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CASE REPORT
Valvular heart disease

Case report: septic embolism through patent foramen ovale in mitral valve endocarditis

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Background Infective endocarditis with septic emboli is a well-recognized sequala. However, emboli almost invariably are found 'downstream' to the vegetation. In the absence of congenital heart disease, the embolization from the left heart to the pulmonary circulation has never been described.

Case summary A 4-year-old boy presents with a background history of pharyngitis treated with antibiotics. Upon ambulatory review, he is noted to have a new murmur. Transthoracic echocardiogram demonstrated mitral valve vegetation with severe mitral regurgitation; in addition, there was a patent foramen ovale (PFO), and there were no congenital heart defects. The patient was treated for infective endocarditis on high clinical suspicion. He subsequently developed septic pulmonary emboli in the absence of right-sided vegetation. Subsequent mitral valve vegetectomy, resection of infected native anterior mitral valve leaflet, mitral valve repair, and valvuloplasty. The patient made an excellent recovery following the completion of antibiotic therapy.

Discussion Although not possible to confirm with certainty, this case demonstrates the most plausible explanation for this child's presentation being septic pulmonary emboli originating from left-sided heart vegetation migrating through a PFO.

Keywords Infective endocarditis • Pulmonary embolism • Patent foramen ovale • Mitral regurgitation

ESC curriculum 2.2 Echocardiography • 2.4 Cardiac computed tomography • 4.3 Mitral regurgitation • 4.11 Endocarditis

Learning points

- Septic emboli across a patent foramen ovale from mitral valve endocarditis to the pulmonary arteries are a possible cause of septic pulmonary emboli.
- In circulations with a raised left atrial pressure, septic emboli through an atrial communication may be a mechanism of embolization to the pulmonary circulation.

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Introduction

Septic emboli in infective endocarditis are a well-recognized phenomenon.[1,2] However, embolus from a left-sided source to a right-sided destination is incredibly rare in a structurally normal heart. We present a case of probable left-to-right embolism of septic emboli across a patent foramen ovale (PFO).

Summary figure

3 weeks prior to presentation	History of pharyngitis, treated in the community by general practitioner with erythromycin.
Presentation	Presented to district general hospital with high fever, coryza, inflamed tonsils, and elevated C-reactive protein (CRP) (119, ref < 1). Commenced on oral co-amoxiclav for 5 days. Symptoms resolved after 24 h.
Day 3	Upon review in ambulatory care, a new onset pan-systolic murmur at cardiac apex with no radiation noted was found. Echocardiogram demonstrated large vegetation on anterior mitral valve leaflet, multiple jets of severe mitral regurgitation, and a small PFO with a left-to-right shunt. Based on vegetation and fever, high suspicion of infective endocarditis and commenced on empirical intravenous antibiotics (flucloxacillin, amoxicillin, and gentamicin) before being transferred to our centre.
Day 4	Transferred to our cardiac institution. The patient's CRP (39, ref < 1) had slightly improved. All blood cultures remained negative. Normal dentition. Consultation with microbiology recommended antibiotic regimen change to intravenous vancomycin (15 mg/kg TDS), rifampicin (10 mg/kg BD), and gentamicin (1 mg/kg BD) for penetrance and cover.
Day 9	Discussed in multi-disciplinary meeting, decision to proceed to surgery given the high risk of embolization. Supplemental cross-sectional imaging to evaluate for any embolization.
Day 12	Clinical deterioration with increased work of breathing, tachypnoea, and recurrent temperature spikes.
Day 13	Patient underwent mitral valve vegetectomy, resection of infected native anterior mitral valve leaflet, mitral valve repair and plasty with replacement of the body of the anterior mitral valve leaflet with mitral homograft, and re-implantation of a chordae from the MV homograft on the head of the posterior papillary muscle with PFO closure. Uncomplicated post-operative period.
Day 24	Tissue culture from the resected valve was negative; 16 s rDNA sequencing PCR detected a sequence of *Streptococcus pneumoniae*, sensitive to penicillin.
Day 42	Antibiotics ceased, no further fevers or clinical deterioration, and patient discharged from ambulatory care.
12 months post-operative	Normal clinical examination with only mild residual mitral regurgitation (MR).

