Polish Annals of Medicine

Journal homepage: https://www.paom.pl

Case report

Multiorgan air embolism as a complication of ruptured gastric ulcer

Marek Kowalczyk[1], *Łukasz Klepacki*[2], *Waldemar Kurpiewski*[3], *Łukasz Dyśko*[4], *Ewa Zieliński*[5], *Maciej Iwanowski*[6], *Lidia Glinka*[6], *Wiesław Pesta*[3]

[1] *Department of Psychology and Social Sciences in Medicine and Public Health, Faculty of Health Sciences, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland*
[2] *Department of Anatomy, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland*
[3] *Department of General and Minimally Invasive Surgery, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland*
[4] *Clinic of Oncological and General Surgery, University Clinical Hospital in Olsztyn, Poland*
[5] *Department of Emergency Medicine and Disaster, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland*
[6] *Department of Anaesthesiology and Intensive Care, School of Medicine, Collegium Medicum,University of Warmia and Mazury in Olsztyn, Poland*

Article info

Article history
Received 14 February 2021
Accepted 9 April 2021
Available online 24 August 2021

Keywords
Peptic ulcer
Left ventricle and ascending aorta gas
Gastric wall gas
Hepatic portal vein gas
Pylorostenosis

Doi
https://doi.org/10.29089/2020.20.00174

Abstract

Introduction: The gas in the vessels of the portal system and arterial vessels is a very disturbing symptom and life-threatening condition.

Aim: The aim of this study is to present a case of gas in the left ventricle (LV), ascending aorta (Ao), superior mesenteric vein (SMV) and hepatic portal vein (HPV) as a complication of gastric ulcer perforation (GUP).

Case study: A 78-year-old male patient, who had undergone a laparotomy due to gas in the HPV, SMV, Ao, and both chambers of the heart with concomitant GUP and critical pyloric stenosis. Laparotomy revealed perforation of the gastric wall with limited wall necrosis and critical pyloric stenosis. The patient died on postoperative day 1 due to multiple organ failure.

Results and discussion: The presence of gas in HPV (HPVG) and systemic circulation is a rare pathological condition associated with various abdominal diseases. GUP with a gas in the HPV, MV and Ao is very rare. While the mechanism of gas entry into the portal veins of the portal system is better understood, the paths of gas entry into the arterial vessels are still not fully understood and their descriptions are casuistic.

Conclusions: The treatment of patients with air in the HPV and systemic vessels is extremely difficult and has a very high risk of failure.

Corresponding author: Łukasz Klepacki, Department of Anatomy, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Warszawska 30, 10-082 Olsztyn, Poland. Tel.: +48 505 144 860. E-mail address: lklep31@gmail.com.

1. INTRODUCTION

The presence of gas in the vessels of the portal system – hepatic portal vein (HPV) gas (HPVG) and in arterial vessels is a very alarming symptom, often life threatening and associated most often with acute abdominal disease, requiring urgent surgical intervention.

Wolf and Evans described the presence of gas in the HPV for the first time in 1955 for infants with necrotizing enterocolitis,[1] while for adults the first description was given by Susman and Senturia in 1960 on the example of a patient with extensive ischemia and necrosis of the small intestine.[2]

Diagnosis is most often established during the diagnosis of acute abdominal disease or cardiovascular complications in the course of undiagnosed abdominal pathology. In such cases, we usually perform abdominal ultrasound, abdominal X-ray, computed tomography (CT) scan of the abdomen and chest. In the case of cardiovascular complications, additionally cardiac echocardiography (Doppler echocardiography) and CT scan of the head with assessment of brain blood supply.[3,4] The presence of gas in the HPV and right heart can be a consequence of many abdominal diseases. According to literature data, these are most often inflammatory and ischemic bowel diseases, intra-abdominal abscesses, peptic ulcer perforation, gastrointestinal obstruction.[5–7] Rare cases of gas in the liver vessels after accidental ingestion of concentrated hydrogen peroxide,[8] liver transplantation,[9] chemotherapy[10] have also been reported in patients with acute pancreatitis, pyelonephritis after extracorporeal shock wave lithotripsy (ESWL), acute appendicitis, inflammation biliary tract after percutaneous endoscopic gastrostomy (PEG) or percutaneous jejunostomy.[3]

2. AIM

The aim of this study is to present a case of a 78-year-old patient with a large amount of gas in the left ventricle (LV), ascending aorta (Ao), superior mesenteric vein (SMV) and HPV as a complication of gastric ulcer perforation (GUP).

