CASE REPORT

Syncope secondary to left ventricular outflow tract obstruction, an interesting presentation of infective endocarditis

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SUMMARY

We describe a case of a 74-year-old woman who presented with symptoms of fever and lethargy, associated with an episode of cardiac syncope and exertional shortness of breath (SOB). She was diagnosed with *Staphylococcus aureus* infective mural endocarditis (IE) and subsequent transoesophageal echocardiogram (TOE) confirmed this diagnosis. As the vegetative mass arose from the septal wall, an unusual location, it caused left ventricular outflow tract (LVOT) obstruction and therefore behaved similarly to a subaortic valvular stenosis. There were no conduction abnormalities on the ECG and no clinical or echocardiographic features of congestive heart failure. The finding of LVOT obstruction explained the unusual presentation with syncope and exertional SOB making this case unique. Owing to the large vegetative mass and thereby its high risk of septic emboli, the patient underwent successful surgical resection of the mass with resolution of the obstruction. She successfully completed intravenous antibiotics and was discharged from hospital.

BACKGROUND

Infective mural endocarditis (IE) is an infection of the endocardial surface of the heart most commonly affecting valvular structures as well as the mural endocardium, that is, the lining of the heart chambers. With the wider use of intravascular devices and intravascular lines, known risk factors for *Staphylococcus aureus* bacteraemia, *S. aureus* has become the primary pathogen in IE.¹ Despite more prompt diagnoses and effective treatment, the mortality associated with IE still remains significantly high,² and is suggested to be increasing.³ The Duke diagnostic criteria are used to make a definitive diagnosis of IE, combining clinical, microbiological, pathological and echocardiographic parameters.⁴

Transoesophageal echocardiogram (TOE) remains the gold standard imaging tool for the diagnosis of IE, with high sensitivity and specificity.⁵ Furthermore, in *S. aureus* bacteraemia, TOE has demonstrated IE even in the absence of clinical or transthoracic echocardiography (TTE) findings.⁴ As visible vegetations carry a poorer prognosis in IE,⁶ it is important to utilise TOE for reliable identification and measurement.

We describe a case of an elderly patient presenting with an episode of suspected cardiac syncope in the context of a 1-week history of fever and lethargy.

CASE PRESENTATION

A 74-year-old woman with hypertension presented with a 1-week history of intermittent fevers and lethargy. Her general practitioner initially suspected a viral illness, and this diagnosis persisted despite multiple presentations and ongoing fever. The patient began to develop shortness of breath (SOB) with reduced exercise tolerance and then presented to accident and emergency (A&E) department with a witnessed transient loss of consciousness. She was sitting in a chair and lost consciousness without pre-syncope or neurological symptoms. She then quickly (<1 min) regained consciousness without any post-ictal behaviour. The history was suggestive of cardiac syncope.

On examination, the patient was febrile at 38.7°C, but haemodynamically stable with a heart rate of 98 bpm and blood pressure of 122/86 mm Hg. Her neurological examination was unremarkable, with a Glasgow Coma Scale score of 15/15 throughout. She had widespread splinter haemorrhages and painful red/purplish nodules on the pads of her fingers, suggestive of Osler nodes. She had an ejection systolic murmur heard throughout the precordium but loudest in the aortic area, with no radiation. The remaining examination was unremarkable.

The 12-lead ECG demonstrated sinus rhythm. Chest radiograph was clear and urine dipstick was positive for microscopic haematuria. Inflammatory markers were raised but the remaining blood tests were normal. In view of the history and findings on examination, IE was the working diagnosis. An urgent bedside TTE was performed, demonstrating a large mobile mass attached to the septal wall and obstructing the left ventricular outflow tract

Figure 1 A vegetative mass (1×1.44 mm) obstructing the left ventricular outflow tract (LA, left atrium; LV, left ventricle; AV, aortic valve).

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Video 1 Transoesophageal echocardiogram video (mid-oesophageal long-axis view) demonstrating a large mobile mass obstructing the left ventricular outflow tract.

