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REVERSIBLE ST-SEGMENT ELEVATION IN THE SETTING OF ACUTE ESOPHAGITIS CAUSED BY CAUSTIC DRAIN CLEANER INGESTION

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ABSTRACT

Background: Caustic ingestion remains a significant clinical challenge, often resulting in severe transmural oesophageal injury. Given the immediate anatomical proximity of the oesophagus to the left atrium and ventricle, severe inflammation can extend to adjacent cardiac structures, potentially mimicking acute coronary syndrome.

Case description: A 22-year-old male presented following the intentional ingestion of sodium hydroxide drain cleaner. Initial endoscopy identified injury that rapidly progressed from Zargar grade 1 to 3B. Simultaneously, the patient developed acute ST-segment elevation and a marked troponin elevation of 85.15 ng/ml. Despite these findings, echocardiography confirmed normal ventricular function and no pericardial effusion. Managed conservatively with multidisciplinary support, the patient's electrocardiographic abnormalities showed progressive normalization that correlated with clinical improvement of the oesophageal injury. Unfortunately, the patient succumbed to pulmonary and vascular complications 28 days later.

Conclusion: Severe transmural esophagitis can produce dramatic, reversible cardiac manifestations through direct inflammatory extension. This case highlights the importance of recognizing non-cardiac causes of ST-segment elevation to avoid unnecessary, high-risk cardiac interventions in critically ill patients. Serial electrocardiograms may serve as a non-invasive marker reflecting the severity and progression of underlying oesophageal necrosis.

KEYWORDS

Caustic esophagitis, ST-segment elevation, Zargar classification, sodium hydroxide ingestion, troponin elevation

LEARNING POINTS

- *Mimicry of acute coronary syndrome:* This case highlights that transmural oesophageal inflammation from alkaline ingestion can cause marked ST-segment elevation and significant troponin elevation (85.15 ng/ml) due to direct inflammatory extension to the adjacent myocardium, mimicking a myocardial infarction.
- *Anatomical clinical correlation:* Understanding the posterior relation of the oesophagus to the left atrium and ventricle allows internists to recognize "oesophageal-cardiac" phenomena, potentially preventing high-risk, unnecessary cardiac catheterizations in critically ill patients with coagulopathy.
- *Prognostic monitoring:* Serial electrocardiograms can serve as a non-invasive marker of transmural disease progression. In this case, the worsening of Zargar grades directly paralleled the evolution of electrocardiographic abnormalities.

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INTRODUCTION

Caustic ingestion affects 5,000-15,000 Americans annually, with alkaline agents like sodium hydroxide causing severe injury through liquefaction necrosis and transmural damage[1]. The oesophagus lies immediately posterior to the left atrium and inferior to the left ventricle, separated only by thin tissue planes, allowing severe oesophageal inflammation to affect adjacent cardiac structures. While ST-segment elevation typically suggests acute coronary syndrome, non-cardiac conditions including severe gastrointestinal pathology can produce similar findings[2]. Previous reports document electrocardiographic changes with oesophageal conditions, but significant ST-elevation with marked troponin elevation in caustic esophagitis from alkaline ingestion remains rarely reported[3]. We present a case of severe caustic oesophageal injury with progressive endoscopic deterioration associated with reversible ST-segment elevation and troponin elevation.

CASE DESCRIPTION

A 22-year-old male with a past medical history significant for bipolar disorder type 1, post-traumatic stress disorder, attention deficit hyperactivity disorder, and anxiety presented to our institution as a transfer following the intentional ingestion of three large swallows of drain cleaner for self-harm. The patient's psychiatric history was notable for multiple prior suicide attempts, including hanging and jumping from a hotel roof three months earlier. Initial presentation at an outside emergency department revealed a critically ill patient who was acutely altered and vomiting blood. He was intubated for airway protection after multiple episodes of hematemesis, sedated, and transferred to our intensive care unit. A computed tomography scan demonstrated severe oedema involving the oesophagus, stomach, and small bowel, raising concern for transmural injury.

