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Necrotizing Infection of the Heart

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Abstract

A case of necrotizing infection of the heart is presented. A 70-year-old woman presented with vague chest and abdominal pain. CT of the abdomen and pelvis was initially obtained, which demonstrated gas in the myocardium of the left ventricle. Subsequent chest CT, endoscopy, and abdominal surgical exploration did not reveal perforated viscus or diaphragm compromise. At median sternotomy, the inferior wall of the heart was found to be necrotic. Culture of the excised tissue grew *E. coli*. The patient expired shortly after surgical exploration.

Keywords

Necrotizing infection; computed tomography; cardiac infection; pneumomyocardium; CT chest; Necrotizing fasciitis

1. Introduction

Necrotizing infections are life-threatening infections that commonly affect the perineum, extremities, and sites with pre-existing wounds or surgical incisions. Affected patients are often immunosuppressed, and there is a strong association with diabetes. When imaging is used in diagnosis, computed tomography (CT) may show gas that dissects affected tissue or fascial planes and can define the extent of disease [1]. We present an unusual case of

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necrotizing infection of the inferior wall of the heart in a patient with no known predilection for cardiac infection.

2. Case presentation

A 70-year-old woman presented to the Emergency Department with three days of vague chest and abdominal pain. Approximately 1-month prior, she was involved in a low-speed motor vehicle collision for which she did not seek medical attention or have any resulting or suspected injury. Past medical history included coronary artery disease, gastroesophageal reflux disease, and melena. Past surgeries included laparoscopic cholecystectomy and a right shoulder arthroplasty, neither of which were recent. Laboratory panels revealed leukocytosis (21,000/uL) and acute renal failure (blood urea nitrogen 106 mg/dL, serum creatinine 3.2 mg/dL). An initial electrocardiogram showed a possible non-ST elevation myocardial infarction, although it was of questionable acuity – the pattern did not change through the duration of the hospital admission. Troponins were mildly elevated and trended upward the first few serial measurements; creatine kinase MB measurements were within normal limits. Initial urine cultures grew *Klebsiella oxytoca* and blood cultures were negative. The initial chest radiograph revealed a subtle oval lucency at the inferior midline aspect of the cardiac silhouette (Figure 1). Given the complaint of abdominal pain associated with leukocytosis, CT of the abdomen and pelvis was obtained which partially profiled apparent pneumomyocardium at the inferior aspect of the heart along with a small to moderate pericardial effusion (not pictured; findings illustrated in Figure 2). There were no acute findings in the abdomen. There was no CT evidence of diaphragm compromise, esophageal perforation, perforated gastroduodenal ulcer, or a fistulous communication to the heart. A subsequent CT of the chest better characterized the cardiac findings (Figure 2). The focus of free air was isolated to the inferior wall of the left ventricle. A transthoracic echocardiogram demonstrated the pericardial effusion, dyskinesia with segments of akinesis of the inferior wall, and a mixed echogenicity appearance of the inferior wall.

With the concern for an occult gastroesophageal perforation and/or penetrating ulcer disease, a barium esophagram and upper gastrointestinal study were obtained and found to be normal (not pictured). Upper endoscopy revealed gastritis and duodenitis but no evidence of perforation. The patient deteriorated clinically and required vasopressors. There was a persistent concern for ulcer disease causing a fistulous tract, which prompted abdominal surgical exploration immediately prior to median sternotomy. Exploratory laparotomy revealed no perforated abdominal viscus, diaphragm compromise, or other abnormality. Median sternotomy revealed frank necrotic tissue at the inferior wall of the heart, which was excised and repaired with a Dacron patch. Surgical pathology confirmed myonecrosis with areas of fibrosis and scarring. *E. coli* was colonized from the necrotic heart tissue. The patient did not clinically improve following resection of the necrotic heart tissue and expired shortly after the procedure.

The underlying cause of this necrotic cardiac tissue with associated myocardial air and colonization of *E. coli* was never discovered. Surgical pathology of the resected heart tissue demonstrated scarring, suggesting that this necrotizing infection originated from a site of

prior myocardial infarction. The patient's low-speed motor vehicle collision one-month prior, urinary tract infection, and acute renal failure may have been precipitating factors.

