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CASE REPORT
Valvular heart disease

AngioVac debulking of a tricuspid valve mass following complex lead extraction in a rare case of Austrian syndrome: a case report

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Background The European Society of Cardiology recommends surgical valvular intervention in right-sided infective endocarditis for persistent vegetations >20 mm after recurrent pulmonary emboli, infection with a difficult-to-eradicate organism with >7 days of persistent bacteraemia, or tricuspid regurgitation causing right-sided heart failure. In this case report, we discuss the role of percutaneous aspiration thrombectomy for a large tricuspid valve (TV) mass as an alternative to surgery due to poor surgical candidacy in a patient with Austrian syndrome, following a complex implantable cardioverter defibrillator (ICD) device extraction.

Case summary A 70-year-old female presented to the emergency department after being found acutely delirious at home by family. Infectious workup was notable for growth of *Streptococcus pneumoniae* in the blood, cerebrospinal, and pleural fluid. Transoesophageal echocardiogram was pursued in the setting of bacteraemia and revealed a mobile mass on the TV consistent with endocarditis. Given the size and embolic potential of the mass and eventual need for ICD replacement, the decision was made to pursue extraction of the valvular mass. The patient was a poor candidate for invasive surgery, so we opted to perform percutaneous aspiration thrombectomy. After the ICD device was extracted, the TV mass was successfully debulked using the AngioVac system without complication.

Discussion Percutaneous aspiration thrombectomy of right-sided valvular lesions has been introduced as a minimally invasive approach to avoid or delay valvular surgery. When intervention is indicated for TV endocarditis, AngioVac percutaneous thrombectomy may be a reasonable operative approach, particularly in patients who are at high risk for invasive surgery. We report a case of successful AngioVac debulking of a TV thrombus in a patient with Austrian syndrome.

Keywords Endocarditis • Thrombectomy • Case report

ESC Curriculum 2.2 Echocardiography • 4.11 Endocarditis • 5.10 Implantable cardioverter defibrillators • 6.2 Heart failure

Learning points

- Austrian syndrome is characterized by the presence of 'Osler's triad' of endocarditis, pneumonia, and meningitis caused by *Streptococcus pneumoniae*.
- Minimally invasive percutaneous debulking techniques may be an appropriate alternate to surgery for management of large tricuspid valve masses in cases of infective endocarditis, including cases of Austrian syndrome.

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Introduction

The European Society of Cardiology (ESC) 2015 guidelines outline three clinical scenarios in which surgical intervention should be considered for management of right-sided endocarditis. These scenarios include the presence of (i) persistent vegetations >20 mm after recurrent pulmonary emboli, (ii) >7 days of bacteraemia despite appropriate antimicrobial therapy, or (iii) tricuspid regurgitation causing right-sided heart failure refractory to medical management.[1] When pursued, surgical intervention for right-sided endocarditis most commonly involves valve replacement or repair.[1] While these strategies carry the intrinsic high risk of an open cardiac surgery, there is emerging evidence for the role of percutaneous aspiration thrombectomy for such cases when patient characteristics portend too high of risk to proceed with surgery.[2,3] In this case report, we discuss the role of percutaneous aspiration thrombectomy for a large tricuspid valve (TV) mass as an alternative to surgery for a patient with poor surgical candidacy.

Timeline

Timeline	Description
Day 0	Patient presented as an outside hospital transfer with *Streptococcus pneumoniae* bacteraemia, endocarditis, meningitis, TV vegetation, and concern for implantable cardioverter defibrillator (ICD) infection.
Day 1	Transoesophageal echocardiogram (TEE) revealed a 3.5 mm × 11.5 mm mobile mass with independent motion on TV, as well as a 9.5 mm × 14.7 mm mobile echodensity on the ICD pacing wire, concerning for endocarditis and cardiac implantable electronic device (CIED)-related infection.
Day 5	Patient underwent ICD device extraction with intraoperative intracardiac echocardiography (ICE).
Day 8	Patient underwent right heart catheterization and AngioVac-assisted extraction of the TV mass, with near-complete removal evidenced on post-debulking TEE images.
Day 11	Patient was discharged to acute rehab with regular follow-up with cardiology and infectious disease.
Day 36	Patient completed 4 week course of intravenous (IV) antibiotics from date of AngioVac procedure per infectious disease.
Day 99	ICD replacement was performed 3 months following date of debulking procedure.

