

Concurrent cardiac and central nervous system complications of acute infective endocarditis: case report

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Background

Cerebral mycotic aneurysms represent a rare but life-threatening complication of infective endocarditis (IE), with high mortality rate when ruptured. Due to the lack of randomized controlled trials, management of infectious aneurysms complicating endocarditis remains a controversial topic.

Case summary

We describe a case of *Streptococcus salivarius* bicuspid aortic and mitral valve endocarditis with concurrent spontaneous mycotic aneurysm rupture and acute subarachnoid haemorrhage (SAH). A 40-year-old man with history of intravenous drug abuse presented to our emergency department with altered mental status and dyspnoea. Echocardiography documented large vegetations on a bicuspid aortic valve and on the mitral valve, causing acute severe aortic and mitral regurgitation. Brain computed tomography imaging documented a ruptured fusiform aneurysm in a distal branch of the right middle cerebral artery causing acute SAH and acute obstructive hydrocephalus. An external ventricular drain was emergently placed and endovascular embolization of the aneurysm was achieved with deployment of six coils. Blood cultures grew *S. salivarius* and antibiotic therapy according to microbiological sensitivities was administered. Hospital stay was complicated by acute heart failure, ST-elevation myocardial infarction, conduction disturbances, cerebral vasospasm, recurrent mycotic aneurysm rupture, and death.

Discussion

Clinicians should be mindful of the rare, potentially severe complication of IE with cerebral mycotic aneurysms to enable prompt treatment. Generally, central nervous system procedures are performed prior to cardiac surgical management of IE, since cardiopulmonary bypass may exacerbate cerebral haemorrhage, ischaemic damage, and oedema in areas of blood–brain barrier disruption. A multidisciplinary collaboration is crucial for optimal patient management.

Keywords

Mycotic aneurysm • Acute subarachnoid haemorrhage • Infective endocarditis • Bicuspid aortic valve • ST-elevation myocardial infarction • *Streptococcus salivarius* • Case report

ESC Curriculum

2.2 Echocardiography • 4.9 Multivalvular disease • 4.11 Endocarditis • 7.3 Critically ill cardiac patient • 9.3 Peripheral artery disease

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Learning points

- Cerebral mycotic aneurysms represent an uncommon but life-threatening complication of infective endocarditis (IE), leading to high mortality rate when ruptured.
- Personalized, multidisciplinary care is crucial to define optimal diagnostic and therapeutic management in patients with concomitant subarachnoid haemorrhage (SAH) and IE.
- ST-elevation myocardial infarction is a rare complication of IE, and SAH itself may result in electrocardiographic changes, confounding clinical assessment.

Primary specialities involved other than cardiology

Neuroradiology, Neuroendovascular intervention.

Introduction

Neurologic complications are common in patients with infective endocarditis (IE) and are potentially life-threatening; they include strokes, intracerebral haemorrhages, mycotic aneurysms, meningitis, cerebral abscesses, and infections of the spine.¹ Mycotic aneurysms are inflammatory vascular lesions with a thin, friable, fusiform shape, generally caused by haematogenous dissemination from septic embolism, usually with bacterial aetiology.² Cerebral mycotic aneurysms represent a rare³ but life-threatening complication of IE, with high mortality rate when ruptured.⁴ Due to lack of randomized controlled trials, management of infectious aneurysms complicating endocarditis remains a controversial topic.⁵

Timeline

Day 1: Presentation to the emergency department with altered mental status and dyspnoea. Transthoracic echocardiogram: mitral–aortic endocarditis, severe aortic [aortic regurgitation (AR)], and mitral regurgitation (MR). Brain computed tomography (CT): acute subarachnoid haemorrhage (SAH), acute obstructive hydrocephalus. Computed tomography angiography: right middle cerebral artery (RMCA) mycotic aneurysm. Emergency external ventricular drain (EVD) placement. Endovascular embolization (six coils) of RMCA ruptured aneurysm. Blood cultures obtained. Empiric antibiotic therapy started

Day 3: Febrile (38.2°C). New electrocardiographic (EKG) ST-segment elevation (STE). Troponin-I 8 ng/mL (cut-off <0.03 ng/mL). Serial troponin levels showed a decreasing trend. Conservative approach was pursued as the patient was at prohibitive risk for surgical/percutaneous coronary revascularization given the high risk of bleeding