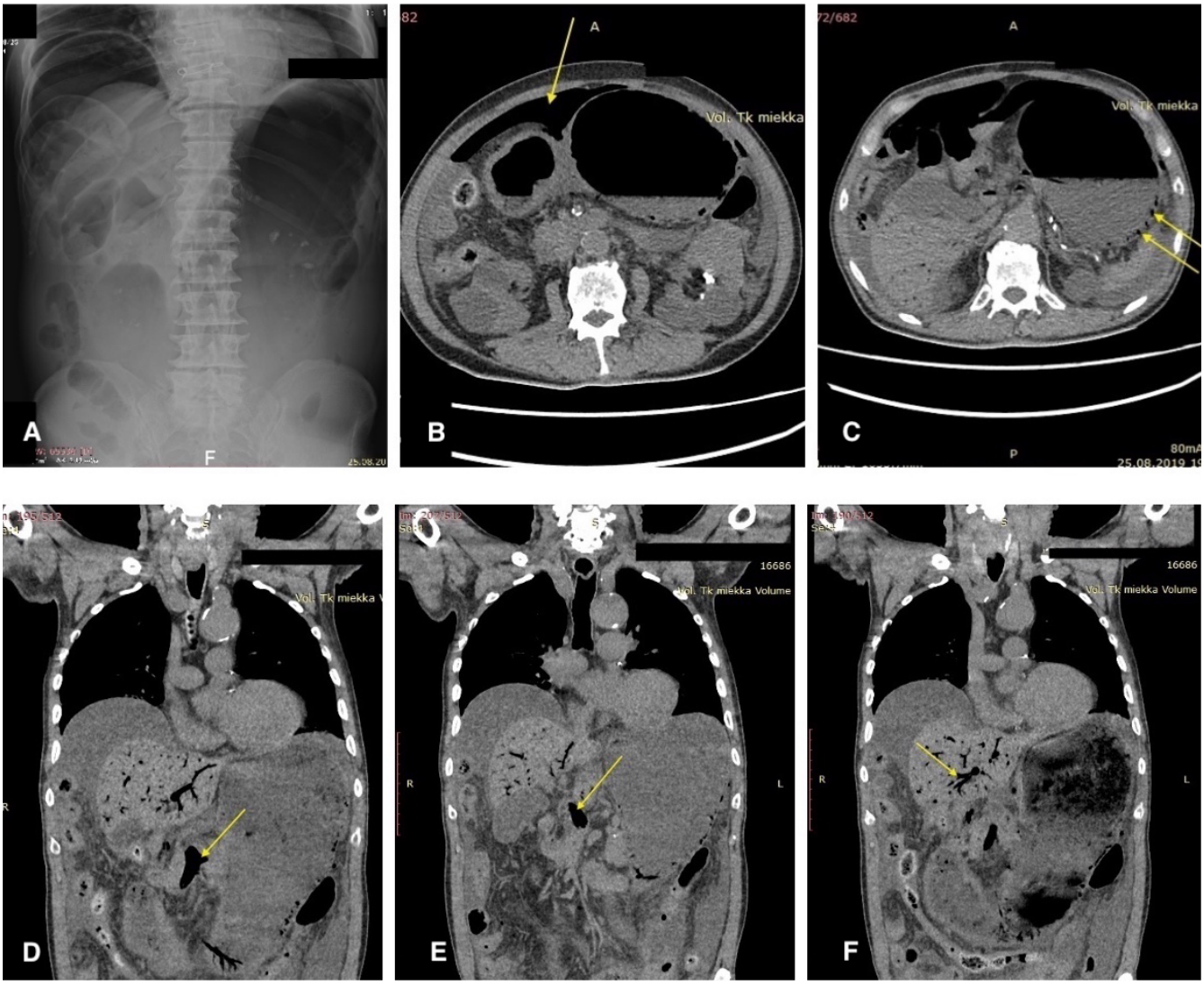

Figure 1. A: X-ray (AP) of the abdominal cavity on the standing. B: Abdomen axial CT. Free gas in the peritoneal cavity (arrow). C: Abdomen axial CT. Gas in the wall of the stomach (arrows). D: Abdomen coronal CT. Gas in the SMV (arrow). E: Abdomen coronal CT. Gas in the HPV (arrow). F: Abdomen coronal CT. Gas in the branches of the HPV (arrow).

