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# Emphysematous gastritis in a patient with coxsackie B3 myocarditis and cardiogenic shock requiring veno-arterial extra-corporeal membrane oxygenation



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## ABSTRACT

**INTRODUCTION:** Emphysematous gastritis is a rare condition in which gas accumulates in the stomach lining usually due to an infectious source.

**CASE PRESENTATION:** We present a 16 year old female with viral myocarditis and cardiogenic shock transferred to our hospital on extracorporeal membrane oxygenation (ECMO) who developed emphysematous gastritis. After listing the patient for heart transplant, patient underwent Bi-VAD placement requiring veno-venous ECMO support. Subsequently, she developed worsening abdominal distention. CT of abdomen/pelvis showed the stomach to be diffusely edematous, thick-walled, containing intramural gas collections, consistent with emphysematous gastritis. Patient underwent nonoperative management and two weeks later had complete resolution of the gastritis. Unfortunately, her overall condition deteriorated in the subsequent days and support was withdrawn.

**DISCUSSION:** Management of emphysematous gastritis usually revolves around supportive care, broad spectrum antibiotics and bowel rest. Our patients' gastritis resolved with non-operative management, albeit, she succumbed to multiorgan failure due to other causes.

**CONCLUSION:** We believe, this is a unique case of a veno-arterial ECMO causing emphysematous gastritis.

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## 1. Background

Emphysematous gastritis is an extremely rare condition in which gas accumulates in the stomach lining secondary to infection with gas-producing organisms. It is often associated with high mortality, and surgical intervention is usually avoided unless uncontrollable sepsis persists or patient develops gastric perforation. We present the case of a 16 year old female with viral myocarditis and cardiogenic shock that was transferred to our hospital on extracorporeal membrane oxygenation and developed emphysematous gastritis that resolved with nonoperative management.

## 2. Case presentation

The patient is a 16 year old female, otherwise healthy, who presented to an outside hospital with a one week history of fever, chest and back pain. On initial workup, she was found to have ST elevation in all leads with troponin of 98 ng/L. Soon after, she became hemo-

dynamically unstable, in cardiogenic shock and respiratory failure requiring intubation and vasopressors. This was complicated by ventricular tachycardia. Lidocaine was administered and patient's rhythm was brought back. However, she continued to deteriorate and it was decided to place her on venoarterial extracorporeal membrane oxygenation (VA-ECMO) via right internal jugular vein into her right femoral vein and femoral artery. A myocardial biopsy was performed that revealed lymphocytic infiltrate. The patient received high dose solumedrol with no improvement in her symptomatology. Echocardiogram revealed severe biventricular dysfunction with biventricular akinesis. Her blood and urine cultures were negative. Our hospital was consulted for possible bridge to recovery and transplant evaluation. On arrival to our hospital, patient stayed intubated with VA-ECMO circuit in place. She was awake and intermittently following commands. Her mean blood pressure was 77 mmHg with no systolic or diastolic component with warm and pulseless extremities. Rest of the examination was unremarkable. She was on milrinone, nipride, furosemide, and heparin infusions. Her initial labs revealed hemoglobin and hematocrit of 11 g/dL and 33%, white count of  $10.4 \times 10(9)/L$ , platelets  $89 \times 10(9)/L$ , creatinine 1.2 mg/dL and bilirubin 1.1 mg/dL. A repeat electrocardiogram revealed asystole and echocardiogram performed at our hospital revealed dense "jello like" soft tissue echoes in the left ventricle extending upto the aortic valve consis-

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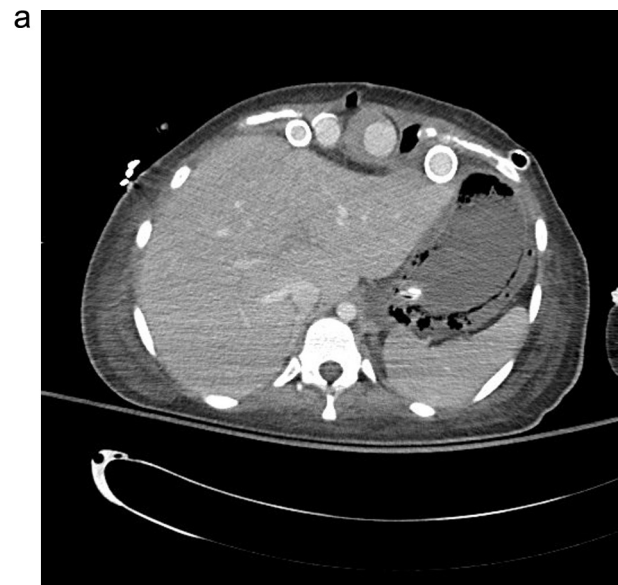