Day 5: Development of acute bilateral upper limb paresis. Cerebral angiography: arterial vasospasm, treated with Verapamil, Nimodipine, Milrinone

Day 6: Transesophageal echocardiogram: bicuspid aortic and mitral valve vegetations, aortic root abscess, pseudo-aneurysm formation, severe AR and MR. Blood cultures: *Streptococcus salivarius*+

Day 7: Electrocardiogram showed new 1st degree atrio-ventricular block

Day 10: Acute pulmonary oedema following self-extubation

Day 11: Development of left upper extremity weakness. Cerebral angiography: RMCA vasospasm, treated with intra-arterial Verapamil and stent angioplasty

Day 12: Electrocardiogram: accelerated junctional rhythm with retrograde P waves

Day 15: Computed tomography angiography: worsening basilar artery vasospasm, new left middle cerebral artery mycotic aneurysm

Day 16: Rising intracranial pressure with blood drainage from EVD. Head CT: new left intraparenchymal haematoma from ruptured mycotic aneurysm. EKG: worsening STE, new bigeminy, and multifascicular block without discernible P waves

Day 24: Patient passed away

Case presentation

A 40-year-old man with history of intravenous drug use presented to the emergency department with altered mental status and dyspnoea. He was afebrile and haemodynamically stable. Auscultation of the chest revealed bibasilar lung crepitations and a 3/6 holosystolic murmur at the left

upper sternal border and apex. Neurological exam revealed no focal deficits. The patient denied any relevant past medical and surgical history.

Laboratory tests identified a neutrophilic leucocytosis (white blood cell count 13.6 K/ μ L, reference range 4.5–11 K/ μ L; absolute neutrophil count 11.7 K/ μ L, reference range 1.9–8 K/ μ L) and elevated C-reactive protein (75.2 mg/L, reference range 0–5 mg/L). 12-lead EKG showed normal sinus rhythm (SR) with no conduction disturbances (*Figure 1A*). Transthoracic echocardiogram identified left ventricle (LV) dilation with an ejection fraction 54%, apical akinesis, aortic and mitral valve vegetations with severe regurgitation of both valves (see *Supplementary material online, Figure S1*). Brain CT showed an acute SAH (*Figure 2A and B*), with acute obstructive hydrocephalus (*Figure 2C*). Computed tomography angiography revealed a 4-mm irregular-shaped aneurysm arising from a RMCA branch (*Figure 2D*).

The patient was intubated and an EVD was emergently placed. Digital subtraction angiography (DSA) documented a ruptured fusiform mycotic aneurysm in a distal parietal branch of the RMCA (*Figure 3A and B*). Under fluoroscopic guidance, a Headway Duo micro-catheter was advanced over a 0.014" micro-guidewire into the RMCA. Embolization of the aneurysm was achieved with deployment of six coils, followed by n-butyl cyanoacrylate glue embolization (*Figure 3C*). Final DSA showed excellent aneurysm obliteration (Raymond Roy Grade 1; *Figure 3D*). Blood cultures

were collected before empiric antibiotic therapy with Vancomycin 1 gr IV every 12 h and Ceftriaxone 2 g IV daily was started.

On Day 3, the patient became febrile (38.2°C) and EKG showed new STE in Leads II, III, aVF, V3–V4 (*Figure 1B*). Troponin-I was 8 ng/mL (cut-off <0.03 ng/mL). Serial troponin levels showed a decreasing trend. A