3. CASE STUDY

The 78-year-old patient was brought to the Hospital Emergency Department by the Medical Rescue Team because of nausea, vomiting and dizziness preventing upright standing. The patient has been treated for many years for hypertension and chronic kidney disease (CKD) in the course of kidney stones and polycystic kidney disease. On November 4, 2017, he underwent percutaneous transluminal coronary angioplasty right coronary artery (PTCA RCA) due to myocardial infarction ST-segment elevation myocardial infarction (STEMI). Two drug eluting stents (DES) were implanted. On November 27, 2017, he underwent coronary artery bypass graft (CABG). Myocardial revascularization was performed on a beating heart without the use of extracorporeal circulation. The left internal thoracic artery was implanted into the anterior descending branch of the left coronary artery and the Y-graft using the left radial artery to the marginal branch of the left coronary artery. After basic examination and neurological and ear nose and throat specialist (ENT) consultations, peripheral and patient dizziness was initially diagnosed and transferred to the Otolaryngology Clinic for further diagnosis and treatment.

On the 3rd day of the clinic's stay in the late evening, the patient experienced nausea, vomiting and pain in the right lower abdomen radiating to the lumbar region and groin. The abdomen was soft, painful during deep palpation in the right lower abdomen and lower abdomen without palpable resistance. Peristalsis was normal. No rectal changes were found during examination. Goldflam's symptom on the right was very positive. Due to the clinical features of right-sided renal colic, which had repeatedly occurred, symptomatic treatment was followed, after which the symptoms resolved. From the morning of the next day the patient ate using the mouth and did not report gastrointestinal complaints. The main problem of the patient was dizziness which persisted from the day of admission to the Clinic.

On the 4th day of hospitalization in the evening, the patient suddenly fainted while trying to use the toilet. He did not lose consciousness, he did not suffer a head injury, however, he complained of severe abdominal pain. Urgent cardiac echocardiography was performed, which showed a large number of gas bubbles flowing into the RV and LV moving into the pulmonary trunk and Ao with indirect features of pulmonary hypertension, respectively. Immediate x-ray of the abdominal cavity was also performed (Figure 1A) and CT of the chest and abdomen without contrast due to high values of serum creatinine (4.15 mg%). Examinations showed the presence of a large amount of free gas in the peritoneal cavity (Figure 1B), a very large, distended with gas and fluid stomach with the features of walls pneumatosis (Figure 1C), the presence of a large amount of gas in the SMV (Figure 1D) and the HPV (Figure 1E) and its branches inside the liver (Figure 1F). A small amount of gas was also found in the Ao (Figure 2A) and the left ventricle (Figure 2B). The consulting surgeon found acute peritoneal symptoms in the course of gastrointestinal perforation and qualified the patient for immediate surgery. Due to the rapidly worsening of the patient's general condition of, it was recommended to transfer him to the operating theater directly from the Otolaryngology Clinic.

Laboratory examinations showed: albumin 2.1 g/dL (reference range 3.5–5.0 g/dL), phosphorus 11.9 mg/dL (2.7–4.5 mg/dL), pH 7.11 (7.35–7.45), creatinine 4.15 mg/dL (0.7–1.2 mg/dL), eGFR 15.2 mL/min (>60 mL/min), urea 121 mg/dL (10–50 mg/dL), potassium 6.9 mmol/L (3.5–5.1 mmol/L), procalcitonin 70.38 ng/mL (<0.5 ng/mL), C-reactive protein 312.2 mg/L (<5 mg/L), troponin T 0.889 ng/mL (0–0.014 ng/mL), glucose 90 mg/dL (60–98 mg/dL), sodium 134 mmol/L (135–145 mmol/L), leukocytes 9.0 × 10^9/L (4 × 10^9/L – 1.1 × 10^{10}/L), hemoglobin 13.2 g/dL (13.5–17.2 g/dL).

