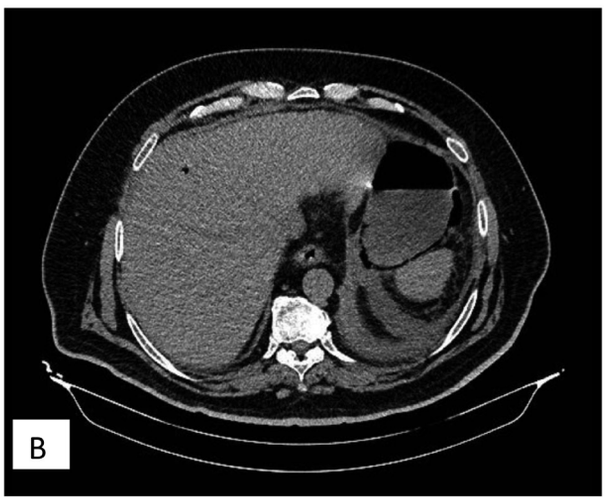
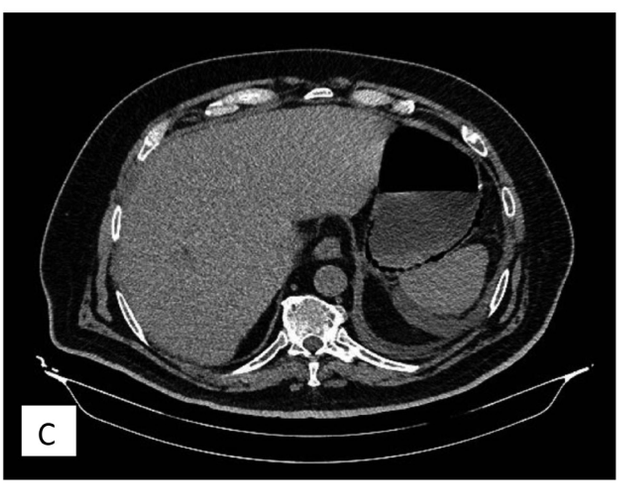
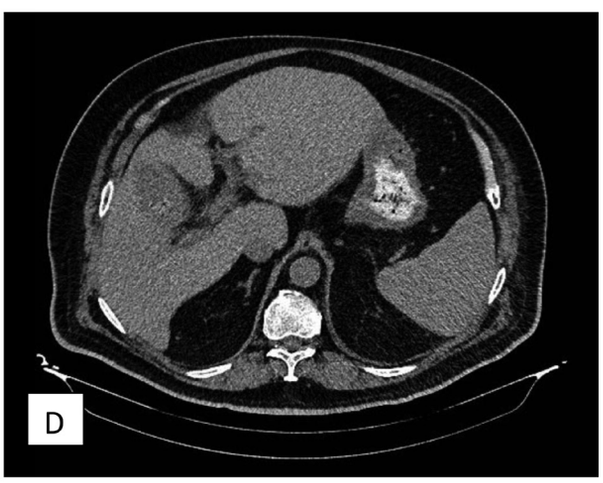
Case description

A 71-year-old man was admitted to the geriatric ward because of epigastric pain, accompanied by nausea, bloating and urge to defecate. Abdominal pain was of moderate intensity. The patient had no fever. He had undergone a total right hip arthroplasty two weeks before. After being discharged from the orthopedic ward, he spent most of his time in bed. He moved very little, only going to the toilet. Although he was already obe-









	1 day	3rd day	4th day	6th day	24th day	Reference Values
WBC count	8,50	14,38	16,2	9,91	5,09	4,23-9,07 G/l
RBC count	3,50	3,69	3,49	3,32	3,76	4,30-5,60 T/l
Haemoglobin	10,7	11,27	10,63	10,31	11,27	13,80-16,46 g/dl
PLT count	247	258	277	280	237	139-387 G/l
C-reactive protein	32,9	49,7	245,5	224,9	23,5	0,0-5,0 mg/l
Procalcitonin		0,98	2,50	0,95		< 0,5 ng/ml: systemic infection is unlikely 0,5-2,0: systemic infection is possible > 2,0: systemic infection is likely
Alanine aminotransferase		18	14	15		0-41 U/l
Aspartate aminotransferase		20	13	16		0-40 U/l
Total bilirubin			1,76	0,94	0,87	0,0-1,4,0 mg/dl
Creatinine	1,1	0,99	1,16	0,89	0,89	0,62-1,06 mg/dl
Urea E	42,6			59,4	31,2	16,56-48,42 mg/dl