Case presentation

A 70-year-old female presented to the emergency department after being found acutely delirious at home by family. The patient's past medical history included hypertension, heart failure with recovered ejection fraction (EF) secondary to non-ischaemic cardiomyopathy, documented history of paroxysmal atrial fibrillation, and remote cardiac arrest secondary to ventricular fibrillation. Additionally, the patient had a cardiac resynchronization therapy defibrillator (CRT-D) placed in 2010 following her cardiac arrest for both primary and secondary prevention, as she had reduced EF and prolonged native QRS at that time. The patient's CRT-D was later converted to a single-chamber ICD in 2016 at the time of battery exchange. This was presumptively due to a lack of arrhythmic events and recovery of a narrow non-pacing QRS in conjunction with recovered EF at the time of battery exchange. The patient's prior cardiologist documented 'non-pacing QRS is now narrow, and EF recovered' just prior to right atrial and left ventricle lead removal, per chart review. The patient remained without arrhythmic events on serial device interrogations for several years. This remained true until ~1 month prior to admission when she had one episode of ventricular fibrillation prompting defibrillation. She had not yet received a device upgrade by the time of this admission.

Vital signs on presentation were notable for fever to 38.4°C, with otherwise normal blood pressure and pulse, saturating >94% on room air. Physical exam was notable for altered mental status, jugular venous distention, and a soft systolic murmur at the left lower sternal border. Initial laboratory studies revealed white blood cell count of 10 880/μL (normal range 4.00–10.00 K/μL), haemoglobin 7900 g/dL (normal range 13.5–17.5 g/dL), and glomerular filtration rate of 78 mL/min/1.73 m^2. Workup of altered mental status included lumbar puncture, and cerebrospinal fluid cultures grew *S. pneumoniae*. A chest radiograph revealed a right-sided consolidation and pleural effusion. Both pleural fluid cultures and blood cultures grew *S. pneumoniae*. Initial transthoracic echocardiogram (TTE) revealed a 1.0 cm × 2.0 cm TV mass with moderate tricuspid regurgitation and estimated left ventricular EF of 45%. Subsequent transoesophageal echocardiography revealed a 3.5 mm × 11.5 mm mobile mass with independent motion on the posterior leaflet of the TV, as well as a 9.5 mm × 14.7 mm mobile echodensity on the atrial aspect of the ICD pacing wire, concerning for endocarditis and CIED-related infection. With evidence of Osler's triad of pneumonia, endocarditis, and meningitis in the setting of disseminated *S. pneumoniae*, she was diagnosed with the rarely seen Austrian syndrome.

The patient was initially treated with IV vancomycin 1250 mg every 12 h and ceftriaxone 2 g every 12 h and later transitioned to monotherapy with ceftriaxone at the same dose. She also received prophylactic anticoagulation with 5000 units of subcutaneous heparin every 8 h. Given evidence of CIED-related infection, our electrophysiologists performed ICD extraction in accordance with ESC guidelines.[1] Intracardiac echocardiography employed during lead extraction provided detailed visualization of the 11-year-old lead and its adherent mass, as well as the TV mass (see Supplementary material online, *Video S1*). Extraction of the lead was complex in this case as a result of insulation build-up encountered along its route. It is a standard practice of our electrophysiologists to employ ICE during complex extractions in which there is high risk of pericardial effusion. Extreme caution is used to avoid dislodgment of any intracardiac masses including potential septic emboli in cases of suspected endocarditis. The lead was ultimately removed intact via traction through the device pocket. There were no evidence of pericardial effusion on ICE post-extraction and no clinical evidence of embolic complication post-procedure.

After multidisciplinary discussion, our colleagues in infectious disease recommended removal of the TV mass primarily to reduce embolic potential, but also to reduce risk of future CIED-related infection upon re-implantation. Given the patient's multiple co-morbidities, she was deemed a poor surgical candidate; consequently, we opted to pursue minimally invasive debulking using AngioVac. Following ICD extraction, the patient underwent debulking of the TV mass using a 25F AngioVac with right internal jugular and right femoral vein access. Intraoperative transoesophageal echocardiography revealed a 1.6 cm × 0.8 cm mobile mass on the right atrial aspect of the TV leaflet (see Supplementary material online, *Video S2*, Supplementary material online, *Video S3*). This mass was successfully debulked with multiple runs under TEE and fluoroscopic guidance, with near-complete removal evidenced on post-debulking images (see Supplementary material online, *Video S4*).

