

Aortic valve endocarditis with root abscess causing superior vena cava obstruction: a case report

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Background

To our knowledge, we report the first case of endocarditis with root abscess causing compressive superior vena cava (SVC) obstruction.

Case summary

An 84-year-old gentleman with previous tissue aortic valve replacement presented with fevers and systemic upset. Blood cultures grew *Streptococcus anginosus* and transoesophageal echocardiogram identified prosthetic valve vegetations with an associated root abscess. Antibiotics were commenced and referral made for surgical consideration. Several days into treatment the patient developed clinical signs of SVC obstruction and computed tomography demonstrated an enlarging root abscess with SVC compression. The patient was discussed with local cardiothoracic centres, but surgery was not an option primarily due to abscess size and vascular involvement. Priority moved from active to palliative treatment given no improvement with antibiotics, unsuitability for surgery, and patient discomfort. Within several weeks, symptoms/signs of SVC obstruction actually improved, likely due to collateral venous circulation formation and the patient was discharged home with palliative care input.

Discussion

There are previous reports of SVC obstruction related to infected SVC thrombus, indwelling intravascular devices, and para-aortic abscess, but none related to infective endocarditis. *Streptococcus anginosus* endocarditis is rare but often associated with abscess formation, and male gender, increasing age, and previous surgery are recognized risk factors.

Keywords

Case report • SVC obstruction • Aortic root abscess • Endocarditis • *Streptococcus anginosus*

Learning points

- Superior vena cava obstruction is a rare complication of endocarditis related root abscess formation.
- Whilst echocardiography remains the initial imaging modality of choice for endocarditis related aortic root abscess formation, multimodality imaging in the form of computed tomography compliments echocardiography in defining abscess position, size, and associated vascular involvement.

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Introduction

Superior vena cava (SVC) obstruction is characterized by signs and symptoms which result from impedance to SVC venous blood return from the head and upper limbs. Typically, symptoms include head, neck, and upper limb swelling, as well as breathlessness. In severe cases, respiratory and neurological compromise can develop. Malignancy remains the most common cause of SVC obstruction.^{1–3}

Timeline

Time	Progress
12 years ago	Tissue aortic valve replacement.
Day 1	Presented with systemic upset and raised inflammatory markers. Developed septic shock and required inotropic support in the medical high dependency unit (MHDU).
Day 2	Stepped down from MHDU. Bedside transthoracic echocardiogram did not reveal obvious vegetation. Computed tomography scan showed abnormal aortic root with adjacent fluid collection.
Day 3	Blood cultures grew <i>Streptococcus anginosus</i> . Full sensitivities were available.
Day 5	Transoesophageal echocardiogram demonstrated vegetations associated with two cusps of the prosthetic aortic valve and an aortic root abscess. Referral made to cardiothoracic centre for consideration of surgery.
Day 13	Peripherally inserted central catheter line inserted for long-term antibiotic administration.
Day 25	Developed clinical signs of superior vena cava (SVC) obstruction. Computed tomography revealed enlarging root abscess with SVC compression.
Day 26	Discussed with two cardiothoracic centres. Surgery considered too high risk primarily due to size of abscess and vascular involvement.
Day 38	No improvement in inflammatory markers/clinical condition. Antibiotics were stopped and palliative approach to treatment began.
Day 48	Clinical signs of SVC obstruction resolved.
Day 54	No longer required symptom control.
Day 66	Discharged home with palliative care input and social support.

Case presentation

An 84-year-old gentleman, with a background of a tissue aortic valve replacement (AVR) 12 years previously, presented to hospital with a several week history of malaise, fevers, and weight loss. His other past medical history included permanent atrial fibrillation, chronic

kidney disease Stage 4, and prostatic carcinoma. On admission, the patient had a blood pressure of 95/50 mmHg, a heart rate of 67 b.p.m. and he was afebrile. He was noted to have an ejection-systolic murmur in his aortic area. He did not have a diastolic murmur or any peripheral stigmata of infective endocarditis. He did not report any focal symptoms suggestive of infection.

Admission blood investigations revealed a haemoglobin of 8.2 g/dL (13.5–18 normal range) with mean corpuscular volume of 88 fL (80–100 normal range), a white cell count of $19.8 \times 10^9/L$ (4.0–11.0 normal range) and C reactive protein of 51 mg/L (<6 normal range). His chest X-ray was unremarkable. Bedside urinalysis was normal. An electrocardiogram demonstrated atrial fibrillation.