Figure 1. A, B, C- Cross-sectional view of the CT scan without contrast of the abdomen showing distended stomach with thickening and formation of irregular and mottled gases in its wall (red arrows). Small amounts of air in the branches of the portal vein (green arrow). D- Cross-sectional view of the CT scan with contrast of the abdomen taken one week later, with marked thickening of the gastric wall with a small amount of gas (blue arrows), E- Table with the results of laboratory tests.

se, in the post-hospital period he gained a lot of weight (over 120 kg). For many years he had suffered from type 2 diabetes mellitus, hypertension and dyslipidemia. In laboratory tests, an increase in the parameters of inflammation was observed: CRP 32.9 mg/l (0.0-5.0), WBC 8.5 G/l (4.23-9.07) on the first day to CRP 49.7 mg/l, WBC 14.38 G/l on the third day of hospitalization. In other laboratory tests, the following were observed: glucose 7.6 mmol/l (3.9-5.5), HBA1c 6.21% (4.8-5.9), Ths 29.7 pg/ml (0-14), NT-proBNP 112 pg/ml (0.0-125.0), D-Dimer 5.7 ug/ml (0.0-0.5), amylase 33 U/l (28.0-100.0), bilirubin 30.2 umol/l (0.0-24), GGTP 118 U/L (10.0-71.0) (figure 1E). Chest X-ray showed no abnormalities apart from minor fibrosis in the left costophrenic angle. Abdominal X-ray without signs of obstruction or perforation of the gastrointestinal tract, Abdominal ultrasound showed numerous small gallstones, without thickening of the gallbladder wall. Gastroscopic examination revealed a 2 cm long infiltration covering 1/5 of the circumference of the esophagus above the gastric cardia and cardia, reaching the upper half part of the body of the stomach in the region of the greater curvature and the lower part of posterior wall, covered with hematin. Computed tomography (CT) of the abdomen showed a significantly dilated stomach with residual fluid and a 7 mm layer of air in its wall. Small amounts of air were also in the branches of the portal vein (figure 1 A,B,C).

The treatment included empirical antibiotic therapy metronidazole and amoxicillin with clavulanic acid, and additionally a proton pump inhibitor, infusion fluids and parenteral nutrition. CT taken one week later showed a thickening of the gastric wall to 27 mm (without gas) (figure 1D). In laboratory tests, we observed a decrease in the parameters of inflammation: CRP 245.5 mg/l, WBC 16.2 G/l, PCT 2.5 ng/ml on the fourth day, CRP 224.9 mg/l, WBC 9.9 G/l, PCT 0.95 ng/ml on the sixth day and CRP 23,5 mg/l on the twenty-fourth day of hospitalization (figure 1E). In the control gastroscopy performed a week after the first one, the esophagus was unchanged, the inflammatory infiltration in the stomach was much smaller, covered with fibrin with foci of normal mucosa. The ailments reported by the patient, especially pain in the epigastric region, also quickly decreased and, over the next few days completely disappeared. The patient was discharged from the hospital after 4 weeks of hospitalization. Histopathological examination of the gastric mucosa specimens taken during the first gastroscopy, showed abundant, neutrophilic inflammatory infiltration with features of moderate, superficial edema, preserved glandular architecture, without features of glandular epithelial dysplasia.

Discussion

More than 200 cases of emphysematous gastritis have been described in the literature. It is characterized by the presence of air in the gastric wall, secondary to a bacterial infection.

Predisposing factors emphysematous gastritis: diabetes mellitus, renal failure, malignancy, peptic ulcer disease, recent surgery, corticosteroid and NSAIDs, alcohol abuse, caustic ingestion, emesis, bowel obstruction, gastrostomy feeding [1-4]. The microorganisms associated with this type of gastritis: Streptococcus sp, Enterobacter sp, Clostridium sp, Escherichia coli, Pseudomonas aeruginosa, Staphylococcus aureus, Candida sp, Mucor sp. [4,5]. In over 40% of cases however, no organism is identified [6]. Emphysematous gastritis typically presents with abdominal pain, nausea and vomiting. Physical examination can range from mild abdominal tenderness to peritonitis in the setting of perforation [3,4,7]. The ischemic injury to the gastric wall seems to be the inciting event for emphysematous gastritis, leading to a secondary infection either from local bacterial invasion through the ulceration or from hematogenous spread [6].