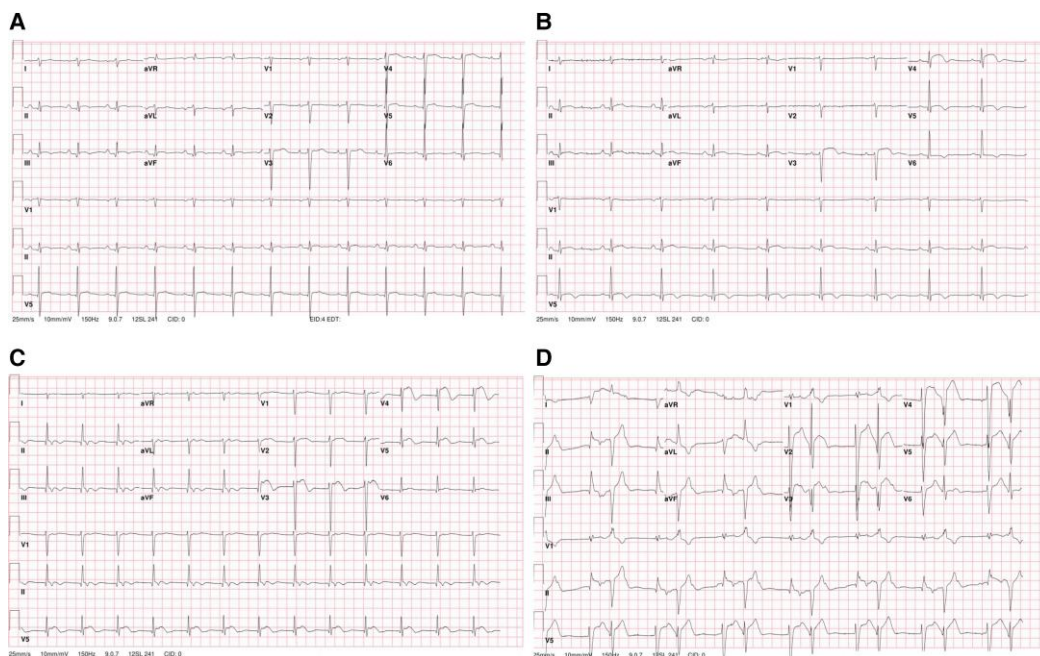


Figure 1 Electrocardiogram. Electrocardiogram evolution: normal sinus rhythm at arrival (A), new ST-elevation in Leads II, III, aVF, V3–V4 (B), accelerated junctional rhythm with retrograde P waves (C).

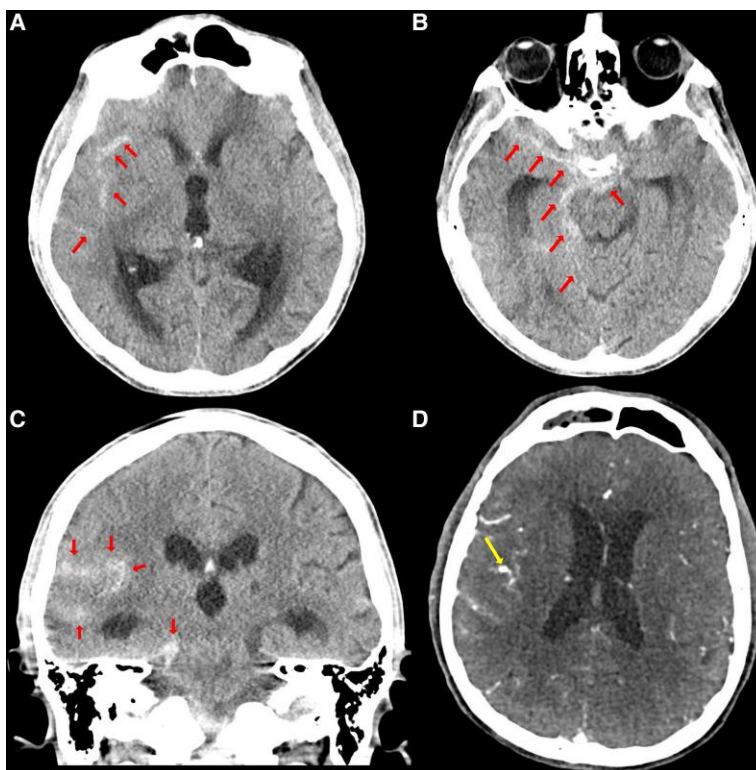


Figure 2 Head computed tomography. Non-contrast head computed tomography demonstrated acute subarachnoid haemorrhage (red arrows) within Sylvian fissure (A) and basal cisterns (B) and acute obstructive hydrocephalus (C, red arrows). Computed tomography angiography showed a mycotic aneurysm (yellow arrow) in the distal right middle cerebral artery branch (2D).