(LVOT), causing peak dynamic gradients of 110 mm Hg. The mass was highly suggestive of a vegetative lesion. The rest of the TTE showed no obvious involvement of the well functioning aortic or mitral valve and preserved biventricular systolic function. As the mass was obstructing the LVOT it behaved very similarly to a subaortic membrane causing subaortic valvular stenosis. Thus the episode of syncope and SOB was most logically a result of the LVOT obstruction secondary to IE. She was started on empirical antibiotics for IE and treated with amoxicillin and gentamicin as per the Trust antibiotic guidelines. Of note, the patient had no known risk factors for IE and besides a diagnosis of hypertension there was no history of hospital admissions or invasive procedures. The patient underwent a TOE to further evaluate the size and valvular involvement of the vegetation. Multiple sets of blood cultures had at this stage grown a fully sensitive *S. aureus* and antibiotics were changed to flucloxacillin as per antibiotic sensitivities, microbiology advice and international guidelines.⁷

INVESTIGATIONS

TOE remains the gold standard for the diagnosis of IE.⁵ In our patient, the TOE elucidated the large mobile vegetation (14×10 mm) attached to the septum in the LVOT (figure 1), in proximity to the anterior mitral valve leaflet (AMVL) and aortic

valve (video 1). Despite the large vegetation, there was, interestingly, no involvement of the aortic valve and no evidence of aortic root abscess. However, there was moderate mitral regurgitation and, even though there was no obvious vegetation or perforation, due to the proximity of the vegetation to the AMVL, there was a high suspicion that it was affected (video 2). The TOE therefore confirmed the diagnosis of mural endocarditis with possible involvement of the AMVL.

OUTCOME AND FOLLOW-UP

A large vegetation (length >10 mm) and *S. aureus* IE are both independent predictors for embolic events,⁸ and, with both, our patient was at high risk. Furthermore, it has been demonstrated that early surgery in those with large vegetative IE is associated with a reduction in all-cause mortality.⁹ In view of this evidence, our patient was referred for surgery and successfully underwent resection of the vegetation, leading to resolution of the LVOT obstruction. Owing to the abnormality of the AMVL, as suspected on the TOE and further confirmed during surgery, she also had a mitral valve replacement. The histopathology and culture results of the resected mass confirmed that it was a vegetative mass secondary to *S. aureus*, the same organism that grew in the blood cultures. This thereby confirmed that the mass obstructing the LVOT was truly vegetation and further confirmed the diagnosis of IE.

The patient completed a total of 6 weeks of intravenous antibiotics, as recommended by existing international guidelines,⁷ and was discharged from hospital. At 6 months of follow-up, she was doing well.

DISCUSSION

IE secondary to *S. aureus* is well recognised to be associated with increased mortality¹⁰ and has been reported as the most important prognostic factor,¹¹ as it is known to cause aggressive and rapid progression of IE. Our patient's short history of symptoms and large vegetative mass was indicative of *S. aureus* IE.

As per existing international guidelines, surgical intervention in IE should be considered in the context of heart failure secondary to valvular destruction or fistula formation, uncontrolled infection either locally or systemically, infection due to fungal or multiresistant organisms and high risk of embolism due to large vegetation(s).⁷ In view of the large vegetative mass and high risk

Video 2 Transoesophageal echocardiogram video (mid-oesophageal 4-chamber view) demonstrating moderate mitral regurgitation.

of embolic events, our patient promptly underwent surgical intervention.

Recognised complications of IE include congestive heart failure, periannular abscess and systemic embolisation.¹² However, there have been no reported cases of IE causing LVOT obstruction, making this case unique. LVOT obstruction can result in chest pain, SOB and pre-syncope/syncope symptoms. It is predominantly seen in the context of hypertrophic cardiomyopathy as a result of septal bulge and systolic anterior motion of the mitral valve.¹³ It is also uncommonly secondary to a subaortic membrane.¹⁴ In our patient, the vegetative mass was acting in a similar fashion to a subaortic membrane and thereby causing subaortic valvular stenosis. Its large size caused extensive obstruction of the LVOT, explaining the impressive high-pressure gradients on TTE.

Syncope secondary to haemodynamic compromise due to underlying infection is possible, however, in the context of this case, and the nature of the syncopal episode, the lack of haemodynamic and neurological compromise on admission, and the significant LVOT obstruction, make it highly suggestive to be as a result of the vegetative mass and thereby IE.

Learning points

- ▶ Independent prognostic markers in infective mural endocarditis (IE) are vegetation size and *Staphylococcus aureus* bacteraemia.
- ▶ IE is an important differential in unexplained fever, even in the absence of well-described risk factors.
- ▶ Thorough examination may provide pathognomic signs and allow prompt further investigation and treatment.
- ▶ The location of the vegetation can influence the patient's clinical presentation and, as a result, cause non-classical symptoms such as syncope.
- ▶ Bedside transthoracic echocardiograms utility deliver prompt, accurate diagnosis and can be an early aid in prognostication.
- ▶ Early surgical intervention is case-dependent, guided by transoesophageal echocardiogram, and often indicated to avoid associated IE complications such as heart failure and embolisation.

Contributors All the authors were actively involved in the management of the patient. SH wrote the first draft and the remaining drafts. MC, FF and AD provided constructive feedback and contributed in writing the subsequent drafts of the case report.

Competing interests None declared.

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