Case presentation

A 4-year-old boy presented to his local hospital with persistent high fever following 3 weeks of pharyngitis, initially treated with 7 days of erythromycin. Physical examination demonstrated inflamed tonsils, and blood tests demonstrated elevated C-reactive protein. Symptoms resolved following 24 h of co-amoxiclav; however, 3 days later at ambulatory review, a new pan-systolic murmur, located at the apex of the child's heart with no radiation, was noted. Transthoracic echocardiogram (TTE) showed a large vegetation (11 × 14 mm) on the anterior leaflet of the mitral valve with suspected perforation and severe mitral regurgitation (MR) (Supplementary material online, *Videos S1* and *S3*), with several jets directed both posterolaterally and anterolaterally thought to be from failure of coaptation and secondary to destruction of the anterior leaflet of the mitral valve. The heart was otherwise structurally normal with a small PFO with a left-to-right shunt. Based on modified Duke criteria for infective endocarditis, the patient did not fulfil the criteria for infective endocarditis; however, there was a strong clinical suspicion based on evidence of endocardial involvement and the presence of fever and the subsequent development of vascular phenomenon in septic pulmonary emboli. He was transferred to our institution after intravenous antibiotics (flucloxacillin, amoxicillin, and gentamicin) were empirically started. Upon admission and after microbiology consultation, antibiotics were converted to vancomycin (15 mg/kg TDS), rifampicin (10 mg/kg BD), and gentamicin (1 mg/kg BD), as blood cultures remained negative. Formal examination of his dentition was unremarkable. Multi-disciplinary meeting concluded that surgical management was required over conservative medical management given the high risk of embolization; however, further cross-sectional imaging would be required to evaluate any embolization.

On Day 12, the patient deteriorated clinically with increased work of breathing, tachypnoea, and recurrent temperature spikes. Chest X-ray demonstrated new focal lesions. Thoracic computed tomography (CT) revealed small peripheral foci of consolidation suggestive of septic pulmonary emboli and no other possible sources of left-to-right shunts (*Figure 1*). Subsequently, it was believed that the source of the septic emboli was from the vegetation on the mitral valve with left-to-right embolus across the PFO. The patient underwent urgent open-heart surgery on Day 13 of presentation and Day 9 of triple therapy antibiotics. Intraoperative transoesophageal echocardiogram (TOE) demonstrated the large vegetation of the mitral valve, the absence of right-sided vegetation, and the presence of a PFO (*Figure 2*, Supplementary material online, *Videos S1–S3*) On surgical inspection, there was destruction of the anterior mitral valve leaflet with gross vegetation and superimposition of thrombotic material on the vegetation. Mitral valve vegetectomy, resection of infected native anterior mitral valve leaflet, mitral valve repair and valvuloplasty replacing the body of the anterior mitral valve leaflet with mitral valve homograft, and re-implantation of the chordae to the head of the posterior mitral valve papillary muscle with PFO closure were performed. Although tissue culture from the resected valve was negative, 16 s rDNA sequencing PCR detected a sequence of *Streptococcus pneumoniae*, sensitive to penicillin.

The patient's post-operative course was uneventful, and antibiotics were completed 6 weeks after initial presentation. At 12 months follow-up, his clinical examination was normal with only mild residual MR on TTE.

