

Dialysis catheter-related sepsis resulted in infective endocarditis, septic pulmonary embolism and acute inferolateral STEMI: a case report

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Background

Embolic myocardial infarction is an uncommon but increasingly recognized complication of infective endocarditis (IE). Although the incidence is low and ranges from 1% to 10%, the mortality rate is high (64%). The characteristics of septic embolism on presentation are nonspecific and usually are unrecognized by clinicians. This case report aims to build a high index of suspicion among clinicians for IE presenting with the complication of embolic myocardial infarction especially in patients with indwelling venous catheters.

Case Summary

A 62-year-old woman with end-stage renal disease on haemodialysis presented with shortness of breath and desaturation. Her history was significant for end-stage renal disease managed with regular haemodialysis by a right-sided double-lumen tunnelled catheter. An initial diagnosis was made of pulmonary embolism, and management with intravenous heparin was initiated. She subsequently developed inferolateral ST-elevation myocardial infarction, and treatment with percutaneous coronary intervention to the posterior descending artery failed. Then, the patient developed complete heart block, aortic valve vegetation, acute severe aortic regurgitation, and shock.

Discussion

Acute coronary syndrome is usually an early and uncommon complication of IE and the risk of embolism decreases after antibiotic therapy is initiated. Due to the low incidence of coronary events in IE, only case reports have been published. Most patients with septic pulmonary embolism have a presentation similar to that for pneumonia. The diagnosis is therefore often delayed, which consequently influences prognosis. Our case report presents an example of IE-related multiple systemic embolization with poor patient outcome due to delayed diagnosis.

Keywords

Infective endocarditis • Myocardial infarction • Sepsis • Septic pulmonary embolism • Case report

ESC Curriculum

2.2 Echocardiography • 2.1 Imaging modalities • 4.11 Endocarditis

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Learning points

- To build a high index of suspicion for disseminated septic embolization in the presence of an infection source. This will allow us to diagnose the disease early which will improve the patient outcome. Our case report presents a patient with IE-related multiple systemic embolization for which the delay in treatment due to a missed diagnosis resulted in a poor outcome.
- To highlight an uncommon mechanism by which sepsis can cause acute coronary syndrome.
- To be able to make a differential diagnosis of pulmonary embolism in the setting of venous catheters and sepsis.

Introduction

Infective endocarditis (IE) is associated with a high in-hospital mortality rate and a high incidence of embolic events. Systemic embolism is a common complication, most often involving the nervous system, spleen, kidney, and mesenteric arteries, whereas acute coronary syndrome is rarely encountered. Acute myocardial infarction caused by septic embolism from IE is an infrequent and fatal condition. This case report presents a patient with IE-related multiple systemic embolization with a poor outcome.

Timeline

Chronological series of the patient events.

Patient past medical history (eight months before presentation)	End-stage renal disease on regular haemodialysis via right-sided double-lumen tunnelled catheter.
19 December 2021	Presented to our emergency department due to progressive shortness of breath and cough for 3 days. CT pulmonary angiogram was done and revealed bilateral segmental and subsegmental pulmonary embolism. The Patient was admitted to the ICU.
21 December 2021 9 p.m.	The Patient developed hypotension, ST-segment elevation in the inferolateral leads. Elevated cardiac enzymes. Echocardiography revealed: dilated right side, significant TR, normal aortic valve.
21 December 2021 10 p.m.	The Patient was shifted to the Cath lab and Coronary angiogram was done and revealed: total PDA occlusion with failure of multiple attempts of aspiration.
23 December	The patient suddenly developed complete heart block, which required temporary pacemaker insertion. In addition, she became feverish and pus oozing from the haemodialysis catheter site was observed.
24 December 11:30 a.m.	Transesophageal Echocardiography was done and revealed: aortic valve vegetations with acute severe AR, right Atrial appendage mass, patent foramen ovale.
24 December 1 p.m.	Patient became shocked, developed VF and VT then, PEA and then died.

Case presentation

A 62-year-old woman presented to our emergency department with progressive shortness of breath and cough for 3 days. Initial physical examination revealed that the patient was tachypneic, and vital signs were temperature 37.7° C, heart rate 110 beats per minute, blood pressure 100/55 mm Hg, and oxygen saturation 85% on room air. Chest auscultation revealed clear lung fields and a pansystolic murmur over the right lower parasternal area. Her history was significant for end-stage renal disease managed with regular haemodialysis by a right-sided double-lumen tunnelled catheter. At this time, the differential diagnosis included pulmonary embolism, fluid overload, and SARS-CoV2 infection.

