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Letter to the editor

Acute Myopericarditis Following Urgent Esophagectomy and Gastrectomy for Caustic Ingestion

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To the Editor,

In this letter to the editor, we would like to present an interesting case of a young adult patient who developed acute myopericarditis following urgent surgery for a caustic esophagitis without signs of perforation.

The patient is a 35-year-old man with no relevant medical history who was admitted to the emergency department of another institution a few hours after voluntarily ingesting an unknown quantity of ammonium hydroxide. On admission, he was clinically stable and did not need respiratory or hemodynamic support. Computed tomography (CT) scanner was performed and showed a grade II esophagitis in the proximal third of the esophagus and grade III in the distal third. Despite the presence of grade III esophagitis, the team in charge of the patient opted for clinical monitoring. After 24 hours, the patient's condition deteriorated, with the onset of fever, chest pain and tachycardia, accompanied by elevated serum inflammatory markers. A second CT scan performed 24 hours after initial admission revealed grade IIb esophagitis in the proximal third and grade III esophagitis extended to the distal third, still with no signs of perforation. Empiric antibiotherapy with piperacillin-tazobactam was initiated and the patient was transferred to our center for surgical exploration. Upon admission to our intensive care unit (ICU), 48 hours post-ingestion, the patient remained hemodynamically stable but reported light chest pain, exacerbated in the supine position. Preoperative blood tests showed a significant inflammatory syndrome (C-reactive protein: 377.5 mg/L, white blood count: 16,260/µL). No preoperative electrocardiogram (ECG) or cardiac biomarkers analysis were performed. After a thorough review of the second CT images by our surgical and radiology team, an urgent surgical exploration was performed. It confirmed extensive necrosis and a pre-perforative state of the posterior part of the stomach and the entire esophagus, without perforation or associated mediastinitis. Total gastrectomy and esophagectomy were performed, followed by esophagostomy and jejunostomy. During surgery, ST-segment elevation was observed on the monitoring, though the patient remained hemodynamically stable. Following surgery, the patient was admitted to the ICU. Upon sedation withdrawal and extubation, the patient reported intense, diffuse, non-radiating chest pain, worsening in supine position. An ECG revealed a significant and diffuse ST-segment elevation. As shown on Figure 1, a troponin-T kinetic study (Fig. 1) showed a marked elevation of the troponin levels, peaking at H+9 with a value of 2488ng/ml. Bedside transthoracic echocardiography (TTE) was normal, with no evidence of pericardial effusion. Based on the clinical presentation, ECG findings and troponin-T kinetics, a diagnosis of acute myopericarditis was suggested. No immediate treatment was initiated. On postoperative day 1, troponin and creatine kinase (CK) levels began to decline (Fig 1). The patient's clinical evolution was reassuring, with no signs of hemodynamic instability or cardiac arrhythmia. However, the patient continued to report diffuse thoracic pain, albeit less severe than the previous day. Perioperative bacteriological samples tested negative, leading to the discontinuation of antibiotic therapy. In the following days, the patient showed continued improvement, with decreasing CK and troponin levels, as well as inflammation markers. By postoperative day 3, ECG showed ST- segment normalization, and the patient was discharged from the ICU. A follow-up TTE on postoperative day 8 was normal, and the patient was deemed ready for discharge on postoperative day 10. Based on the cardiology consultant's recommendations, no specific management was deemed necessary except for a follow-up consultation at 6 months.

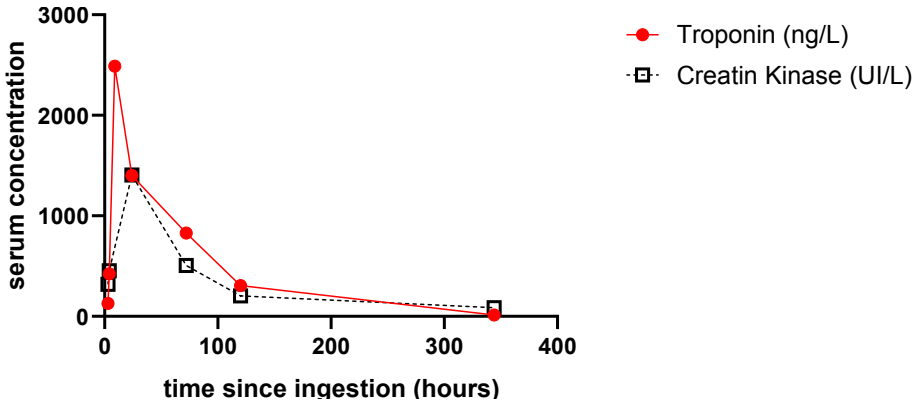

Fig. 1 — Time kinetics of troponin (continuous red line, ng/L) and creatinin kinase (black dotted line, UI/L) serum concentrations.

Acute myopericarditis is a rare condition, typically associated with viral infections or autoimmune diseases[1]. However, its occurrence following caustic esophagitis is a poorly described phenomenon, with only a few documented cases in the literature, particularly without esophageal perforation[2]. It represents a major diagnostic and therapeutic challenge for all physicians involved in perioperative care, given a high risk of systemic complications.

According to ESC diagnostic criteria for acute pericarditis[3], at least two of the following four criteria must be met for diagnosis: pericardial chest pain, pericardial friction rub, electrocardiographic changes, and/or pericardial effusion. Supporting findings include elevated inflammatory markers and imaging evidence of pericardial inflammation. In cases of myopericarditis, a concomitant rise in myocardial injury markers such as troponin and creatine kinase, is also observed. Our patient met these criteria, exhibiting elevated troponin levels, electrocardiographic changes and clinical symptoms consistent with myopericarditis. Notably, the ESC guidelines do not specifically address pericarditis secondary to caustic ingestion, highlighting a gap in the management of such cases. Our patient recovered spontaneously from his myopericarditis without specific management and did not develop cardiac arrythmias despite prolonged ICU surveillance for 72 hours post-surgery.

To the best of our knowledge, this is the first reported case of a perioperative acute myopericarditis following severe caustic esophagitis in the absence of perforation or fistula formation. One case report[2], detailed the case of sodium hypochlorite ingestion, without signs of perforation, which led to a lethal toxic myocarditis but four months after initial intoxication.

The mechanisms linking causal severe esophagitis to myopericarditis are uncertain. Inflammatory reactions secondary to caustic esophagitis may play a pivotal role in its pathogenesis. Even though no perforation has been formally confirmed, it is also important to emphasize that, in our case, surgical exploration revealed necrotic and pre-perforative state of the esophagus and the posterior part of the stomach. This condition may have played a pathophysiological role by inducing local inflammation. Transmural inflammatory phenomena have been described in the literature as potential causes of myopericarditis, such as in the case of epiphrenic diverticulum[4]. Similarly, foregut interventions, such as Toupet's fundoplication for gastroesophageal reflux, has been linked to a case of myopericarditis[5] but the exact etiology is uncertain. Therefore, it remains unclear whether myopericarditis in this case was caused by caustic esophagitis, by esophageal surgery, or both.

In summary, this clinical case illustrates that caustic esophagitis and/or its surgical management may cause a myopericarditis even in the absence of perforation. Even if the patient's clinical course was spontaneously favorable, physicians should remain vigilant for such rare complications in patients undergoing surgery for caustic ingestion, whether in the perioperative or postoperative period. Prompt diagnosis, prolonged cardiac monitoring and supportive care are crucial to achieving favorable outcomes.

Conflicts of interest: None.

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