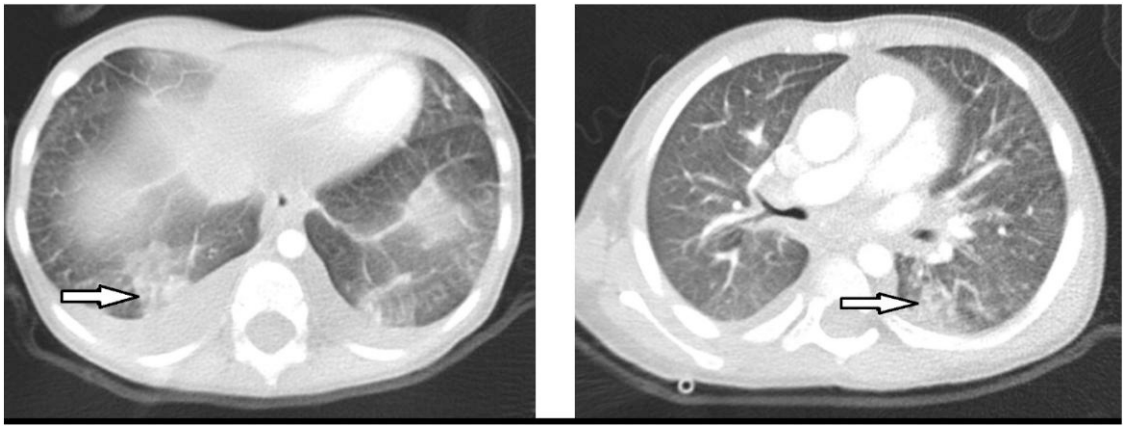

Figure 1 Computed tomography sections demonstrating focal areas of consolidation (arrow), consistent with septic pulmonary infarcts.