Immediately after entering the procedure the circulation suddenly stopped. Resuscitation was undertaken for approximately 30 minutes with 3-fold defibrillation with good results. After stabilization of the circulation, a laparotomy was performed, during which a large amount of free gas was released under pressure. After aspiration of about 1.5 L of cloudy grey-yellow content, a puncture was found in the pylorus on the anterior wall with a diameter of about 1 cm with limited necrosis of the gastric wall of the ulcer area. The

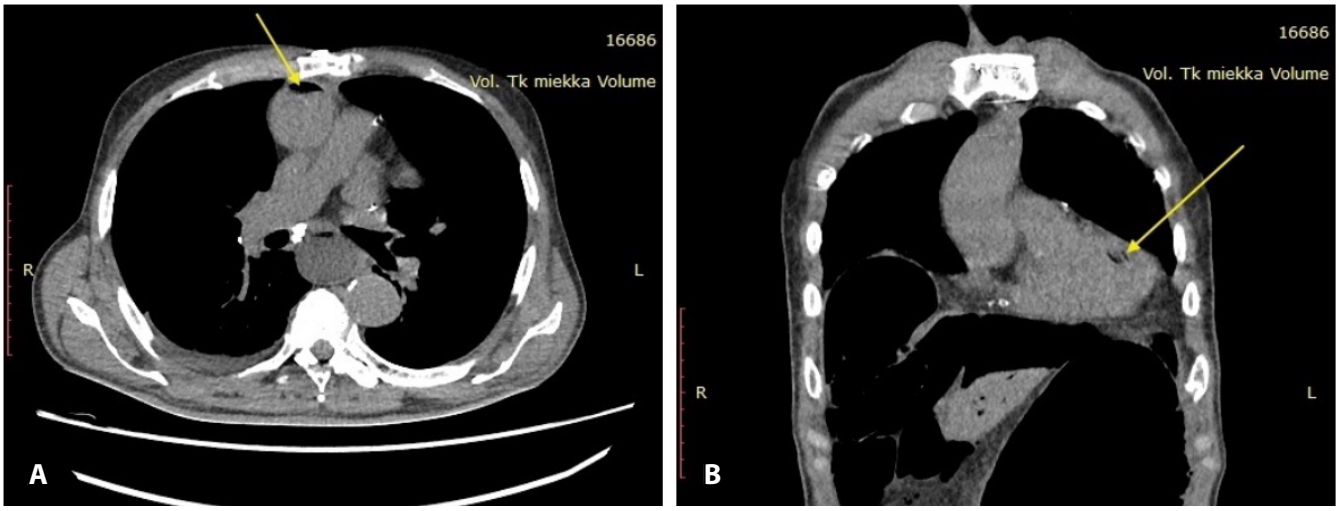

Figure 2. A: Thorax axial CT. Gas in the Aorta (arrow). B: Thorax coronal CT. Gas in the left ventricle (arrow).