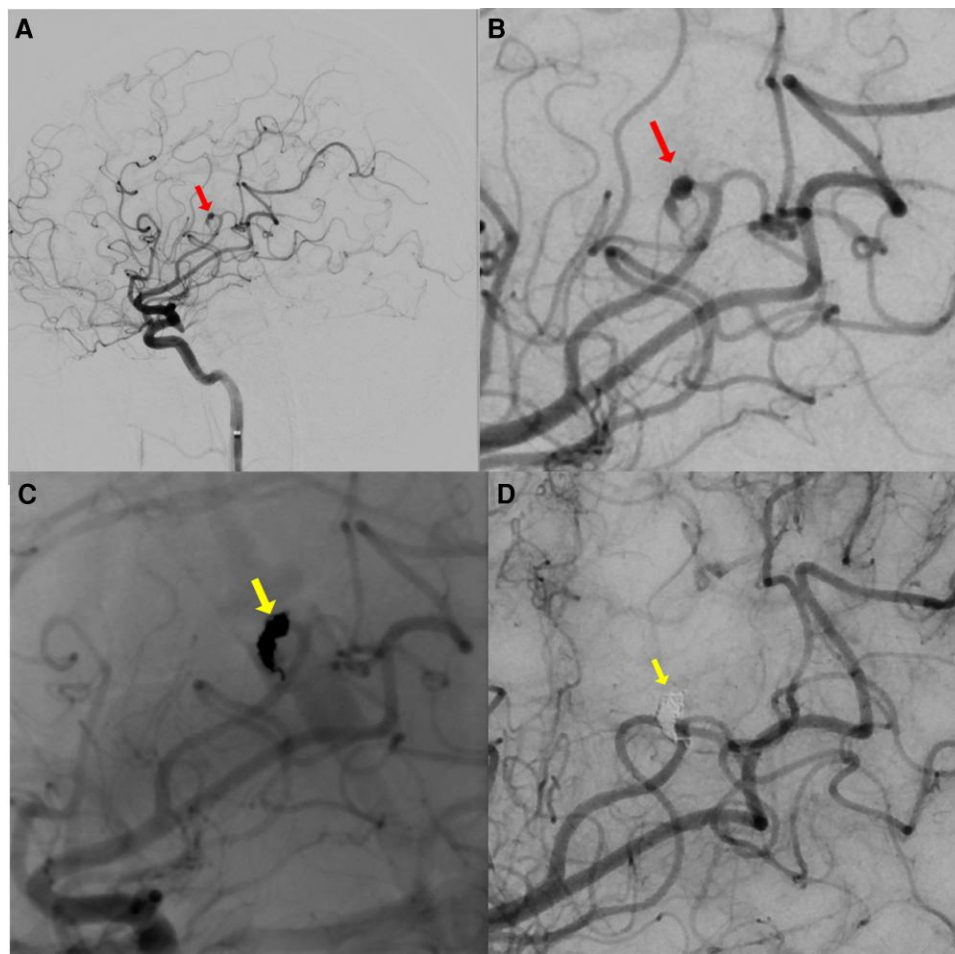


Figure 3 Aneurysm embolization. Right internal carotid artery injection demonstrating fusiform aneurysm (red arrow) in distal right middle cerebral artery branch (A). Magnification of (A) demonstrates the aneurysm (red arrow) in greater detail (B). Embolization cast completely obliterating the aneurysm (C, yellow arrow), with Raymond Roy grade 1 result (D, yellow arrow).

conservative approach without diagnostic coronary angiography was pursued, as the patient was at prohibitive risk for surgical or percutaneous revascularization if significant coronary artery disease was confirmed, given the attendant bleeding risk in the setting of recent SAH and EVD placement.

On Day 5, the patient developed bilateral upper limb paresis. Cerebral angiography demonstrated RMCA and anterior cerebral artery vasospasm, which was treated with Verapamil, Nimodipine, Milrinone, and monitored with transcranial Doppler.