The most sensitive diagnostic modality (of choice) is imaging with CT, showing stomach distention, thickening of the gastric wall (thickened folds and oedema gastric mucosal) with cystic pockets of air (irregular mottled gas in the gastric wall, especially in the fundus and greater curvature), associated air in portal venous system and pneumoperitoneum [1,3,4,8]. The extent of gastric emphysema as well as presence of portal venous gas and pneumoperitoneum do not correlate with the severity of disease [7]. The role of esophagogastroduodenoscopy in the diagnosis of emphysematous gastritis has not been clearly defined. It usually identifies an inflamed, erosive, or necrotic area of the mucosa [6].

Conservative management is the mainstay of therapy. Treatment consists of bowel rest, hydration, and early initiation intravenous administration of broad-spectrum antibiotics. Surgical intervention is not indicated during acute infection, even in the presence of portal venous air or pneumoperitoneum (increased mortality and postoperative complications such as anastomotic leak and fistula formation). It is reserved for patients who have failed optimal medical management, who deteriorate, with perforation, peritonitis, strictures, and uncontrolled sepsis [5,7,9]. The reduction in mortality (59.4% before 2000 versus 33.3% after 2000) has been also partially attributed to the lower rate of surgical intervention (62.5% before 2000 versus 22.2% after 2000) [6]. The factors associated with higher mortality are elevated serum lac-

tate, serum creatinine and concomitant pneumatosis in small bowel and colon [10].

It is important to distinguish emphysematous gastritis from gastric emphysema with is a benign, secondary to barotrauma disease caused by disrupted mucosa and air entry to the wall (linear distribution of gas, without portal venous air) due to endoscopy, nasogastric tube placement, severe vomiting. However, the patient remains asymptomatic and the condition resolves spontaneously [3,4,8,9,11].

Conclusions

Emphysematous gastritis is a severe, high mortality disease caused by gastric wall invasion by gas-forming microorganisms. Early diagnosis, initiation of treatment with intravenous broad-spectrum antibiotics and supportive measures is crucial for patients survival. Computed tomography examination which reveals gastric wall thickening, intramural gas, sometimes portal vein gas and pneumoperitoneum, remains the modality of choice for diagnosis emphysematous gastritis.

Conflict of interest
none

References

1. Takano Y, Yamamura E, Gomi K et al. Successful conservative treatment of emphysematous gastritis. Intern Med. 2015;54(2):195-8. doi: 10.2169/internalmedicine.54.3337.
2. Matsushima K, Won EJ, Tangel MR et al. Emphysematous gastritis and gastric emphysema: similar radiographic findings, distinct clinical entities. World J Surg. 2015;39(4):1008-17. doi: 10.1007/s00268-014-2882-7.
3. Ogbue OD, Haddad A, Daw H. A Rare Case of Emphysematous Gastritis Secondary to Chemotherapy. Cureus. 2021;13(10):e18895. doi: 10.7759/cureus.18895.
4. Alameri A, Al Hillan A, Museedi AS et al. Emphysematous Gastritis in the Setting of Chronic Abdominal Pain. Eur J Case Rep Intern Med. 2020;7(9):001749. doi: 10.12890/2020_001749.
5. Sharma P, Akl EG. A combination of intramural stomach and portal venous air: conservative treatment. J Community Hosp Intern Med Perspect. 2016;6(1):30519. doi: 10.3402/jchimp,v6.30519.
6. Watson A, Bul V, Staudacher J et al. The predictors of mortality and secular changes in management strategies in emphysematous gastritis. Clin Res Hepatol Gastroenterol. 2017;41(1):e1-e7. doi: 10.1016/j.clinre.2016.02.011.
7. Nasser H, Ivanics T, Leonard-Murali S et al. Emphysematous gastritis: A case series of three patients managed conservatively. Int J Surg Case Rep. 2019;64:80-84. doi: 10.1016/j.ijscr.2019.09.046.
8. AlHatmi A, Raniga SB, Mittal A. Emphysematous Gastritis on Computed Tomography. Sultan Qaboos Univ Med J. 2022;22(3):432-4. doi: 10.18295/squmj.10.2021.142
9. Matsushima K, Won EJ, Tangel MR et al. Emphysematous gastritis and gastric emphysema: similar radiographic findings, distinct clinical entities. World J Surg. 2015;39(4):1008-17. doi: 10.1007/s00268-014-2882-7.
10. Jehangir A, Rettew A, Shaikh B et al. A case report of emphysematous gastritis in a diabetic patient: favorable outcome with conservative measures. J Community Hosp Intern Med Perspect. 2015;5(4):28010. doi: 10.3402/jchimp.v5.28010.
11. Jeong MJ. Emphysematous gastritis: Impressive radiological findings. Radiol Case Rep. 2022;17(11):4257-9. doi: 10.1016/j.radcr.2022.08.046.