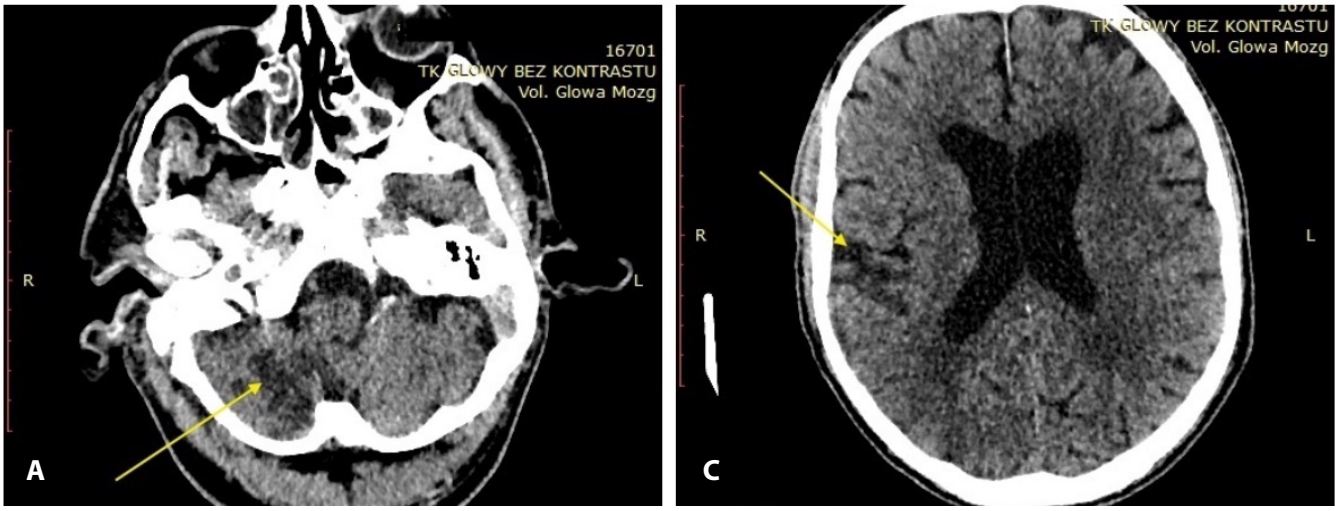

Figure 3. A: Head axial CT. Infarction of the right hemisphere of the cerebellum (arrow). B: Head axial CT. Infarction of the right parietal lobe (arrow).

passage from the stomach to the duodenum was very narrow, dysplasia, reaching with the lower part to the smaller pelvis. The perforation site along with the wall of dubious vitality was cut out, followed by a plastic surgery using the Heineke–Mikulicz method. Peritoneal cavity material was collected for bacteriological examination. The resected gastric lesion was sent for histopathological examination. Meropenem (*Meropenemum*) was given. The patient was not woken up after the procedure. He was mechanically ventilated with continuous infusion of norepinephrine and was transferred to the Oncological and General Surgery Clinic. In the presence of persistent circulatory and respiratory failure and growing.

In the absence of persistent cardiopulmonary insufficiency and increased renal failure, the patient was transferred to the Intensive Care Unit.

Due to the anisocoria that was found during the surgery, a CT scan of the head was performed without intravenous administration of a contrast agent. Hypodense areas were found in the right hemisphere of cerebellum (Figure 3A) and in the right parietal lobe (Figure 3B), most likely ischemic in nature, but no gas was found in the cerebral vessels. Despite intensive therapy, a gradual increase in the symptoms of multiorgan failure was observed and after a day or so after surgery, sudden cardiac arrest occurred in the mechanism of asystole. Resuscitation was undertaken however, cardiopulmonary function could not be restored and the patient died.

Escherichia coli was grown from the fluid collected during surgery from the peritoneal cavity. Histopathological examination of the resected gastric fragment showed perforated gastric ulcer with necrosis and hemorrhagic inflammation in the immediate vicinity of the ulcer. In this case, no autopsy was performed because the Hospital Administration approved the close family's request to withdraw from the autopsy.

4. RESULTS AND DISCUSSION

The presence of HPVG and systemic circulation is a rare pathological condition associated with various abdominal diseases. Due to advances in radiological imaging diagnostics, the number of clinical cases described increased, however, it should be noted that these are still single cases. GUP with a large amount of gas in the portal vein, mesenteric vein and aorta is very rare. According to the data provided in the literature, among all causes of HPVG it accounts for only 3%-4%.[3,11]

While the mechanism of gas entry into the portal veins of the portal system is better understood, the paths of gas entry into the arterial vessels are still not fully understood and their descriptions are casuistic. An example of this can be presented by Igarashi et al. a case of perforation of an ulcer in a trapped esophageal hernia with the formation of a fistula between the stomach and the LV. CT scan revealed gas bubbles in the LV, coronary vessels and hepatic artery. In this case, an autopsy was performed, which showed the presence of gas in the vessels of the heart, liver, spleen, kidneys and brain. The cause of this systemic gas embolism was material from the stomach.[12]