Transesophageal echocardiography (TEE; [Figures 4 and 5](#), see [Supplementary material online, Figure S2 and Video S1](#)) identified a Sievers Type 0 bicuspid aortic valve with a large vegetation (1.8×1 cm) arising from the left cusp; there was associated left cusp perforation resulting in severe AR; perivalvular infection was also seen, with an aortic root abscess, and pseudo-aneurysm formation. The mitral valve also had a large vegetation (2.4×1.2 cm) with anterior mitral leaflet aneurysm and perforation, resulting in severe MR. There was no TEE evidence of left main (LM) coronary ostium obstruction. Blood cultures revealed growth of *Streptococcus salivarius*; Ceftriaxone and Vancomycin were continued according to microbiological sensitivities.

On Day 7, an EKG showed SR with new 1st degree atrio-ventricular block and stable V3–V5 STE. The clinical course was then complicated by development of hypotension, acute pulmonary oedema, pneumonia,

and progressive left upper extremity weakness due to cerebral vasospasm, requiring intra-arterial Verapamil and stent angioplasty at the first segment of the RMCA. Electrocardiogram showed stable V3–V5 STE and accelerated junctional rhythm with retrograde P waves ([Figure 1C](#)).

On Day 15, fluctuating conscious levels and neurological deficits prompted CT-angiography, which showed worsening basilar artery vasospasm and a new mycotic aneurysm in the distal M2 left middle cerebral artery branch (see [Supplementary material online, Figure S3A](#)). The patient progressively deteriorated with rising intracranial pressure and drainage of blood from the EVD. There was also additional cardio-respiratory decompensation. Head CT showed a new left parieto-temporal intraparenchymal haematoma from a ruptured left distal mycotic aneurysm, with rightward midline shift and left lateral ventricle effacement (see [Supplementary material online, Figure S3B–D](#)). Electrocardiogram showed worsening anterior STE with new bigeminy and multifascicular block without discernible P waves ([Figure 1D](#)). Following multidisciplinary team meetings and Ethics committee reunion, the patient was managed conservatively and subsequently passed away on Day 24.

Discussion

Streptococcus salivarius (viridans streptococci family) is a commensal microorganism of the human oral mucosa and gut, that may rarely