Pathology of the extracted mass revealed thrombus with no growth on specimen cultures. Notably, the patient received over 1 week of broad-spectrum antibiotics (vancomycin plus ceftriaxone) prior to debulking. The patient was discharged with a wearable cardioverter defibrillator due to recent episode of ventricular fibrillation. She completed a 4 week course of IV ceftriaxone from the date of debulking. The patient achieved impressive functional recovery following treatment of her systemic infection. She subsequently underwent reimplantation of a single-chamber ICD 3 months after discharge.

Discussion

Austrian syndrome is characterized by the presence of 'Osler's triad' of endocarditis, pneumonia, and meningitis caused by *S. pneumoniae*. While *S. pneumoniae* remains the most common cause of bacterial pneumonia and meningitis worldwide, it accounts for <3% of cases of infective endocarditis (IE).[4–6] The most common presenting symptoms of pneumococcal endocarditis are fever (71% of cases) and heart murmur (55% of cases).[6] Among individuals presenting with pneumococcal endocarditis, Osler's triad is seen in about 15–29% of cases.[6,7] The aortic valve is affected in approximately 75% of cases, and vegetations tend to be large, imposing a high risk of systemic embolization.[6] Alcoholism is the most common risk factor, seen in nearly 40% of patients. Other risk factors mirror those of non-pneumococcal forms of IE, including immunodeficiency and intrinsic lung disease. Austrian syndrome bears a daunting mortality rate around 30%.[8]

Medical management of Austrian syndrome follows established guidelines for treatment of pneumococcal endocarditis and meningitis. Intravenous antibiotic therapy should be initiated with vancomycin and a third- or fourth-generation cephalosporin and should be narrowed based on case-specific susceptibility when available, keeping in mind the need for adequate cerebrospinal penetration. Duration of IV therapy is typically 4–6 weeks.[1] Surgical intervention may also provide a mortality benefit. Current ESC guidelines recommend surgical intervention be considered in right-sided IE when certain complications are present, including refractory right heart failure due to tricuspid regurgitation, sustained bacteraemia >7 days despite appropriate antimicrobial therapy, and vegetations >20 mm in diameter persisting after recurrent septic emboli. These recommendations hold for both TV and pulmonic valve lesions.[1] In a review of 197 cases of pneumococcal endocarditis, patients who received both medical and surgical management had a 32% mortality compared with 62% among patients who received only medical management.[6] The most common surgical modalities for TV endocarditis include valve replacement, valve repair, and valvectomy.[1] While these surgical strategies carry the high risks of an open cardiac procedure, percutaneous thrombectomy has been introduced as a minimally invasive approach to avoid or delay valvular surgery. For example, percutaneous aspiration of TV vegetations has been shown to be an effective temporizing bridge to surgical TV repair or replacement in IV drug users as they pursue rehabilitation and await surgical candidacy.[9] Furthermore, a recent expert statement on management of right-sided endocarditis in IV drug users supported involvement of interventional cardiology for consideration of percutaneous thrombectomy as part of a multidisciplinary approach to management of right-sided endocarditis among IV drug users.[10]

Approved by the Food and Drug Administration (FDA) since 2014 for removal of intravascular thrombi, the AngioVac device consists of two venous cannulas, one for venous drainage and another for venous return, connected by a bypass pump and filter. When used to debulk a TV thrombus, the venous drainage cannula is advanced into the right atrium allowing proximity to valvular lesions. When the circuit is turned on, blood and thrombotic material are suctioned into the extracorporeal system, thrombotic material is removed, and filtered blood is returned to venous circulation. A retrospective study comparing outcomes between TV surgery and percutaneous TV debulking (PTVD) using AngioVac found similar 1 year mortality between the two cohorts, despite the PTVD cohort being likely sicker overall, as a majority of this cohort was declined surgical intervention due to co-morbidities. Additionally, length of stay and blood transfusion requirements were lower among the PTVD group, likely due to the less invasive nature of the procedure. Overall, these findings support AngioVac PTVD as a reasonable operative approach in TV endocarditis.[3]

Lead author biography

Megan Middleton is currently a third-year resident physician in Internal Medicine at Rush University Medical Center and an incoming first-year fellow physician in Pulmonology and Critical Care at Cleveland Clinic. Within medicine and beyond, Megan is a dedicated advocate for health equity and has published research on issues such as discrimination in health care. In her personal life, Megan enjoys spending time outdoors and is an avid hiker.

Supplementary material

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

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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 the images and associated text has been obtained from the patient in line with COPE guidance using the *European Heart Journal—Case Reports* Consent Form.

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