In the intensive care unit, while being monitored on telemetry, acute ST-segment elevation was noted, prompting an electrocardiogram (ECG) that confirmed ST-segment elevation *(Fig. 1A)*. A broad differential was considered, including acute coronary syndrome, pericarditis, and non-cardiac causes of ST-segment elevation. Initially troponin I was markedly elevated at 85.15 ng/ml. Despite these findings, the patient remained hemodynamically stable without shock. Transthoracic echocardiography demonstrated normal left ventricular systolic function, a moderately dilated right ventricle, and a patent foramen ovale, with no evidence of pericardial effusion or wall motion abnormalities. Based on normal cardiac function and the clinical context, our working diagnosis was ST-segment changes secondary to esophagitis. Serial ECGs continued to demonstrate ST-segment changes *(Fig. 1B and C)*, though troponin levels began to downtrend. Initial management was supportive, including ventilator management, appropriate analgosedation, and antimicrobials, with multidisciplinary consultation from gastroenterology and thoracic surgery. The patient was maintained strictly nil per OS.

Endoscopy performed on day 2 revealed extensive caustic injury throughout the upper gastrointestinal tract, including severe oedema and ulceration of the oropharynx *(Fig. 2A)*. The oesophagus showed a gradient of injury severity: Zargar grade 1 caustic esophagitis with oedema and hyperaemia in the upper third *(Fig. 2B)*, grade 2A injury with superficial ulceration, erosions, friability, blisters, exudates, haemorrhages, and whitish membranes in the middle third *(Fig. 2C and D)*, and grade 3A injury with small scattered areas of multiple ulceration and necrosis with brown-black or greyish discoloration in the distal oesophagus *(Fig. 2E)*. The stomach demonstrated grade 3B injury with extensive necrosis. Repeat endoscopy on day 4 demonstrated significant progression of the caustic injury, with Zargar

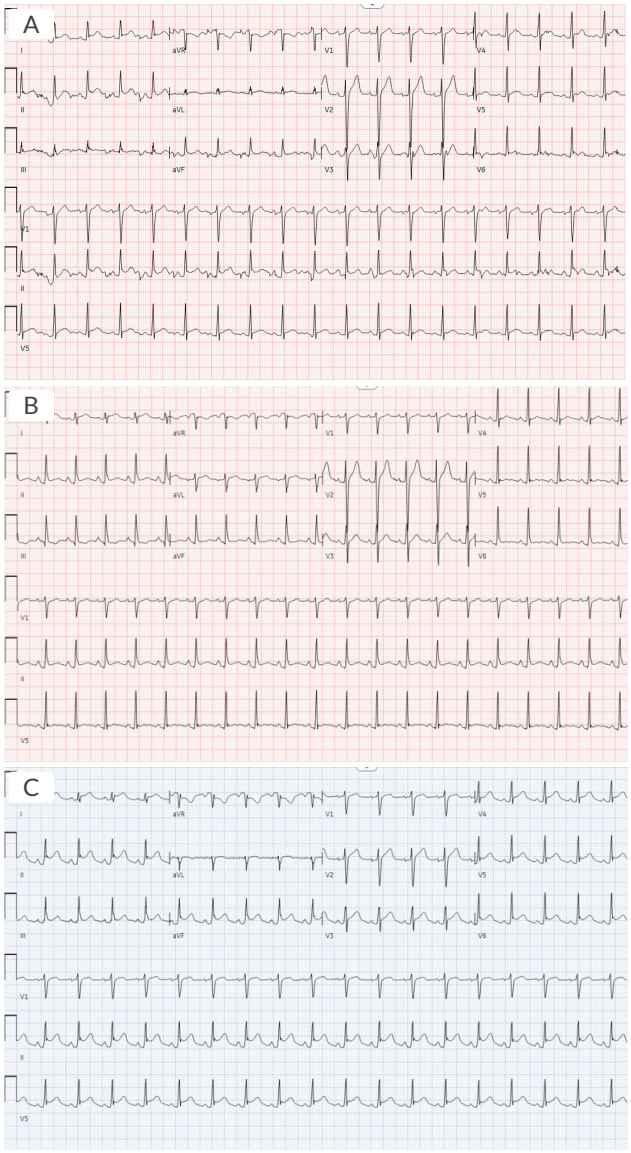


Figure 1. *Serial ECGs during the acute phase of caustic esophagitis: A) Initial ECG at 18:42 on hospital day 1 showed ST-segment elevation in inferior and lateral leads; B) Follow-up ECG at 22:57 on hospital day 1 with persistent ST-segment abnormalities; C) ECG at 10:55 on hospital day 2 demonstrated continued ST-segment changes. Abbreviations: ECG, electrocardiogram.*