Within 12 h of admission, the patient developed septic shock requiring inotropic support. Given the several week history of systemic upset, no localizing infective symptoms and background of previous AVR, infective endocarditis was top of the differential diagnosis for the source of sepsis and an urgent bedside transthoracic echocardiogram (TTE) was performed. Whilst no obvious vegetations were revealed on TTE (Figure 1A–F), clinical suspicion for infective endocarditis remained high, and a transoesophageal echocardiogram (TOE) was planned.

To rule out an occult abscess, a computed tomography (CT) scan of the chest, abdomen, and pelvis was performed. No intrabdominal abscess was seen; however, the CT revealed an abnormal aortic root and ascending aorta with an adjacent fluid collection (Figure 2). The collection was compressing, but not obstructing, the drainage of the SVC into the right atrium.

Blood cultures grew *Streptococcus anginosus*. Intravenous amoxicillin 1 g three times per day and full dose gentamicin once per day was initiated to cover infective endocarditis, as per European Society of Cardiology (ESC) guidelines and microbiology advice.⁴ On Day 3 of admission, full sensitivities were confirmed and antibiotics switched to IV benzylpenicillin and synergistic gentamicin.

A TOE (Figure 3A–D) on Day 5 of admission identified vegetations on the prosthetic aortic valve (Figure 3B) with an associated root abscess. There were no mitral or tricuspid vegetations.

The patient was referred to the local cardiothoracic surgical centre for consideration of surgical intervention. A peripherally inserted central catheter (PICC) line was inserted to facilitate antibiotic administration.

On the 25th day into antibiotic treatment, the patient developed new bilateral upper limb and facial swelling, dilated neck/chest veins, and shortness of breath associated with an oxygen requirement. When asked to raise his arms, he developed worsening breathlessness and facial congestion, consistent with Pemberton's sign. He was believed to have symptoms and signs in keeping with SVC obstruction. This was considered likely to be secondary to compression from enlargement of the known aortic root abscess. However, as the patient had a PICC line *in situ*, an associated thrombus in the SVC with resultant obstruction was also plausible. Given the patient's previous TTE imaging failed to show the abscess, and a TOE not being immediately available, an urgent CT scan was performed to differentiate between these possibilities. This confirmed an enlarging root abscess with SVC compressive obstruction (Figure 4A and B) and excluded a PICC line-associated thrombus (Figure 4C).

The patient's case was discussed with two cardiac surgical centres; however, both felt that surgery was too high risk. This was primarily due to the extent of the operation required, given the large size of