There are several possible ways for gas to enter the portal system vessels. In the case of significant pyloric stenosis, the stomach wall is stretched and thinned. This gives a greater possibility of diffusion of excessively accumulated gas in the stomach to the venous vessels of the stomach wall and then to the portal circulation. Stretching of the stomach wall and inflammatory infiltration at the point of interruption of its continuity causes after an hour an increase in the production of free radicals in the stomach wall. As a result of the activation of multinucleated granulocytes and local secretion of cytokines, local hypoxia and dilatation of capillaries occur, which can promote the intake of gas from the environment into the venous system.[13,14] If the stomach wall is healthy, its stretching may lead to the situation when the venous vessels may collapse, while during ulcers the stomach wall is pathologically altered, hard and not elastic, venous vessels may not collapse and be the gate for the accumulated in the stomach

gas, which can freely move through venous vessels to the liver. An important role in this pathology may also be played by the mechanism of blood suction by the damaged venous perforation of the larger venous vessel. This mechanism plays an important role in the movement of venous blood towards the heart in the drainage of the inferior vena cava.

The displacement of gas from the portal vein system to the inferior vena cava system, and then to the right heart in our case in our opinion resulted primarily from the large amount of gas migrating to the vessels. An important role in this process should also be attributed to the fact that physiologically the blood in the portal vein system is at a higher pressure than in the inferior vena cava system at the same level. Also, the transfer of excessive gas to the systemic circulation via the left gastric vein and then the odd vein cannot be excluded, as described by Lun and Shum.[15]

In our case, it is definitely more difficult to explain the mechanism of gas displacement to the left heart, coronary vessels, aorta and its branches. In the presence of gas in the right heart, simple embolism most often occurs i.e., gas from the right cavities of the heart enters the pulmonary vessels. Pulmonary obstruction further releases inflammatory mediators and vasoconstrictor factors, which ultimately leads to increased vascular permeability and interstitial lung edema. Although there is no confirmation of pulmonary embolism in CT of the chest of our patient, it should be remembered that this test was performed without the use of contrast due to high values of creatinine. An indirect symptom confirming pulmonary embolism of the patient are the features of pulmonary hypertension found by echographic examination.

In the case we describe, there cannot be excluded the occurrence of so-called paradoxical embolism, which may occur in the case of an early undiagnosed heart defect in the form of an atrial or ventricular opening. Although echographic examination did not show such a defect, it should be noted that it was performed bedside. The cardiologist interpreting the results pointed to great difficulties in performing the examination due to the lack of cooperation with the patient and difficulties in obtaining a satisfactory projection of the sub-bridge and RV. This could have resulted in difficulties in visualizing a possible leak between the heart cavities.

We know that the patient underwent coronary artery bypass surgery due to myocardial ischemia, he had double coronary stenting. Ischemia and myocardial infarction are described in the literature for people with pathological left coronary artery deviation from the pulmonary artery. This defect most often happens with infants, but thanks to compensatory mechanisms and the formation of collateral circulation, it can also manifest itself in adulthood (about 10% of cases). This situation can promote gas leakage from the right to the left heart. There have also been cases of the presence of different sizes and amounts of coronary artery connection with systemic or pulmonary vessels, with heart cavities and main veins, which also allows gas to enter the venous system into the arterial system without having to leak through a hole in the septum or the pulmonary circulation.[16] Most often these are birth defects, but in rare cases they can be acquired, e.g. as a result of percutaneous coronary intervention. The patient has had coronary angiography in the past and coronary fistulas after coronary angiography are reported in the literature with a frequency of 0.3%–0.8%.[17]

Another reported cause of gas entering the portal circulation is the formation of large amounts of bacterial gas in the gastrointestinal wall and the possibility of bacteria producing large amounts of gas into the venous vessels. This is especially true for bacteria that produce large amounts of gases: *Clostridium difficile*, *Enterobacter* spp., *Klebsiella pneumoniae*, *Staphylococcus aureus*, *S. pneumoniae*, *E. coli*, *Pseudomonas aeruginosa*, *Proteus* spp. and some fungi.[18] Most of these bacteria develop when the stomach is dilated and gastrointestinal stasis in patients with pyloric stenosis. Significant pneumatosis of the stomach wall found in CT of the abdominal cavity presented to the patient also creates excellent conditions for the penetration of the above bacteria into the stomach wall and into the blood vessels. In the case we described, *E. coli* was grown from the peritoneal cavity and *Candida glabrata* and *P. aeruginosa* from the throat.