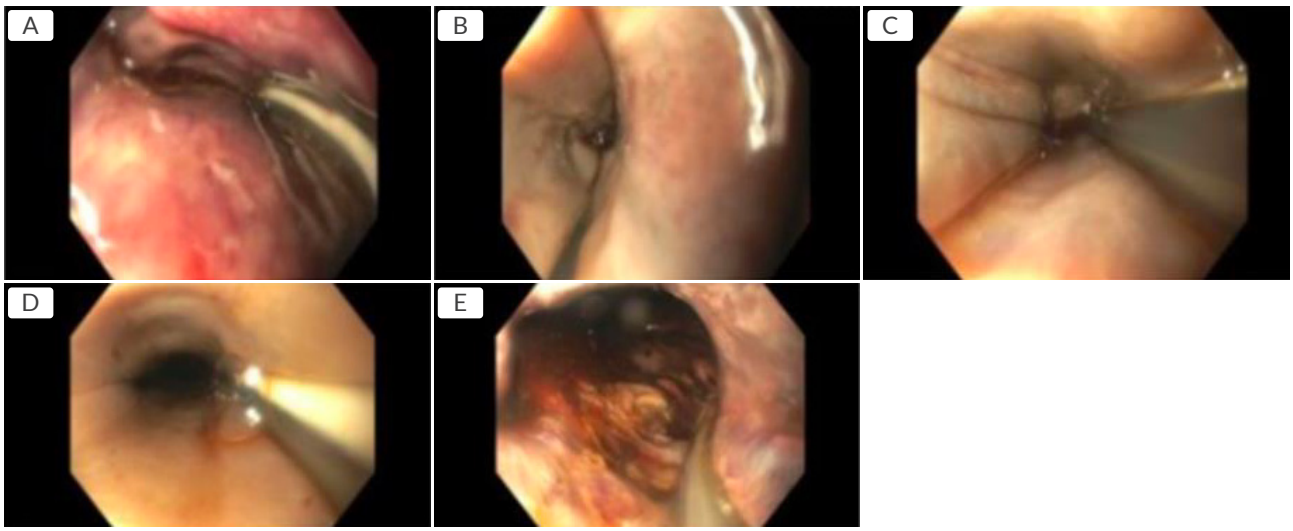


Figure 2. Day-2 esophagogastroduodenoscopy showing caustic injury from the A) oropharynx through the B) upper, C-D) middle, and E) lower thirds of the oesophagus.

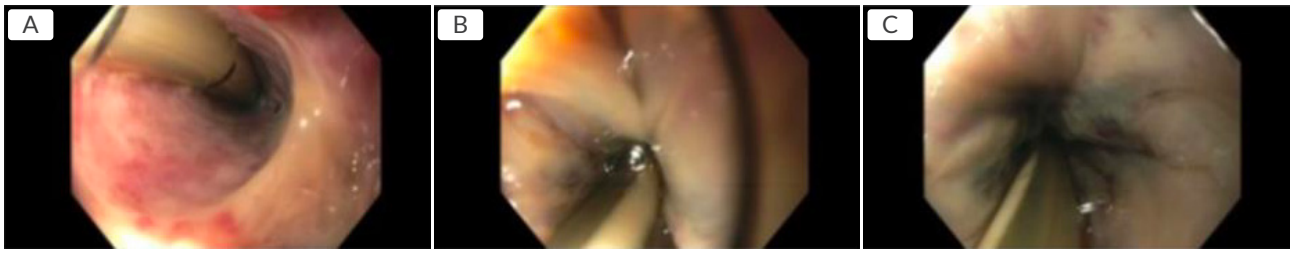


Figure 3. Day 4 repeat esophagogastroduodenoscopy demonstrates progressive, high-grade caustic injury. Zargar grade 3A esophagitis was identified at the cricopharyngeus muscle. A) Grade 2A injury in the upper third of the oesophagus, B) grade 3B injury in the middle third of the oesophagus, and C) grade 3B injury in the lower third of the oesophagus.

grade 3A caustic esophagitis at the cricopharyngeus muscle, grade 2A injury in the upper third, and progression to grade 3B injury in both the middle and lower thirds of the oesophagus *(Fig. 3)*. Despite these severe findings, the patient remained hemodynamically stable with no clinical evidence of perforation.