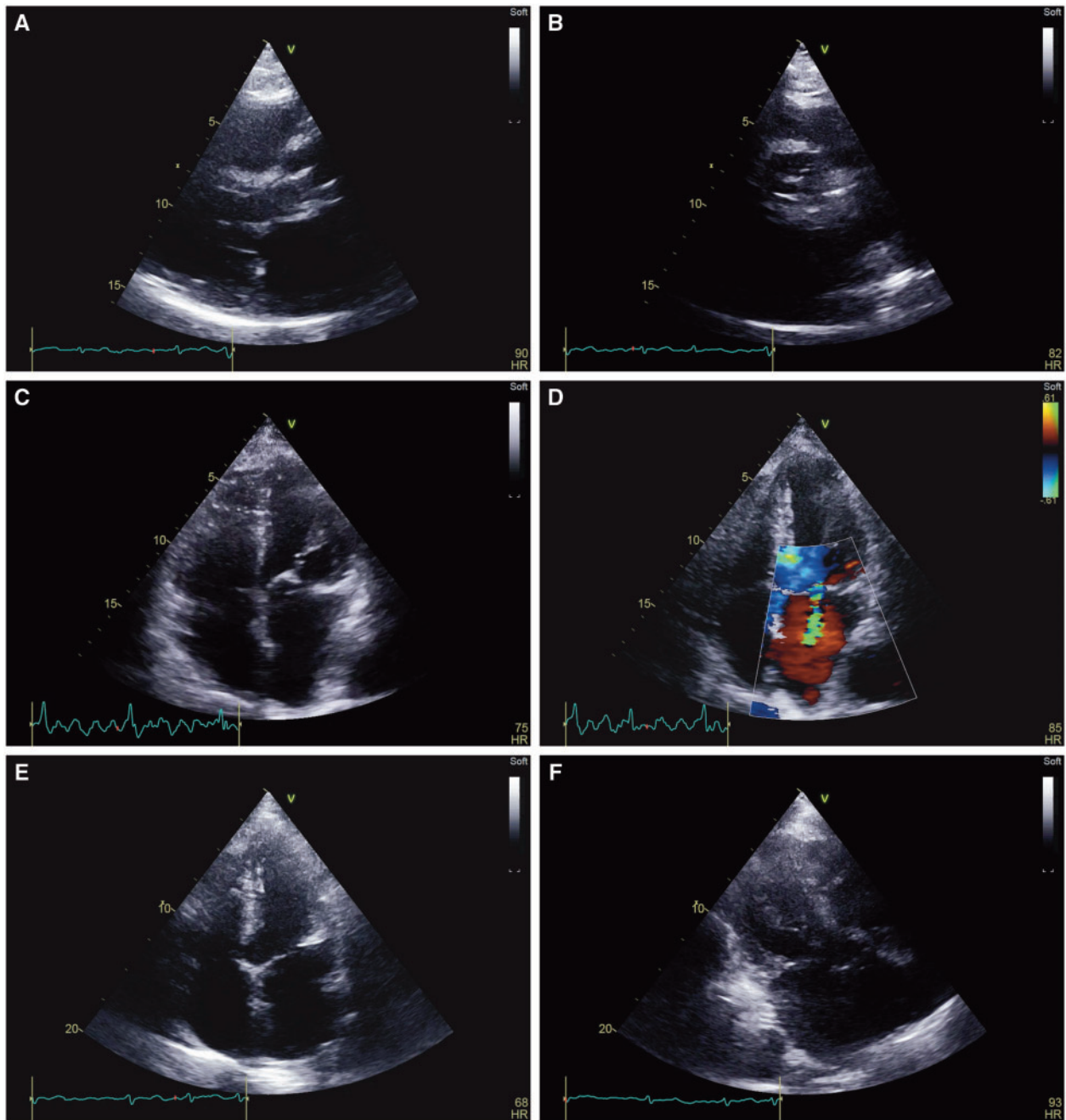


Figure 1 (A–F) Bedside transthoracic echocardiogram showed a non-dilated left ventricle (left ventricular internal diameter 3.9 cm), mild left ventricular hypertrophy (intraventricular septum diameter 1.2 cm), abnormal left ventricular septal motion in context of previous aortic valve replacement, and overall appearance of preserved left ventricular systolic function (suboptimal imaging to perform Simpson’s biplane assessment). The right ventricle was mildly dilated (basal right ventricular diameter 4.7 cm) with impaired longitudinal function (tricuspid annular plane systolic excursion 1.6 cm). The mitral valve had a thickened, calcified, and restricted posterior leaflet, with the appearance of a mild to moderate central jet of mitral valve regurgitation (visual assessment) and without obvious vegetation. The tissue aortic valve replacement was poorly visualized without obvious vegetation; however, there was an abnormal thickness of the aortic annulus and aortic root. Doppler assessment of the aortic valve replacement did not suggest significant regurgitation/stenosis. There was a normal-appearing tricuspid valve with severe tricuspid regurgitation (tricuspid regurgitation maximum peak gradient 49 mmHg). There was normal inferior vena cava inspiratory collapse. Both left and right atria were dilated. There was no pericardial or pleural effusion visible.



Figure 2 Admission computed tomography chest/abdomen/pelvis (sagittal view) showing abnormal aortic root with 4.8 cm fluid collection (A) compressing, but not obstructing, the superior vena cava (B). (C) Non-dilated internal jugular vein.

the abscess and associated vascular involvement. In addition, surgical teams thought it unlikely that the patient had the functional reserve to survive surgery and the immediate post-operative period, because of his age and comorbidities.

Conservative management continued, but despite optimal antibiotic therapy for 38 days, neither clinical or biochemical improvement in his condition was observed. On the grounds that surgery was not an option, the probability of successful treatment with antibiotics alone being remote and the patient's increasing discomfort, active treatment was discontinued in favour of a focus on symptom control and optimizing his quality of life. This decision was made after informed discussions with the patient and his family.

