Necrotizing Fasciitis Mimicking Acute ST-Elevation Myocardial Infarction: A Case Report

Hao-Neng Fu[1], Wei-Hsian Yin[1,2], Tien-Ping Tsao[1,3]

[1]*Division of Cardiology, Cheng Hsin General Hospital, Taipei, Taiwan*
[2]*Faculty of Medicine, School of Medicine, National Yang-Ming University, Taipei, Taiwan*
[3]*Division of Cardiology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan*

Abstract

Soft-tissue infections involve microbial invasion of the layers of the skin and underlying soft tissues. The patient's initial presentation can vary from local symptoms to profound sepsis and systemic inflammatory response syndrome (SIRS). Acute ST-elevation myocardial infarction (STEMI) secondary to soft tissue infection and SIRS has rarely been reported, and the exact mechanism of myocardial injury remains unclear. We present the case of a 59-year-old gentleman who developed SIRS secondary to extensive soft tissue infection of the left chest wall and who initially presented with acute STEMI with heart failure. The emergency coronary angiography revealed no coronary artery lesions. He subsequently developed multiple organ dysfunction syndromes, including acute respiratory failure, acute liver dysfunction, and acute kidney dysfunction. Fortunately, the patient made a full recovery due to quick diagnosis and intensive treatment. Medical practitioners should be aware of acute myocardial injury caused by SIRS that prompts aggressive treatment of the underlying disease.

Keywords: type 2 myocardial infarction, ST elevation myocardial infarction, sepsis, sepsis-induced cardiomyopathy, streptococcal infection related perimyocarditis

Introduction

Soft-tissue infections are clinical entities of variable presentation, etiology, and severity. They involve microbial invasion of the skin and underlying soft tissues and range from mild infections, such as pyoderma, to severe life-threatening infections, such as necrotizing fasciitis. The affected area may have erythema, edema, warmth, and pain. In severe infection, the patient may present with systemic inflammatory response syndrome (SIRS).

A variety of severe clinical insults, such as infection, pancreatitis, trauma, surgery, and other critical illness, can lead to SIRS. The diagnosis of SIRS requires the presence of at least two of the following conditions: (1) body temperature > 38°C or < 36°C; (2) heart rate > 90 beats/minute; (3) respiratory rate > 20 breaths/minute or partial pressure of CO_2 < 32 mmHg; (4) Leukocyte count

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Address for correspondence: Dr. Hao-Neng, Fu
Division of Cardiology, Cheng Hsin General Hospital; No. 45, Cheng Hsin St., Beitou, Taipei, Taiwan
Tel: +886-2-2826-4400 (Office), +886-928324554 (Mobile); E-mail: kklwin.9985@gmail.com

> 12000 or < 4000/μL or > 10% immature forms or bands.[1]

SIRS is a clinical process characterized by generalized inflammatory hyper-reactivity caused by various severe clinical insults triggered by infectious factors and non-infectious host stimulatory agents.

Extensive capillary leakage and mesenchymal edema caused by inflammatory mediators and cytokines reduce tissue perfusion pressure to vital organs and ultimately result in multiple organ dysfunction syndrome (MODS) or multiple organ failure (MOF). When SIRS progresses to MODS and MOF, the mortality rate increases to 30-80%.[2]

Cardiovascular complications of SIRS include pericardial and myocardial involvement with myocardial dysfunction and even shock. Sepsis-induced cardiomyopathy (SICM) or sepsis-induced myocardial dysfunction (SIMD) is a reversible myocardial dysfunction in patients with sepsis. Its prevalence is not uncommon and reportedly 10-70% in septic patients.[3] In the setting of SIRS secondary to chest wall soft tissue infection, the patient can present with chest pain and ST-T changes on electrocardiography (ECG). However, reports of soft tissue infection involving the left chest wall accompanied by electrocardiographic ST-elevation and cardiac enzyme rise mimicking acute STEMI are rare in the literature.

We report a case of a 59-year-old gentleman who initially presented with STEMI and acute heart failure but was eventually found to have SIRS complicating extensive soft tissue infection. The patient developed MOF, including acute respiratory failure, acute liver dysfunction, and acute kidney dysfunction.

Case report

A 59-year-old gentleman presented at our emergency department (ED) with intermittent left chest pain for three days. The patient had blunt trauma to the left chest wall four days before ED presentation. Focal pressure could aggravate the chest pain, which did not radiate to the arm, neck, or back.