Computed tomography pulmonary angiography was performed and revealed bilateral segmental and subsegmental pulmonary embolism (figure 1). Polymerase chain reaction testing for SARS-CoV2 was negative. The patient was admitted to the intensive care unit, and intravenous heparin infusion 18 U/kg/h was initiated and up titrated till 28 U/kg/h to achieve APTT of 80 ms. She subsequently stabilized. However, 2 days later, she suddenly developed hypotension with blood pressure of 70/40 mm Hg.

Transthoracic Echocardiography (TTE) revealed a dilated right-side, significant tricuspid regurgitation, pulmonary artery systolic pressure was 56 mmHg with normal aortic valve morphology and flow. Electrocardiography showed ST-segment elevation in the inferolateral leads (figure 2). Laboratory studies showed a high-sensitive cardiac troponin-T values that increased from 5000 ng/mL to 21 957 ng/mL (reference range: 0–18 ng/mL). The patient was immediately moved to the catheterization laboratory for coronary angiography, which revealed total occlusion of the posterior descending artery. Multiple attempts for aspiration and balloon inflation to restore the flow failed. The patient then was relocated to the ICU.

Two days later, the patient suddenly developed complete heart block, which required temporary pacemaker insertion. In addition, she became feverish with a temperature of 38.9° C, and pus oozing from the haemodialysis catheter site was observed. Line sepsis was diagnosed, and the catheter was removed with a large amount of pus. Subsequent blood culture and catheter tip culture revealed *Methicillin-resistant Staphylococcus aureus*.

Transesophageal echocardiography (TOE) was performed and revealed aortic valve vegetations with acute severe aortic regurgitation, right atrial appendage mass, and patent foramen ovale. A multidisciplinary team meeting was urgently done, and the decisions were immediate initiation of antibiotics and urgent preparation for surgery. Intravenous vancomycin 30 mg/kg/d in three doses was initiated.

After 90 min, the patient developed a disturbed level of consciousness and resistant shock. She was mechanically ventilated, intravenous inotropes were increased to the maximum dose and extracorporeal membrane oxygenation system was inserted. The patient continued to rapidly deteriorate, and then died.

Discussion

Embolic myocardial infarction is an uncommon but increasingly recognized complication of IE. The incidence ranges from 1% to 10%^{1–3} but

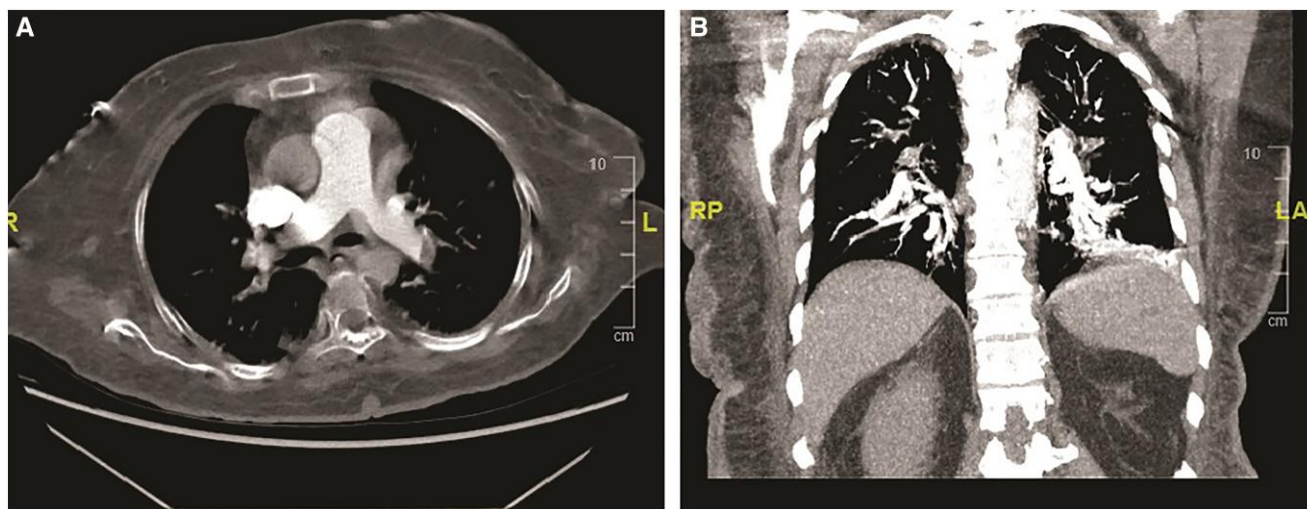


Figure 1 (A & B) Computed tomography pulmonary angiogram shows segmental and subsegmental pulmonary embolism.