There have been reports in the literature of gastrectomy in cases of gastric pneumatosis as one of the treatment options. Taking into account the patient's age, clinical condition and comorbidities, we performed the procedure with the smallest possible extent – excision of the perforation site and suturing of the stomach with Heineke–Mikulicz pyloroplasty. Due to the high mortality associated with such clinical conditions, we try to limit surgery to a minimum.[19] Immediately before surgery, the anesthesiologist calculated the risk of perioperative death and the risk of complications. According to the Physiologic and Operative Severity Score for the enUmeration of Mortality and Morbidity (POSSUM) scale, the patient received 81.45% for the risk of death and 99.28% for the risk of postoperative complications. An important factor worsening the prognosis in the case of pneumatosis of the gastric wall and the presence of gas in the portal vein, mesenteric vein and arterial vessels are comorbidities such as generalized atherosclerosis and intestinal ischemia, diabetes, renal failure, hypertension, immunity-reducing diseases, reducing visceral flow and altering bacterial flora.[20]

In this case, the patient was treated for hypertension, chronic coronary artery disease, chronic renal failure and atherosclerosis. Poor prognosis in our patient also resulted from sudden cardiac arrest immediately before surgery, long-term resuscitation (28 minutes), multi-site central nervous system (CNS) ischemia, and increasing renal failure that did not respond to renal replacement therapy Continuous Veno-Venous Hemofiltration calcium/ionised serum calcium (CVVHD CiCa).

Suspicion of ischemia and necrosis of the gastrointestinal wall requires urgent laparoscopy or laparotomy, as delay in surgery is associated with high mortality (75%–100%).[21–23]

5. CONCLUSIONS

The treatment of patients with air in the hepatic portal vein and systemic vessels is extremely difficult and has a very high risk of failure. Laparoscopy or laparotomy should be

performed immediately, since withdrawal or delay in surgery is associated with high mortality in this group of patients.

Conflict of interest
None declared.

Funding
None declared.