The physical examination at the ED revealed a temperature of 37.5°C, while the blood pressure was 90/52 mmHg and the heart rate was 110 beats/min. There were skin rashes over the left lateral chest wall with erythematous change and tenderness. The ECG showed impressive ST-segment elevation in leads I, II, aVL, and precordial V2 to V6 leads (Figure 1A). Chest radiography showed borderline cardiomegaly and cephalization of pulmonary vasculature. Laboratory studies revealed neutrophil leukocytosis with elevated band form, while serum creatinine level, liver enzymes, and cardiac enzymes were elevated (Table 1). Echocardiography at the ED showed global hypokinesia of the left ventricle with an ejection fraction of 40%. There was no evidence of valvular dysfunction, pericardial thickening, or pericardial effusion.

Under the impression of acute extensive anterior wall myocardial infarction, we immediately sent the patient to the cardiac catheterization laboratory. We performed coronary angiography that demonstrated insignificant luminal irregularity at the mid-right coronary artery, thereby excluding coronary atherosclerosis-related STEMI (Figure 1B). Chest computed tomography (CT) showed marked soft tissue swelling with diffuse fat stranding, localized fluid collection over the left lateral chest wall and bilateral pleural effusions, comparable with necrotizing fasciitis (Figure 2A).

Based on the clinical and imaging findings, the patient was diagnosed with SIRS secondary to necrotizing fasciitis and MODS, including acute myocardial injury, acute hepatic and renal dysfunction, and acute respiratory failure.

Although we administered carbapenem to control the infection, the patient developed dyspnea, oxygen desaturation, and hypotension. He underwent endotracheal intubation with mechanical ventilator support and intravenous inotropic infusion. The involved area over the left lateral chest wall progressively extended to