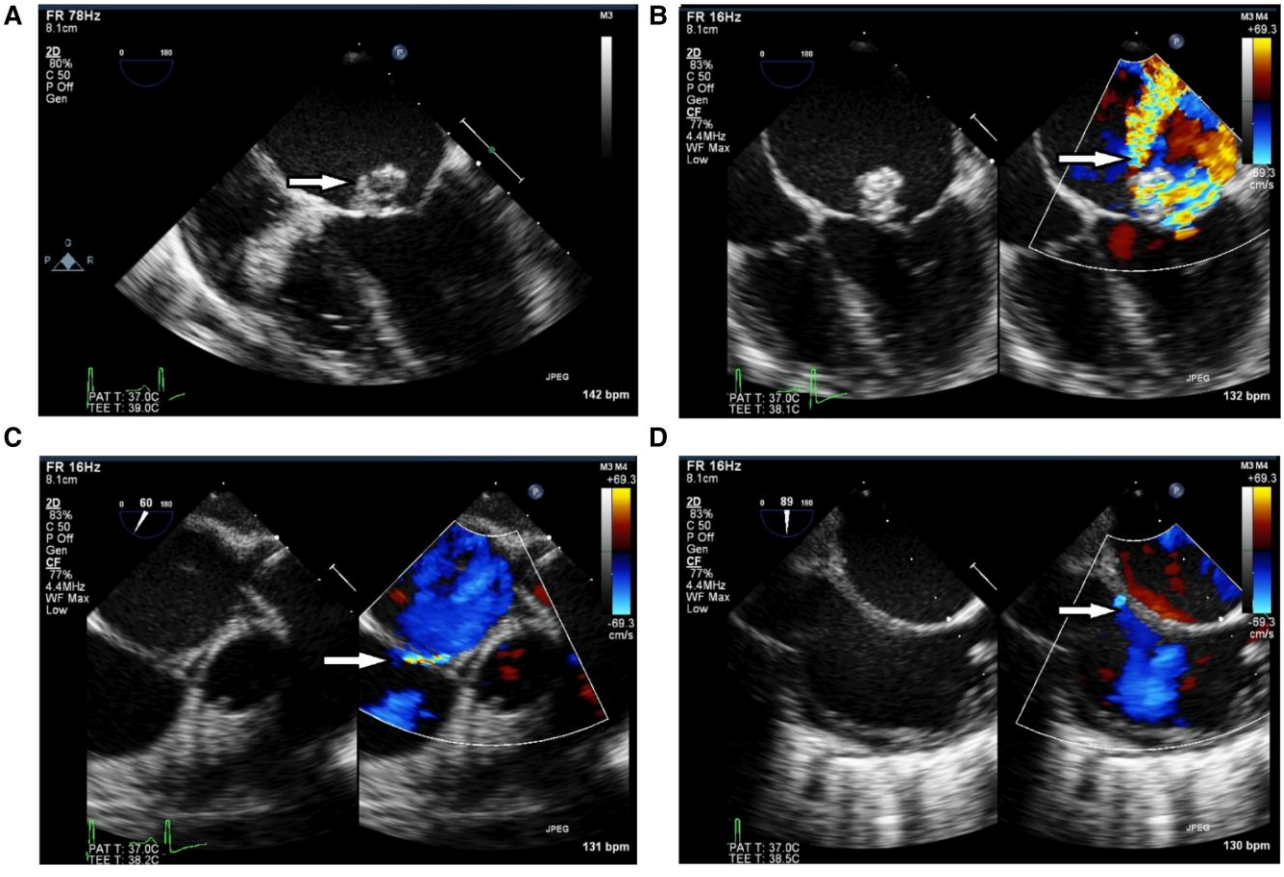

Figure 2 Intraoperative transoesophageal echocardiogram. (*A*, *B*) Large and very mobile vegetation on the anterior leaflet of the mitral valve with severe mitral regurgitation. (*C*) Short-axis view demonstrating left-to-right flow across the patent foramen ovale [left atrium (LA) end 1.6 mm, right atrium (RA) end 2.6 mm, and tunnel length 1.6 mm]. *(D)* Bicaval view demonstrating the PFO demonstrating left-to-right flow across the patent foramen ovale.

Discussion

Septic pulmonary emboli from left-heart endocarditis through a PFO have not previously been described. An individual case report outlining a case of mitral valve endocarditis migrating through a secundum atrial septal defect has been reported in an adult patient; however, this patient also had septic emboli to multiple systemic organs.[3] A small series of patients experiencing septic pulmonary emboli has also been reported in the adult

population; however, most patients in this series had an underlying cardiac condition such as ventricular septic defects or were in an immunocompromised state. Those that had no underlying medical conditions presented with right-sided cardiac foci with no emboli between the systemic and pulmonary circulations.[4] Although it is not possible to say with certainty that the origin of the septic pulmonary emboli was from the mitral valve vegetation, it remains the most likely explanation. Alternative explanations would be that the bacterial burden was sufficiently high giving rise to a secondary infective source disseminating to the lungs such as tricuspid or pulmonary valve infective endocarditis that was then partially treated. However, the patient did not demonstrate any other localizing symptoms or signs prior to their deterioration. A further possibility may be that the septic pulmonary emboli were derived from the original pharyngitis when the bacterial burden was at its highest, although this does not correlate to the onset of respiratory deterioration on Day 12 of presentation. It is our opinion that in this case, septic emboli were likely facilitated by a high left atrial pressure in the setting of severe MR across the atrial septum. Interestingly, the direction of the MR jets was not directed towards the atrial septum. As PFO exists in 25% of the population, similar types of right-sided septic embolism may occur in a context of patients with mitral valve endocarditis and raised left atrial pressures. The reason why this is not a more common phenomena can only be speculated. However, the constellation of a large, mobile vegetation in relatively close proximity to the atrial septum, raised left atrial pressures, and several severe jets of MR reaching the extremities of the atrium and thereby possibly causing currents towards the atrial septum could explain the probable infective embolism.

Lead author biography

Dr Mohammad Abumehdi is a Congenital Interventional Cardiologist. Having trained in Glasgow, Birmingham and London, he is currently working in The Royal Brompton Hospital, London. His interventional interests are pulmonary artery and vein rehabilitation.

Supplementary material

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

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

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