3. Discussion

We believe this case to be the first reported case of a necrotizing infection involving the myocardium. 'Necrotizing infection' is likely the best descriptive term as 'necrotizing fasciitis' may be an imperfect descriptor given the myocardial location; it may also be termed a 'necrotizing myositis.' The authors consider this necrotizing infection along the same disease spectrum as necrotizing fasciitis given the surgical and CT findings. Controversy exists in naming and classifying necrotizing infections [2]. Gas forming infections in the pericardial space have been well described [3, 4] and implantable cardiac devices have resulted in superficial soft tissue necrotizing fasciitis, such as the chest wall reservoir site for pacemaker generators [5], but no prior reported cases of necrotizing infection within the myocardium have been reported to our knowledge. A search of [PubMed.gov](https://pubmed.ncbi.nlm.nih.gov/) and Google Scholar using terms such as 'myocardial necrotizing infection,' 'necrotizing infection of the heart,' 'necrotizing myositis of the heart,' 'necrotizing fasciitis of the heart,' 'pneumomyocardium,' and related search terms found no similar cases. One case of pneumomyocardium unrelated to an infectious etiology has been reported in a premature neonate who died in the first month of life. In that case, Lee and Hutchins [6] describe a pocket of air within the myocardium interventricular septum, which was not associated with any inflammatory infiltrates, organisms, or necrotic debris. The authors postulated this post-mortem finding was due barotrauma given the patient's bronchopulmonary dysplasia and prior development of a pneumothorax associated with prolonged intubation [6]. Our literature search did not reveal any additional cases of pneumomyocardium.

Pneumopericardium may mimic pneumomyocardium and tends to be the sequela of surgery or trauma. Traumatic causes include blunt and penetrating trauma, barotrauma, and iatrogenic following thoracic or cardiac surgery. Spontaneous pneumopericardium is rare compared to posttraumatic pneumopericardium but is a more well-described finding compared to pneumomyocardium [7]. Prior described associations between perforated ulcers and other causes of fistulous connections between the esophagus or stomach and the pericardium drove the ongoing clinical suspicion that the free gas in the myocardium was due to a perforated gastrointestinal viscus. Spontaneous causes of pneumopericardium include perforated esophageal or gastric tumors and perforated peptic ulcers [7, 8]. Necrotizing infections with gas producing organisms have been described in the pericardial space and associated with pneumopericardium [3, 4]. In such cases, the underlying cause is often not found. Direct extension from an infectious ascending aortic aneurysm has been reported in at least one case [3].

4. Conclusion

A necrotizing infection of the heart with a myocardial location has not been previously described to our knowledge. Oral contrast fluoroscopy studies, direct visualization with endoscopy, and direct external visualization with surgical laparotomy and median

sternotomy failed to reveal any perforated viscus or diaphragm compromise. Myocardial scarring seen on surgical pathology along with the patient's coronary artery disease and electrocardiogram findings may suggest that this necrotizing infection originated from a site of prior myocardial infarction. This case illustrates that necrotizing infection can affect the heart and manifest as pneumomyocardium on CT.

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Highlights

- CT demonstrated pneumomyocardium at the inferior wall of the heart.
- Appearance at surgery, surgical pathology, and positive culture suggested necrotizing infection.
- Pneumomyocardium is a rare CT finding; pneumopericardium is more common.

Figure 1. Initial portable chest radiograph demonstrates a subtle oval lucency (arrow) projecting at the midline inferior aspect of the cardiac silhouette.

Figure 2.

Computed tomography (CT) of the chest without intravenous contrast (contrast was withheld in all CT examinations due to impaired renal function)(A-C). Axial (A, B) CT images along with coronal (C) reconstructions confirm that the pneumomycardium is confined to the inferior wall (A-C, thick arrows). A small to moderate pericardial effusion is demonstrated (A-C, thin solid arrows). Small bilateral pleural effusions (A, B), coronary artery calcifications (A-C), and calcifications of the thoracic aorta (A-C) are also noted.