CASE REPORT

Are we missing anaerobic infective endocarditis in some acute coronary syndromes?

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SUMMARY

A 76-year-old man presented with a 3-week history of intermittent fevers and dyspnoea on exertion after a dental bridge placement 2 months ago. The patient's medical history was significant for mild to moderate mitral valve prolapse. Initial evaluation was notable for a 3/6 systolic apical murmur. Laboratory investigations revealed leucocytosis and elevated erythrocyte sedimentation rate, C reactive protein and cardiac biomarkers. Patient was treated initially for non-ST elevation myocardial infarction. A 2-dimensional echocardiography was concerning for a new mitral regurgitation and a questionable vegetation adjacent to the mitral valve annulus. Transoesophageal echocardiography study confirmed the diagnosis. Subsequent microbial identification was notable for *Peptostreptococci* and he was started on intravenous penicillin therapy. The unexplained illness with underlying valve disease prompted consideration of infective endocarditis. This case describes a rare occurrence of anaerobic endocarditis imitating an acute coronary event.

BACKGROUND

The initial evaluation of infective endocarditis (IE) is challenging owing to the subtle presentations and wide range of involved microbial agents. A high index of suspicion is essential for the diagnosis. Head and neck manipulation, principally dental, can be associated with a clinically significant bacteraemia with potential progression to cardiac involvement. Anaerobic IE is a rare clinical scenario. *Peptostreptococci* have been described as a causative pathogen in a few reported cases. It mimics acute coronary syndrome but these two different conditions need to be treated differently. Antithrombotic therapy increases the risk of embolism and intracerebral haemorrhage in patients with IE. Anticoagulant and antiplatelet therapy do not reduce the risk of embolism in IE. Increase in cardiac biomarkers may have other explanations.

CASE PRESENTATION

A 76-year-old obese man presented with a 3-week history of intermittent fevers, chills and shortness of breath on exertion. The patient's medical history was significant for gout, hypertension and moderate mitral valve prolapse (MVP), which was diagnosed with transthoracic echocardiography (TTE) 9 years ago.

He has never had a heart attack in the past and has never had a heart catheterisation. Only significant surgical history was a dental procedure,

approximately 8 weeks ago with a permanent bridge placement. He had no history of tobacco or illicit drug misuse.

His vitals were temperature of 101°F, regular pulse of 107 bpm, blood pressure of 147/50 mm Hg, respiratory rate of 19 breaths/min and oxygen saturation of 99% in room air.

On physical examination, oral cavity was notable for poor dentition with no abscesses or gingival tenderness. The lungs were clear. Cardiac auscultation showed normal heart sounds and 3/6 systolic murmur, which was best heard over the apex and radiating to the axilla. He had trace bilateral pitting oedema. There were no Osler's nodes, Janeway's lesions or splinter haemorrhages. Other physical examination was unremarkable.

INVESTIGATIONS

Laboratory investigation revealed leucocytosis with a white cell count of 13.7 k/μL, neutrophilia 79.1%, haemoglobin 14 g/dL, mean corpuscular volume 90 fL, normal platelet count and no bandemia. No abnormality was noted on peripheral smear. Erythrocyte sedimentation rate (ESR) was 58 mm/h and C reactive protein (CRP) was 19.8. Cardiac pro-BNP was elevated at 953 pg/mL with elevated cardiac biomarkers with serial troponins of 0.487, 0.563, 0.55 ng/mL (normal 0–0.034 ng/mL) and elevated alanine aminotransferase 180 IU/L (normal 11–66 IU/L) with normal aspartate aminotransferase, alkaline phosphatase, albumin and bilirubin. No previous laboratory studies were available for comparison. Hepatitis serology was also negative. Urine analysis was within normal limits. Chest X-ray was suggestive of mild pulmonary congestion. ECG was consistent with a presumptively new right bundle branch block. CT of the chest was negative for pulmonary embolism.

TREATMENT

The patient was treated initially for non-ST elevation myocardial infarction and was started on heparin drip, aspirin, clopidogrel, statin with intravenous diuretics for worsening dyspnoea.

OUTCOME AND FOLLOW-UP**Inpatient course**

The patient demonstrated no significant clinical improvement. A TTE showed mild concentric left ventricular hypertrophy, borderline global hypokinesia, moderate left atrium dilation, normal right atrium, dilated inferior vena cava, moderate calcification of the mitral valve (MV) with prolapse of the posterior leaflet and moderate mitral

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regurgitation (MR). A new 1 cm vegetation was noted at the membranous septum in the outflow tract just beneath the attachment of the non-coronary cusp of the aortic valve at the left ventricular side adjacent to the anterior mitral valve annulus. A new mild aortic regurgitation was also noted.

The patient was started on vancomycin and ceftriaxone for empiric treatment for IE. Two of the two blood cultures were positive for Gram-positive cocci. Transoesophageal echocardiogram showed multiple vegetations on both sides of MV leaflets and vegetation that extends into the left ventricular outflow tract. Subsequent microbial identification was notable for *Peptostreptococci* and the patient was started on intravenous penicillin therapy according to the susceptibility report.