Over the next 3 weeks, his swelling and breathlessness unexpectedly resolved. He wished to go home and was deemed fit to do so with palliative care input and social support. At the time of writing, he remains alive 5 months after discharge.

Given there was nothing further he could be offered from a cardiology perspective he was not followed up in the community.

Discussion

We report a case of *S. anginosus* tissue AVR endocarditis, complicated by root abscess causing SVC compression with symptomatic

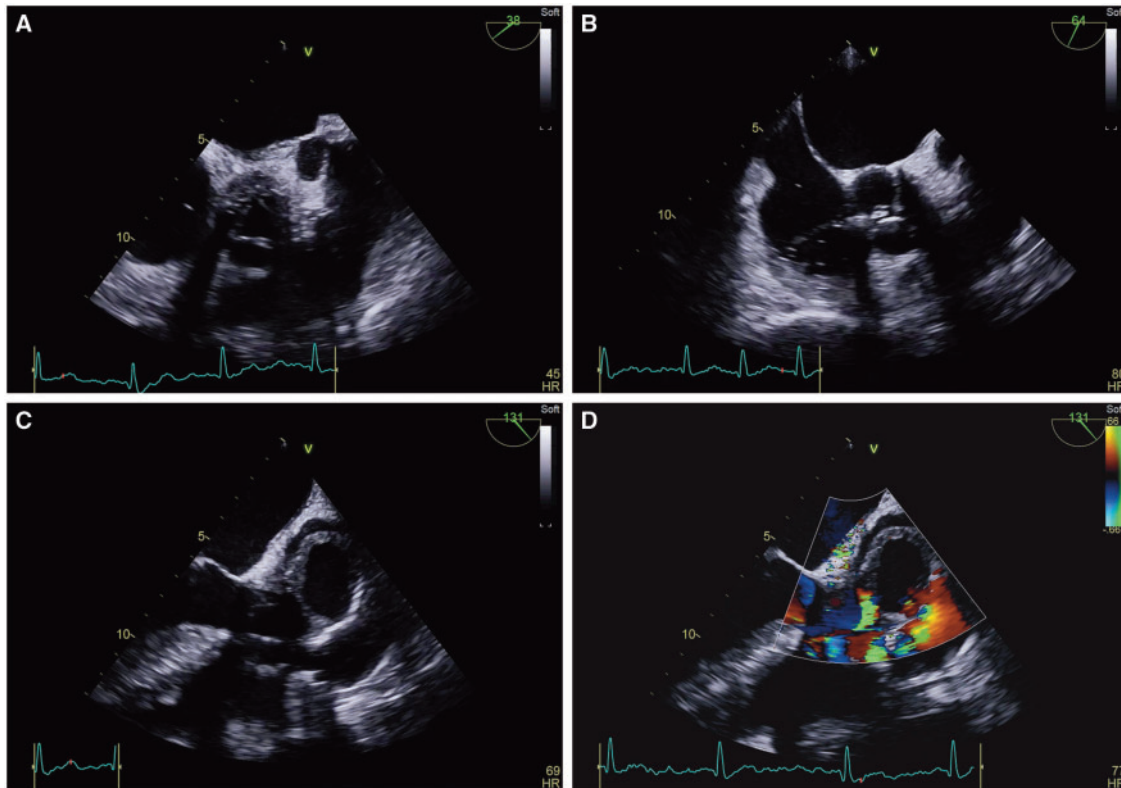


Figure 3 (A–D) Transoesophageal echocardiogram mid-oesophageal short-axis views (A/B) showing vegetations at 9 o'clock on the upper left and right-sided cusps which correspond in topography to the non-coronary and left coronary cusps of a native aortic valve. Transoesophageal echocardiogram mid-oesophageal long-axis views (C/D) showing large non-communicating echogenic collection in a dilated aortic root suspicious for an aortic root abscess.