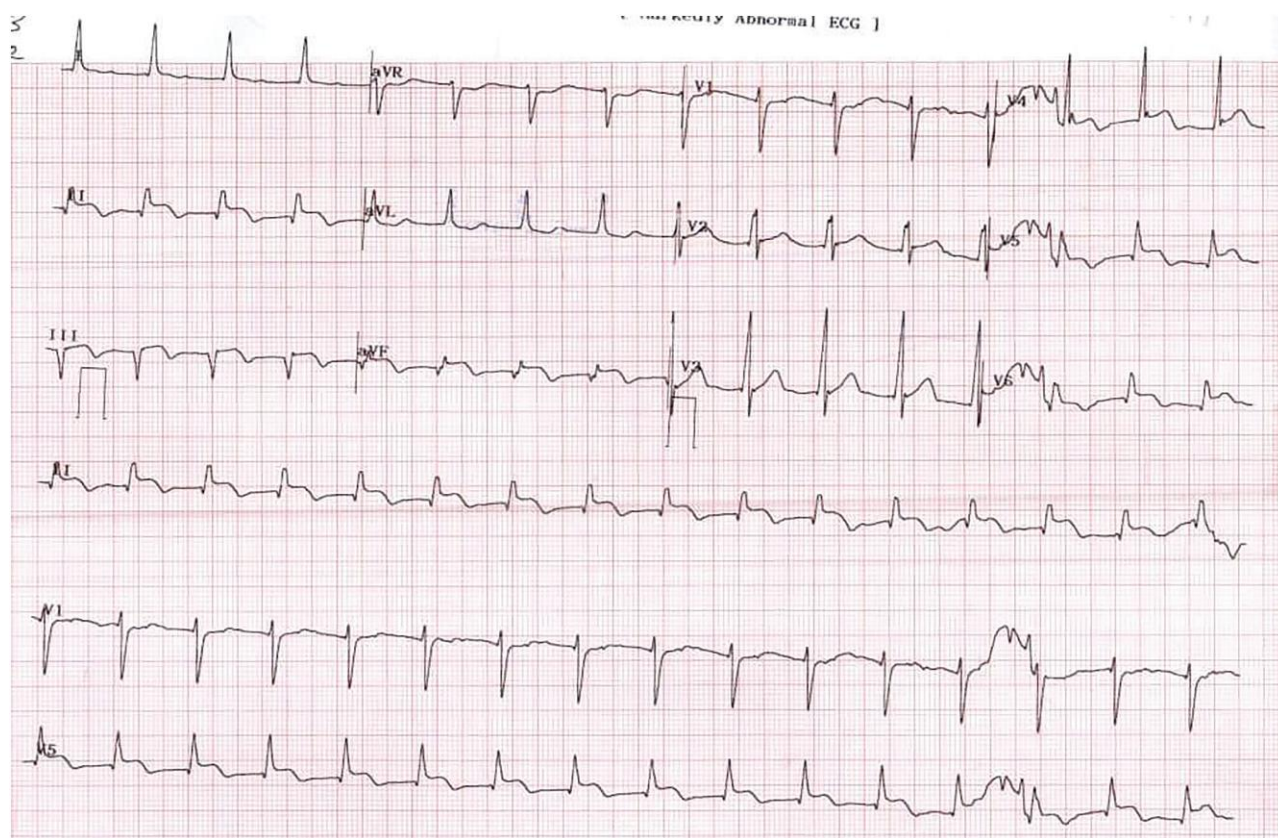


Figure 2 Electrocardiography shows ST-segment elevation in the inferolateral leads.

the mortality rate is high (64%). Acute coronary syndrome is usually an early and uncommon complication of IE.² The following factors increase the risk of embolism: the presence of a mobile vegetation, vegetation length >10 mm, infection with staphylococci or non-viridians streptococci, and a previous embolism.^{3,4}

In previous series, such as a Spanish series of 586 patients,¹ the most common cause of acute coronary syndrome during the course of IE has been extrinsic coronary compression due to periannular complications (e.g. pseudoaneurysm, abscesses), followed by septic coronary embolism. In more recent series,² however, the predominant mechanism is the embolization of vegetations in the coronary arteries.