Outpatient follow-up

The patient completed the course of antibiotic regimen with resolution of blood cultures, inflammatory markers and vegetation on TTE.

DISCUSSION

Infections due to anaerobic bacteria are common and can be life-threatening.¹ Normal skin and mucous membrane flora are the main port of entry. IE due to anaerobic bacteria is an uncommon and is frequently overlooked, accounting for 2–16% of all cases of IE.^{2,3} Organism virulence and predisposing procedures play a critical role for bacteraemia and subsequent endocarditis evolution.

Most cases of anaerobic IE are caused by anaerobic and microaerophilic streptococci, *Propionibacterium acnes* and *Bacteroides fragilis*.^{4,5} IE caused by *Peptostreptococci* is rare.² Dental procedures, periodontal infections, genitourinary infections, cardiac prosthetic devices and septic arthritis are the most acknowledged risk factors.^{2,5} Anaerobic bacteraemia is seen in more than half of the patients who undergo dental procedures. Less than 80% of patients with IE have an underlying structural heart disease. Currently rheumatic heart disease accounts to less than 10% of IE in developed nations while more prevalent degenerative heart disease, such as MVP causing MR, is found in 30% of IE cases. Mitral prolapse without MR is not a common predisposition for IE.³ Anaerobic IE has a lesser prevalence in a pre-existing valve lesion in comparison to non-anaerobic pathogens.^{1,2}

Non-specific symptoms as unexplained fatigue, fever, myalgia and sudden onset heart failure could aid in early anticipation of the unexpected presentation. Late diagnosis carries a higher rate of complications including valvular destruction, septic emboli, septic shock and death.

There are no differences in the clinical diagnostic features between endocarditis due to anaerobic bacteria and that due to aerobic or facultative bacteria.⁵ Anaerobic Gram-negative bacilli have increased their resistance to penicillin and other antimicrobial agents in the past two decades, so identification of the pathogens and determining their antimicrobial susceptibility and β -lactamase production are essential for the adequate selection of effective antibiotic therapy.^{1,2,4,5}

Diagnostic clues for IE include elevated ESR, CRP, serum rheumatoid factor, cardiac biomarkers, haematuria, anaemia and low serum complement. Appropriate blood cultures with susceptibility testing are essential in the era of increasing microbial resistance. Echocardiography is an important diagnostic technique for imaging. Sensitivity and diagnostic accuracy has improved with the use of Doppler and transoesophageal imaging studies.

Peptostreptococcal endocarditis is largely penicillin susceptible. Penicillin G or vancomycin was been commonly prescribed. Second-line options include metronidazole or clindamycin if proven susceptibility. Surgery is indicated in persistent bacteraemia, mobile vegetation, two or more embolic sequelae, worsening heart failure, or valvular insufficiency or leaflet rupture. Annular abscess of the valve, myocardial abscess and secondary conduction disturbances caused by septal abscess are other accepted surgical indications.

Severe infections can lead to a variable degree of dysrhythmias, valve destruction, abscess formation, mycotic aneurysms, aortitis and septic or cardiogenic shock. The mortality rate for patients with anaerobic IE is 21–43%. Peptostreptococcal IE however has a better prognosis.

In our patient, the unexplained illness with underlying cardiac valvular disease prompted consideration of IE. A combination of fever, inflammatory markers, and echocardiography findings were diagnostic for IE. Although IE can be associated with intracoronary septic embolisation from the valvular vegetation resulting in acute myocardial infarction (MI), absence of significant ST-T wave changes, significant rise in cardiac biomarkers and segmental wall abnormality argue against but cannot certainly exclude the occurrence of MI. Coronary angiography was offered but was declined by the patient. This case describes a rare occurrence of peptostreptococcal endocarditis imitating an acute coronary event. A high index of suspicion is essential for prompt diagnosis. Antithrombotic therapy increases the risk of embolism and intracerebral haemorrhage in patients with IE. Anticoagulant and antiplatelet therapy do not reduce the risk of embolism in IE.

Patient's perspective

I have no medical knowledge or background so I only write from my own perspective and experience. Initially, I was told that I had a small heart attack. When I had the echocardiography a concerning infection in my prolapsed valve was explained to me. I was really grateful for the timely diagnosis and therapy. I could say that my recent dental procedure had saved my life otherwise I would not have come to the hospital! I am sure I will continue to see my dentist again.

Learning points

- ▶ Infective endocarditis (IE) due to anaerobic bacteria is rare.
- ▶ Overlooked anaerobic IE is associated with significant morbidity and high rates of mortality.
- ▶ Suspected IE needs to be ruled out in acute coronary syndrome since antithrombotic therapy increases the risk of embolism and intracranial bleeding.
- ▶ The mainstay of treatment for IE involving anaerobes includes the use of effective antibiotic therapy against these organisms.
- ▶ Increased cardiac biomarkers could have other explanations rather than acute coronary syndrome.

Contributors All authors provided patient care and involved in writing the manuscript.

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