References
1 Wolfe JN, Evans WA. Gas in the portal veins of the liver in infants; a roentgenographic demonstration with postmortem anatomical correlation. *Am J Roentgenol Radium Ther Nucl Med*. 1955;74(3):486–488.
2 Susman N, Senturia HR. Gas embolization of the portal venous system. *Am J Roentgenol Radium Ther Nucl Med*. 1960;83:847–850.
3 Abboud B, El Hachem J, Yazbeck T, Doumit C. Hepatic portal venous gas: Physiopathology, etiology, prognosis and treatment. *World J Gastroenterol*. 2009;15(29):3585–3590. https://dx.doi.org/10.3748%2Fwjg.15.3585.
4 Suzuki T, Ando T, Usami A, et al. Cerebral air embolism as a complication of peptic ulcer in the gastric tube: case report. *BMC Gastroenterol*. 2011;11(1):139. https://doi.org/10.1186/1471-230X-11-139.
5 Lai WH, Hwang TL, Chen HW. Portomesenteric venous gas in acute bowel ischemia: Report of a case. *Surg Today*. 2008;38(7):656–660. https://doi.org/10.1007/s00595-007-3678-2.
6 Şen M, Akpinar A, Inan A, Şişman M, Dener C, Akin K. Extensive hepatic-portal and mesenteric venous gas due to sigmoid diverticulitis. *World J Gastroenterol*. 2009;15(7):879–881. https://dx.doi.org/10.3748%2Fwjg.15.879.
7 Domínguez Jiménez JL, Puente Gutiérrez JJ, Marín Moreno MA, Bernal Blanco E, Gallardo Camacho JI, Uceda Vañó A. [Gastric pneumatosis and gas in the portal venous system secondary to peptic ulcer] [in Spanish]. *Gastroenterol Hepatol*. 2008;31(8):494–496. https://doi.org/10.1157/13127091.
8 Dasa O, Moroi MK, Ruzieh M. Gas in the Liver. *Gastroenterology*. 2019;157(2):313–314. https://doi.org/10.1053/j.gastro.2019.04.007.
9 Chezmar JL, Nelson RC, Bernardino ME. Portal venous gas after hepatic transplantation: Sonographic detection and clinical significance. *Am J Roentgenol*. 1989;153(6):1203–1205. https://doi.org/10.2214/ajr.153.6.1203.
10 Kung D, Ruan DT, Chan RK, Ericsson ML, Saund MS. Pneumatosis intestinalis and portal venous gas without bowel ischemia in a patient treated with irinotecan and cisplatin. *Dig Dis Sci*. 2008;53(1):217–219. https://doi.org/10.1007/s10620-007-9846-9.
11 Kinoshita H, Shinozaki M, Tanimura H, et al. Clinical features and management of hepatic portal venous gas: Four case reports and cumulative review of the literature. *Arch Surg*. 2001;136(12):1410–1414. https://doi.org/10.1001/archsurg.136.12.1410.
12 Igarashi I, Suzuki K, Hosono F, et al. Perforation of a peptic ulcer in a hiatal hernia into the left ventricle with systemic air and food embolism. *J Comput Assist Tomogr*. 2018;42(5):767–770. https://doi.org/10.1097/rct.0000000000000747.
13 Bhargava P, Parisi M. Gastric pneumatosis and portal venous gas: Benign findings in hypertrophic pyloric stenosis. *Pediatr Radiol*. 2009;39(4):413. https://doi.org/10.1007/s00247-008-1088-2.
14 Zeitouni NE, Chotikatum S, von Köckritz-Blickwede M, Naim HY. The impact of hypoxia on intestinal epithelial cell functions: consequences for invasion by bacterial pathogens. *Mol Cell Pediatr*. 2016;3(1):14. https://dx.doi.org/10.1186%2Fs40348-016-0041-y.
15 Lun C-T, Shum H-P. The aquarium in the right heart. *Echocardiography*. 2018;35(9):1482–1483. https://doi.org/10.1111/echo.14110.
16 FCE Editor Office. [Anomalies of coronary arteries] [in Polish]. 2001;8(Suppl B):B22–B23.
17 Gowda RM, Vasavada BC, Khan IA. Coronary artery fistulas: Clinical and therapeutic considerations. *Int J Cardiol*. 2006;107(1):7–10. https://doi.org/10.1016/j.ijcard.2005.01.067.
18 Singh K. Emphysematous gastritis associated with Sarcina ventriculi. *Case Rep Gastroenterol*. 2019;13(1):207–213. https://doi.org/10.1159/000499446.
19 Yalamanchili M, Cady W. Emphysematous gastritis in a hemodialysis patient. *South Med J*. 2003;96(1):84–88. https://doi.org/10.1097/01.smj.0000048085.35271.75.
20 Chan SC, Wan YL, Cheung YC, Ng SH, Wong AMC, Ng KK. Computed tomography findings in fatal cases of enormous hepatic portal venous gas. *World J Gastroenterol.* 2005;11(19):2953–2955. https://dx.doi.org/10.3748%2Fwjg.v11.i19.2953.
21 Hou SK, Chern CH, How CK, Chen JD, Wang LM, Lee CH. Hepatic portal venous gas: Clinical significance of computed tomography findings. *Am J Emerg Med*. 2004;22(3):214–218. https://doi.org/10.1016/j.ajem.2004.02.017.
22 Peloponissios N, Halkic N, Pugnale M, et al. Hepatic portal gas in adults: Review of the literature and presentation of a consecutive series of 11 cases. *Arch Surg*. 2003;138(12):1367–1370. https://doi.org/10.1001/archsurg.138.12.1367.
23 Lasocki J, Kowalczyk M, Dyśko Ł, et al. A chicken bone stuck in a divertic sigmoid colon, accidentally detected during a colonoscopy. *Pol Ann Med*. 2020;27(2):205–208. https://doi.org/10.29089/2020.20.00134.