Serial ECGs obtained throughout hospitalization showed progressive improvement, with the study obtained on day 7 demonstrating near-complete resolution of the previously noted ST-segment abnormalities *(Fig. 4)*. The patient had a prolonged hospital stay of 28 days complicated by pneumonitis, acute respiratory distress syndrome, acute kidney injury requiring dialysis, and hospital-acquired infections. He underwent tracheostomy, which was complicated by a tracheoinnominate fistula, and unfortunately died after experiencing massive airway haemorrhage.

DISCUSSION

This case demonstrates how progressive caustic esophagitis can produce dramatic, reversible ST-segment elevation without primary cardiac disease, illustrating a critical phenomenon gastroenterologists must recognize when managing caustic ingestions[1,2]. While it is known that oesophageal conditions like reflux or spasm can cause ECG changes[3], this case is unique due to the extreme troponin elevation (85.15 ng/ml) and the direct correlation with Zargar-staged endoscopic deterioration. The intimate anatomical relationship between the oesophagus and cardiac structures explains this presentation, as the oesophagus courses immediately posterior to the left atrium and along the inferior surface of the left ventricle, separated only by thin tissue planes. In severe transmural oesophageal inflammation, as seen in our patient's progression from Zargar grade 1 to grade 3B injury[4], the inflammatory process can extend directly to adjacent cardiac structures, mimicking acute coronary syndrome. This mechanism accounts for both the ST-segment elevation and marked troponin elevation, with gradual normalization paralleling clinical improvement.

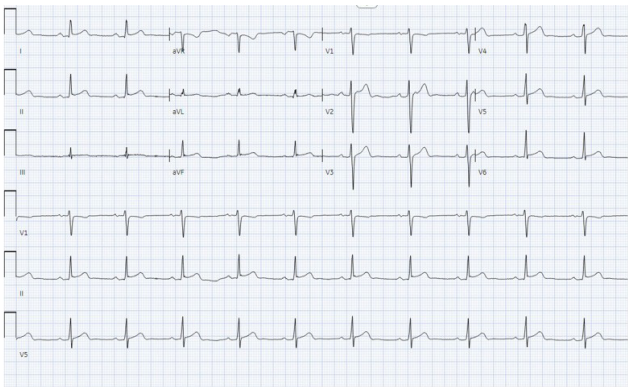


Figure 4. Day 7 follow-up 12-lead electrocardiogram demonstrating sinus rhythm with resolution of the initial ST-segment elevations.

The differential diagnosis of ST-segment elevation extends beyond acute myocardial infarction to include pericarditis, myocarditis, and severe oesophageal pathology. What is significant here is the absence of reciprocal ST depression and the lack of Q-wave evolution, which, alongside normal ventricular function on echocardiography, points toward an inflammatory rather than ischemic aetiology. Because troponin elevation in the setting of systemic inflammation and critical illness is a recognized but complex finding[5], its presence here likely reflects direct myocardial irritation from the contiguous oesophageal necrosis. This case underscores that endoscopic progression can parallel cardiac manifestations, suggesting serial ECGs may serve as non-invasive markers of disease severity and treatment response.

These findings directly support our conclusion that aggressive management should remain focused on the oesophageal injury[6]. Recognition of this association is crucial for preventing unnecessary cardiac catheterization in critically ill patients with coagulopathy, hemodynamic instability, and perforation risk. The complete reversibility of these changes with conservative management supports a non-invasive approach when the clinical context of caustic ingestion is clear[7]. Early recognition should prompt electrocardiographic evaluation and echocardiography to exclude primary cardiac pathology while maintaining focus on the underlying oesophageal injury.

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