**Fig. 1.** Gaseous distention of the stomach as well as dilated small bowel measuring 5 cm in caliber.

tent with thrombotic stasis. Similar findings were seen in the right ventricle. Initial computed tomography (CT) of chest, abdomen and pelvis had extensive consolidation in the lower lobes with small bilateral pleural effusions and pericardial effusions, with moderate ascites in abdomen and pelvis, edematous gallbladder, and no evidence of bowel obstruction. The stomach was pathologically normal on review of initial work-up.

After patient was evaluated and listed for heart transplantation, she was taken to the operating room for placement of Thoratec biventricular assist device (Bi-VAD). Once the Bi-VAD was allowed to take over the circulation, patient became hypoxic with weaning off cardiopulmonary bypass, and the decision was made to place the patient on veno-venous ECMO (VV-ECMO). The femoral artery cannula was removed and repaired. The right femoral vein cannula was used as the inflow, and the right internal jugular cannula was used as the outflow for the ECMO circuit. Left ventricular biopsy revealed severe lymphocytic infiltrate with marked myocyte necrosis. There were no giant cells, eosinophils, or evidence of vasculitis. Additional studies on the myocardium involving polymerase chain reaction and in-situ hybridization showed active replication of coxsackie B3 virus.

Few days later, patients urine output continued to decline with increase in creatinine upto 2.6 mg/dL. She was diagnosed with acute kidney injury secondary to her critically ill state by nephrology and they recommended continuous renal replacement therapy. She developed hemorrhagic pulmonary edema and after bronchoscopy she was found to be positive for *Escherichia coli*. The next day patient was found to have massive abdominal distention with liquid non-bloody stools. Abdominal X-ray revealed gaseous distention of the stomach (Fig. 1) as well as small bowel measuring up to 5 cm in caliber, finding consistent with small bowel obstruction/ileus. A CT of abdomen and pelvis showed stomach to be diffusely edematous, thick-walled, containing intramural gas collections, consistent with emphysematous gastritis (Fig. 2). Small bowel was found to have thickened valvulae conniventes but appeared to perfusing normally. A subsequent echocardiogram demonstrated good pulsatile LVAD inflow with a velocity of 2.23 m/s and outflow with a velocity



**Fig. 2.** Stomach is diffusely edematous and thick-walled and contains both linear and rounded intramural gas collections consistent with emphysematous gastritis.

of 2.4 m/s. RVAD inflow had a velocity of 1.6 m/s with outflow of 1.2 m/s. Severe decrease in left and right ventricular function were also seen, as expected. There was no mass/thrombus visualized in the left atrium. Following this, patient was kept NPO, on nasogastric tube decompression, on broad spectrum antibiotics, including vancomycin, meropenem and fluconazole; and aggressive resuscitation involving multiple vasopressors was continued.

Two weeks later, patient was found to have complete resolution of emphysematous gastritis with return of stomach wall thickness to normal [Fig. 3]. Normal caliber loops of small and large bowel were found with easy transit of oral contrast into the colon. However, patient continued to be in progressive respiratory failure requiring ECMO support for which she underwent right mini thoracotomy and open lung biopsy. Pathology revealed hemorrhagic diffuse acute lung injury with features most consistent with hemorrhagic diffuse alveolar damage. Additional stains were performed on the specimen to rule out viral cause, all of which were negative. Patient continued to be in multisystem organ failure with persistent elevation in liver function tests involving ALT of 329 IU, AST of



Fig. 3. Resolution of the gas collections and gaseous distention of the stomach.

692 IU, Alkphos of 362 IU and total bilirubin of 30.2 mg/dL. Patient underwent a repeat viral serology screen at which point she had a positive CMV serology of greater than 60 and PCR which confirmed the presence of disseminated CMV for which ganciclovir was started.

Hepatology was consulted who recommended a transjugular liver biopsy. Pathology revealed severe hepatocanicular and cholangiolar cholestasis with centrilobular feathery degeneration and focal central lobular hepatocyte dropout, consistent with nonobstructive cholestatic process. However, CMV immunohistochemical stain was negative.