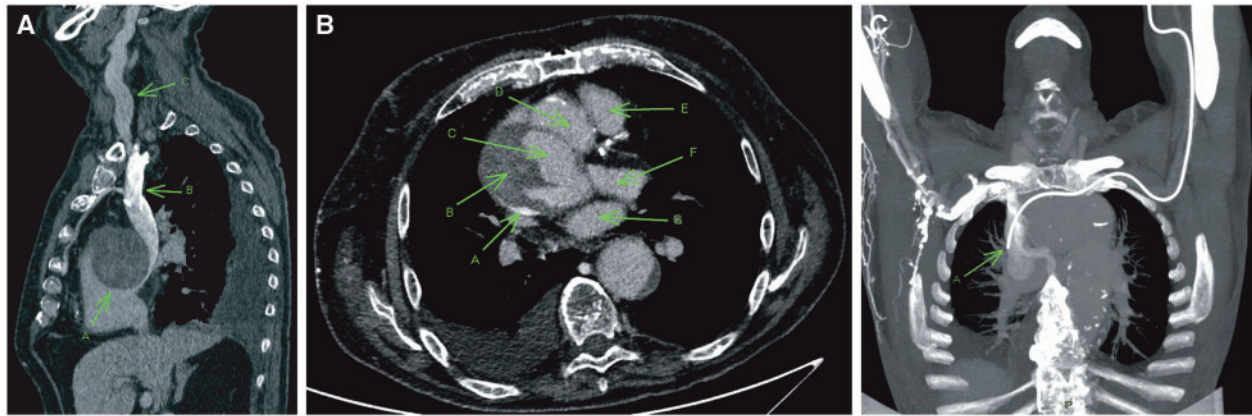


Figure 4 (A) Computed tomography chest/abdomen/pelvis (sagittal view) showing increase in size of the aortic root abscess to 6.2 cm (A) with compression of the superior vena cava and dilatation of the superior vena cava (B)/internal jugular vein (C) above the level of compression. (B) Computed tomography chest/abdomen/pelvis (transverse view) showing superior vena cava appearing as a slit-like structure (A) due to compression from the aortic root abscess (B) with false aneurysm (C). (D) Ascending aorta. (E) Pulmonary trunk. (F) Left atrial appendage. (G) Left ventricle. (C) Computed tomography chest/abdomen/pelvis (coronal view) showing tip of peripherally inserted central catheter line (A) in the superior vena cava without associated thrombus.

obstruction. To our knowledge, there is no previous published case of SVC obstruction due to compression from an endocarditis with root abscess. There are reports of para-aortic abscess/infected aortic aneurysm causing the syndrome in the absence of endocarditis.^{5,6} Likewise cases of infected thrombus within the SVC, without endocarditis, have been shown to cause SVC obstruction.^{7,8} Interestingly, the use of indwelling intravascular devices such as pacemakers, implantable cardioverter-defibrillators, and central catheters are increasingly recognized as causes of SVC obstruction as a result of vessel wall inflammation, fibrosis, and thrombus formation.^{2,3} In this case, we considered a PICC-associated thrombus but ruled this out on CT imaging.

Streptococcus anginosus is a member of the *Streptococcus milleri* group and part of the normal flora of human mucous membranes. Whilst an infrequent cause of endocarditis, the organism is commonly associated with abscess formation. Male gender, increasing age, and previous surgery are recognised risk factors, making our patient's case and complications typical of the organism.^{9–11} The 2015 European Cardiology Society (ESC) guidelines for the management of infective endocarditis specifically recognize *S. anginosus* as a high-risk organism (as well as for Group B, C, and G streptococci) for endocarditis associated abscess formation and highlight the importance of considering adjunctive surgery.⁴ In this case, surgery was deemed too high-risk primarily due to the abscess size and its vascular involvement.

The 2015 ESC guidelines also state that decisions regarding the management of endocarditis should be individualized. With regards to our patient, the risk-benefit ratio of surgical intervention was considered by two separate surgical centres and felt to be unacceptable. Likewise, given the very low probability of a successful resolution of the abscess with conservative treatment alone, continuing inpatient intravenous antibiotics was felt likely to have an overall detrimental impact on the patient's quality of life and comfort. These decisions

should be, and were made, in consultation with the patient and his family.⁴

We observed resolution of the clinical signs of SVC obstruction several weeks after antibiotics were withdrawn. We postulate that this is likely due to formation of collateral venous circulation via the azygous venous system.¹

Lead author biography



Malo Marcel Francois Scullion is a Clinical Fellow in Cardiology at University Hospital Hairmyres in East Kilbride, United Kingdom. He completed his Medical degree and a BSc in Medical Sciences at the University of Aberdeen.

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 author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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