According to the 2015 ESC guidelines, the indications for surgery for left-sided valve IE are: heart failure, uncontrolled infection, and prevention of embolism. Early consultation with a cardiac surgeon is recommended to determine the best therapeutic approach.⁴

The trend appears to be towards the use of primary percutaneous coronary intervention. However, the efficacy of and evidence for this approach are limited. Balloon inflation can result in displacement of the vegetation and a greater risk of further embolic phenomena.⁵

Stent implantation may be helpful, but the risk of coronary artery mycotic aneurysm or stent infection should be considered. Routine thrombus aspiration is not recommended during primary percutaneous coronary intervention according to the 2017 ESC Clinical Practice Guidelines,⁶ however, in patients with IE complicated by embolic myocardial infarction, manual thrombus aspiration could be beneficial because of the removal of infected material, although no data to support this approach are available.⁶

The characteristics of septic pulmonary embolism (SPE) at presentation are nonspecific and are usually unrecognized by clinicians. The initial clinical features vary from a low-grade fever and respiratory symptoms, including cough, haemoptysis, chest pain, and dyspnoea. Most patients with SPE have a presentation similar to that for pneumonia. The diagnosis of SPE is therefore, frequently delayed, which consequently influences prognosis. With the increasing use of indwelling catheters and devices, cardiac SPE is now generally associated with cardiac device implants, intravascular catheters, and an immunocompromised state.^{7,8}

According to the 2019 ESC guidelines, initial risk stratification of suspected or confirmed PE based on the presence of haemodynamic instability, is recommended to identify patients at high risk of early mortality. In patients without haemodynamic instability, further stratification of patients with acute PE into intermediate and low risk. Also, the Assessment of the RV by imaging methods or laboratory biomarkers, the PESI, or sPESI scores should be considered. It is recommended that anticoagulation with UFH be initiated without delay in patients with high-risk PE. Thrombolytic therapy leads to faster improvements in pulmonary obstruction compared with UFH alone.⁹

Both TTE and TOE are the fundamental methods used to diagnose, manage, and follow cases of IE. Importantly, TTE must be performed rapidly as soon as IE is suspected.¹⁰ If the result of TTE is negative and the clinician's suspicion is high for a diagnosis of IE, then a TOE should be performed.¹⁰

Conclusions

Embolic myocardial infarction is an uncommon but increasingly recognized complication of IE and represents important cause of mortality. A high index of suspicion is required in patients with acute coronary syndrome and fever. Because each situation is unique, a multidisciplinary team discussion is essential to choose the best treatment option.

Cardiac SPE remains a diagnostic challenge for clinicians. In patients with venous catheters, signs of infection, and pulmonary embolism, SPE should be strongly suspected and managed with proper treatment options. For suspected cases, blood culture, TTE/TOE, and computed tomography pulmonary angiography are the cornerstones for early diagnosis and treatment.

Aggressive management, including early cardiac surgery to remove the source of infection, is very critical. We believe that if the case was suspected earlier, this could have led to an early surgical intervention which possibly could have led to a better patient outcome.

Lead author biography



Islam Ahmed, MD is a graduate of Al-Azhar School of Medicine where he also attended his post graduate studies in the field of cardiology. Dr. Islam trained in Al-Azhar University Hospitals, Department of Cardiology. During his residency, he was able to provide superior care and consultations that resulted in overall improvement in patients care. Afterwards, he completed his Cardiology Fellowship Program at Al-Azhar University Hospitals, Cairo, Egypt at 2017. His practice focuses on

Interventional Cardiology and Echocardiography. Currently, he serves as a cardiologist in Armed Forces Hospital, Southern Region, KSA. He received many appreciation letters during his career for his outstanding performance, fruitful cooperation with co-workers.

Supplementary material

Supplementary material is available at European Heart Journal—Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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