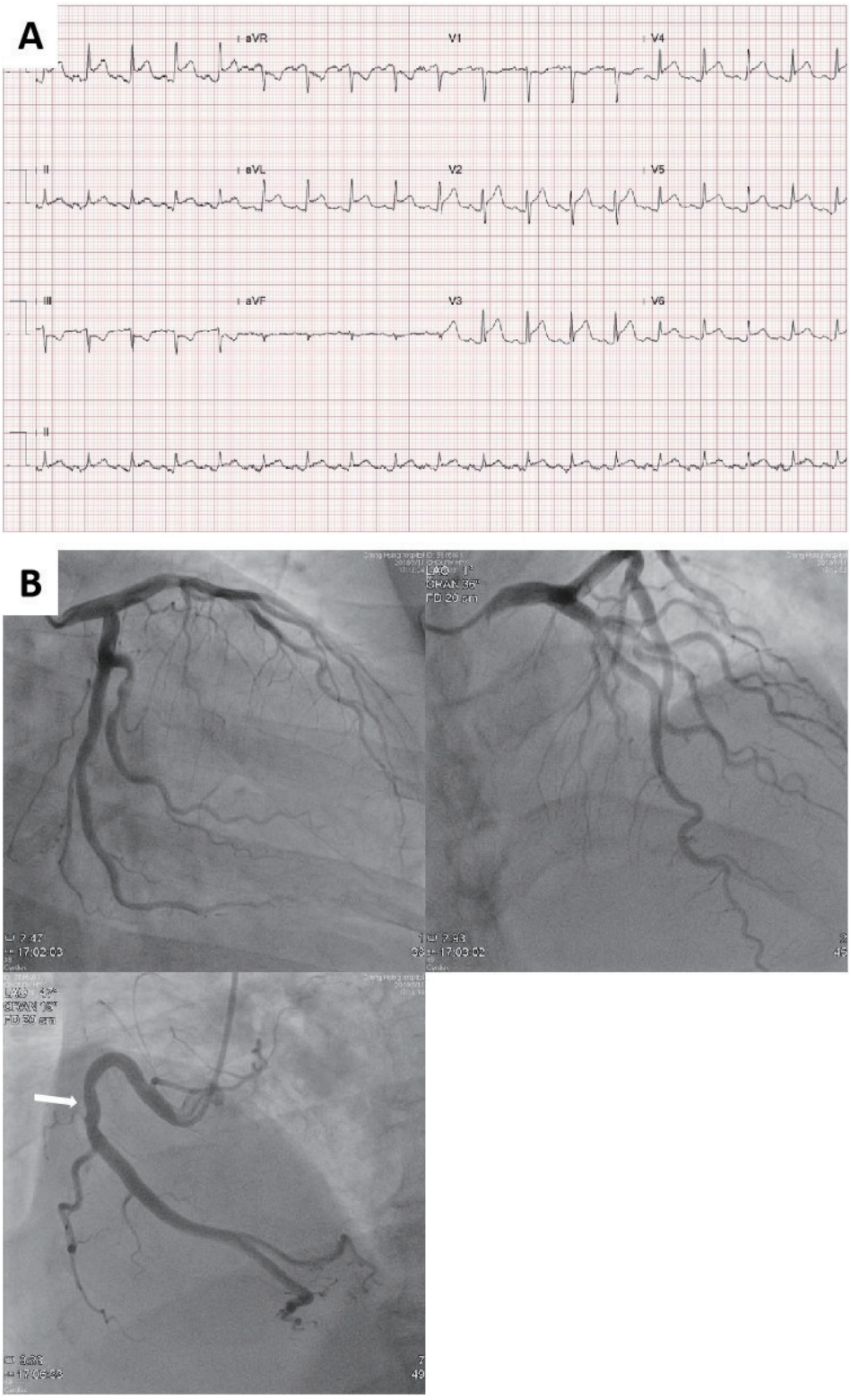

Figure 1. A. Electrocardiogram on arrival to the emergency department showed ST-segment elevation in leads I, II, aVL and V2-6. **B.** Selective coronary angiography demonstrated insignificant coronary artery lesion at mid-right coronary artery (arrow).

Table 1. Laboratory studies

	Result	Reference range
White blood cells (WBC)	12 x 103 cells/uL	4.0 - 10 x 103 cells/uL
Neutrophil	90%	40 - 70%
Band neutrophil	3.6%	-
Lymphocytes	1.8%	20 - 10%
Blood urea nitrogen (BUN)	74.6 mg/dL	8 - 20 mg/dL
Serum creatinine	7.31 mg/dL	0.64 - 1.27 mg/dL
Total bilirubin	6.4 mg/dL	0.4 - 2.0 mg/dL
Aspartate transaminase (AST)	182 IU/L	5 - 50 IU/L
Alanine transaminase (ALT)	52 IU/L	5 - 50 IU/L
Troponin-I	28.75 ng/mL	< 0.056 ng/mL
Creatine kinase-MB (CK-MB)	> 300 ng/mL	< 3.6 ng/mL
Creatine phosphokinase (CPK)	1867 IU/L	49 - 397 IU/L

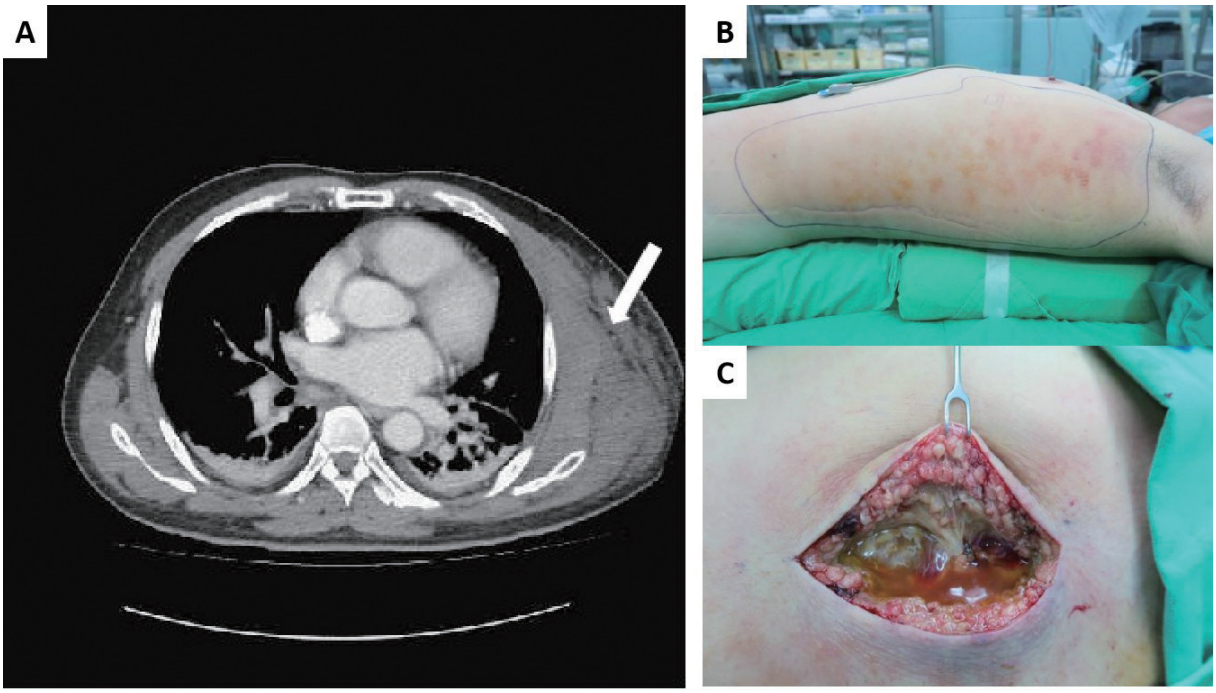

Figure 2. A. Contrast enhanced computed tomography scan of chest showed marked soft tissue swelling with fat stranding (arrow) and bilateral pleural effusions. **B.** Extended erythematous and edematous change with blisters formation over left lateral chest wall. **C.** Wild-spreading necrosis of underlying soft tissue and typical dish-washer gray exudate.