In the next few days, patient continued to deteriorate hemodynamically with increasing norepinephrine requirements as high as 24 mcg/min. Due to the patient's now hypercapnic and hypoxic respiratory failure, oliguric renal failure, shock liver, biventricular systolic failure on biventricular assist devices, with persistent *E. coli* pneumonia, CMV viremia, coxsackie B myocarditis, superficial dehiscence of most of the surgical wounds, a family meeting was done. The patient was not a candidate for heart/lung transplant or ECMO at this point. No more escalation of therapy was decided and the family agreed for withdrawal of support.

### 3. Discussion

Emphysematous gastritis is a rare, potentially fatal condition, first described in 1889 [1]. It usually presents with abdominal pain, nausea, emesis and florid septic shock, with a mortality rate of 55–61% [2–3]. Diagnosis is usually made based on a CT scan which typically shows gastric wall thickening and intramural gas [4–5]. The gas pattern is more prominent around the fundus and the greater curvature as described before [4].

Several etiologies have been reported such as vessel occlusion, gastric infarction or gastric volvulus. However, rarely, it has also been associated with nasogastric tube placement, persistent emesis, severe gastroenteritis, especially following chemotherapy or related to gastric ulcer disease. Gastric pneumatosis may also occur

after caustic ingestion, excessive ingestion of carbonated beverages, iatrogenic injury or sometime even idiopathic [6].

There are a number of predisposing factors that have been identified: immunosuppression including diabetes, malignancy, alcohol, medication abuse (especially non-steroidal anti-inflammatory drugs), post abdominal/pelvic surgery and corrosive ingestion [7–10]. Usually causative organisms can include Gram positive cocci, Gram negative rod (*E. coli*) and sometimes even fungi (*Candida albicans*) [4,5,11]. However, there has been no association reported in the literature with viral dissemination, either Coxsackie virus or CMV. Our patient presented to us as a result of viral myocarditis and subsequently deteriorated. It is difficult to ascertain viral organisms playing a direct role as they are not gas forming. However, in the setting of extreme ill state with relative immunosuppression, it may be reasonable to assume, that this lead to concomitant bacterial invasion of the gastric lining.

Furthermore, our patient did not have a nasogastric tube inserted before the event and nor did she have an infectious diarrhea. She began to have liquid non-bloody stools when she developed abdominal distention but stool studies did not reveal any organism. It may be possible that she harboured an ulcer but resolution of the findings with just nonoperative management does not favour that. In one of the series [6], only 38% of the patients had a portal venous air present. On further analysis, there was no significant difference observed in patients that survived and those that did not. Even though, it is considered a hallmark sign of pneumatosis, whether small bowel, colonic or even stomach related, the absence of portal gas does not rule it out and caution should always be exercised.

Another possibility is the relative low-flow state that was present in our patient. Even though, the patient was supported using VA-ECMO and was transitioned to Bi-VAD, the ongoing sepsis proved it extremely difficult to maintain adequate flows to achieve good perfusion. This could have potentially lead to bacterial translocation of the gastrointestinal tract that resulted in emphysematous gastritis. Why this would affect the stomach more so than small or large bowel, is unclear. It is also interesting that despite her worsening overall hemodynamic instability, her gastric emphysema did get better and eventually resolved. One could argue that even with her low flow state, the appropriate use of antibiotics along with bowel rest gave her gastric mucosa enough time to recover and avoid a perforated viscus. To our knowledge, there has been no reported case of such a condition in patient with ECMO. With increasing use of this technology, it is only possible that we may see more of this in the future.

Management of emphysematous gastritis usually revolves around supportive care, broad spectrum antibiotics and bowel rest [12,13]. The mortality due to surgical intervention can be as high as 21% [12] and usually reserved for cases with refractory medical management, uncontrollable sepsis, or perforated viscus [14]. Our patients' gastritis resolved with non-operative management, albeit, she succumbed to multiorgan failure due to other causes.

### 4. Conclusion

Emphysematous gastritis is a potentially fatal condition. If diagnosed early, it can be managed with supportive care with surgery reserved for refractory cases. We believe our patient is a unique case of a veno-arterial ECMO causing emphysematous gastritis.

### Conflicts of interest

All authors have no conflicts of interest.

**Funding**

None.

**Ethical approval**

Case report was exempted from Institutional Review Board at the Mayo Clinic.

**Consent**

Patient is deceased. Multiple attempts have been made to contact the next of kin with no success.

**Author contributions**

A.A. was involved in data collection, analysis and interpretation. A.B. was involved in study concept and design. A.A. and A.B. were involved in writing the paper, proofreading and approving for final consideration.

**Guarantor**

Dr. Alyssa B. Chapital MD, FACS.

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