the back, and blisters appeared on the overlying skin on the second day of hospitalization. Due to inadequate response to the empiric antibiotic, surgical debridement to the left chest wall was performed (Figures 2B & 2C). The blood and wound discharge culture showed streptococcus pyogenes (Group A streptococcus). We changed the antibiotic to cefpirome and teicoplanin in response to the blood culture and sensitivity results. The patient received emergency hemodialysis due to acute renal failure with severe metabolic acidosis. Cardiac troponin level peaked on the 2nd day of hospitalization at 200 ng/mL, and then gradually decreased. By the 5th day of hospitalization, the ST segment had normalized on EKG. His condition improved progressively, and he left the hospital having made a full recovery.

Table 2. Third universal definition of myocardial infarction (MI)

Type of MI	Description
Type 1	Spontaneous MI related to primary coronary event
Type 2	MI secondary to an ischemic imbalance
Type 3	MI resulting in death when biomarker values are unavailable
Type 4a	MI associated with percutaneous coronary intervention (PCI)
Type 4b	MI related to stent thrombosis
Type 5	MI related to coronary artery bypass graft

Discussion

Few case reports presenting SIRS-induced myocardial infarction have been published in the literature. We initially diagnosed the patient with STEMI based on the elevation of cardiac enzyme levels and ECG findings. We also considered the possibility of acute pericarditis due to more extensive ST-segment elevation. The echocardiography at the ED showed depressed left ventricular function and no thickening or effusion of the pericardium. Additionally, the laboratory data revealed elevation of cardiac enzymes, including troponin I, creatine kinase-MB (CK-MB), and creatine phosphokinase (CPK). Based on the clinical findings, we favored the diagnosis of STEMI at ER.

The third universal definition of myocardial infarction classifies various types of myocardial infarction by pathogenesis, as described in Table 2.[4] The patient was in type 2 myocardial infarction, and cardiac damage induced by SIRS was the most probable mechanism of STEMI.

The chest wall infection spread rapidly and could not be controlled with initial empiric antibiotic treatment. The patient underwent surgical debridement on the affected chest wall due to the aggravated infection. This revealed extensive necrosis of the soft tissue of the left lateral chest wall, consistent with necrotizing fasciitis. Blood and wound cultures were positive for streptococcus pyogenes (Group A streptococcus). Necrotizing fasciitis is a life-threatening condition. It manifests as an insidiously developing soft-tissue infection characterized by widespread fascial necrosis. Group A streptococcus is the most common pathogen and is characterized by extensive tissue destruction and aggressive clinical course with a high mortality rate (38 to 45%).[5] Reversible cardiomyopathy can also be seen in group A streptococcal necrotizing fasciitis with streptococcal toxic shock syndrome.[6-8] Another uncommon cardiac complication of streptococcal infection is perimyocarditis. The patient may present with chest pain, elevated ST segment in EKG and elevated cardiac troponin, which can mimic STEMI.[9]

Local and systemic inflammation may play an essential role in the acute involvement of pericardium and myocardium, with the ECG pattern mimicking STEMI.[10] Invasive coronary angiography should be considered to obtain the correct diagnosis and avoid thrombolytic therapy, potentially avoiding lethal complications.

Sepsis-induced cardiomyopathy (SICM) or

sepsis-induced myocardial dysfunction (SIMD) can manifest as transient myocardial dysfunction in septic patients. During sepsis, multiple myocardial depressant factors such as cytokines & complement factors cause myocardial dysfunction. Vascular leakage and local inflammation also result in heterogeneous microvascular flow and myocardial edema, ultimately leading to myocardial dysfunction.[11,12]

In summary, SIRS secondary to sepsis can lead to acute STEMI and acute heart failure. Both myocardial ischemia and injury resulting from the reduced tissue perfusion and direct myocardial insult by pathogens and endogenous factors can lead to acute myocardial infarction and myocardial dysfunction. At our patient's initial presentation, the manifestation of skin lesions was not noticeable. The left chest pain with ST-elevation on ECG supported the diagnosis of acute STEMI. The strategies of prompt diagnosis of the STEMI-mimicking condition and employment of aggressive treatment including surgical debridement with excision of all necrotic tissues were crucial in stabilizing the patient's condition. Additionally, antibiotic therapy, intensive care support, and early parenteral nutrition are indispensable in critically ill patients.

Conclusion

In our case, the patient presented with necrotizing fasciitis with SIRS which led to a clinical mimicking of acute STEMI. Early coronary angiography to exclude acute coronary occlusion was required to enable correct diagnosis and effective treatment.

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