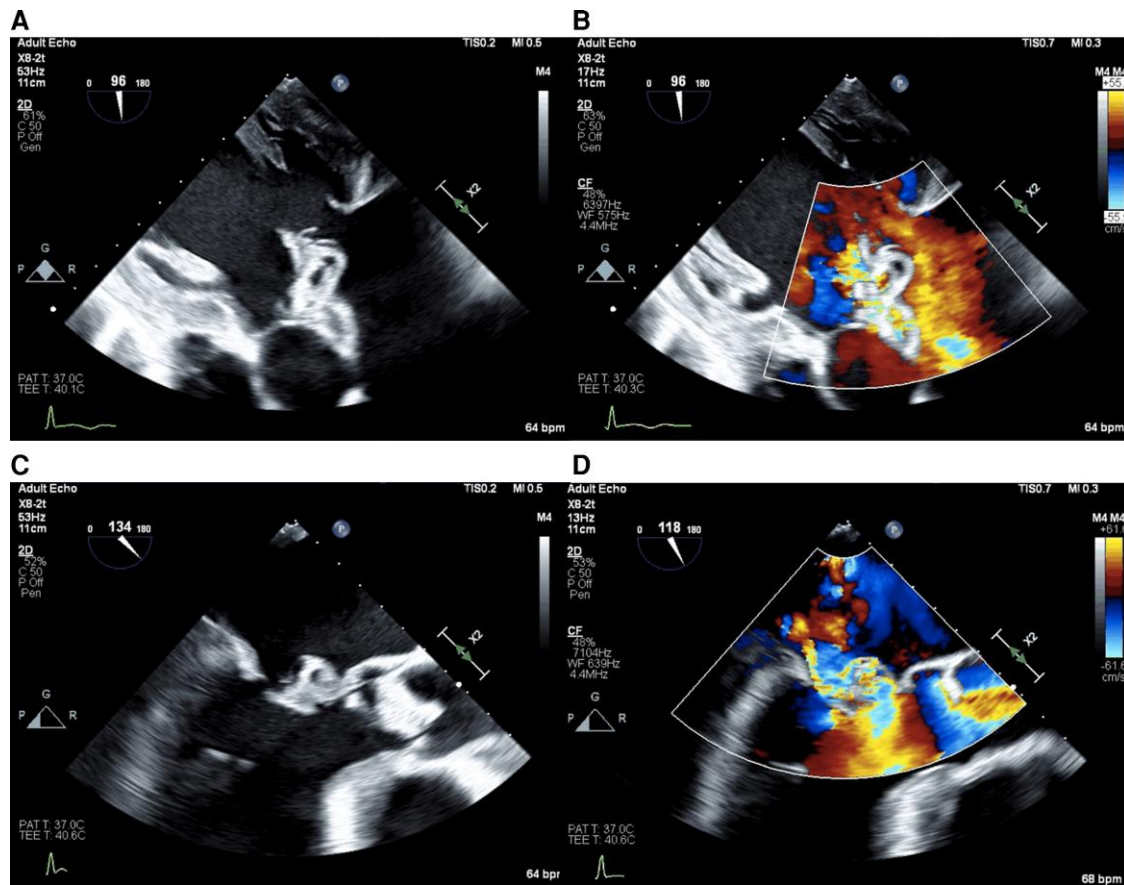


Figure 4 Transesophageal echocardiogram. Aortic and mitral valve endocarditis, with aortic root abscess and pseudo-aneurysm (A–D).

cause bacterial IE and mycotic aneurysms.⁶ Referring to modified Duke’s criteria,⁷ our patient met both major criteria (two positive blood cultures for known endocarditis-causing bacteria and positive findings for endocarditis on TEE) and multiple minor criteria (bicuspid aortic valve, intravenous drug use, mycotic aneurysms, and intracranial haemorrhage).

Symptomatic neurological complications occur in 15–30% of patients with IE.⁸ The reported incidence of mycotic aneurysms in IE patients is only 2–3%, but the true incidence is probably higher since they are often clinically silent prior to rupture and often resolve on antibiotic therapy.³ Viridans group streptococci and *Staphylococcus aureus* are responsible for 57–91% of infectious aneurysms.⁵ They are multiple in up to 25% of cases and have a predilection for the distal branching points of the middle cerebral artery.⁵ A ruptured mycotic aneurysm is associated with high mortality rate (80%)³ and no reliable predictor of rupture has been identified.⁶ Subarachnoid haemorrhage represents 5% of IE neurologic complications⁹ and rarely is seen at the time of presentation.

Computed tomography scanning is the most useful initial test to evaluate a patient with endocarditis and neurological symptoms, particularly when intracranial or SAH is suspected,⁵ although conventional angiography is considered the gold standard for mycotic aneurysms detection due to their propensity for distal locations.¹⁰

Due to lack of randomized controlled trials, management of infectious aneurysms complicating endocarditis remains a controversial topic.⁵ Generally, central nervous system procedures (endovascular or neurosurgical) are performed prior to cardiac surgical management of IE, since cardiopulmonary bypass may exacerbate cerebral haemorrhage, ischaemic damage, and oedema in areas of blood–brain barrier

disruption.³ A 2-to-3 weeks waiting period on antibiotics after craniotomy for a ruptured aneurysm has been recommended before undergoing cardiac surgery,¹¹ but patients treated endovascularly may need a shorter waiting period.¹² Selective endovascular coil embolization of the aneurysmal sac is the preferable method for infected and distal branch intracranial aneurysms.²

ST-elevation myocardial infarction is a very rare (3%) complication of IE, generally related to coronary artery embolism, coronary ostia obstruction by a large vegetation, coronary vasospasm, or coronary artery compression by an abscess.¹³ In our patient, the LM coronary ostium appeared patent on TEE and type II mechanisms may have contributed to myocardial infarction through supply-demand mismatch: severe AR with reduced effective diastolic coronary blood flow, and AR related rise in LV filling pressures and wall stress increasing oxygen demand. There was additional reduction in cardiac output and coronary perfusion due to severe MR. Also, patients with SAH and no significant obstructive coronary artery disease may develop EKG changes, segmental wall motion abnormalities, and elevated cardiac biomarkers, through enhanced sympathetic activity, increased catecholamine release, myocardial contractility, and increased myocardial demand leading to myocardial necrosis. It is crucial to consider neurological factors in the setting of IE that may result in EKG changes, as antithrombotic therapy and delayed diagnosis in SAH could result in poorer outcomes.¹⁴ Takotsubo cardiomyopathy is another alternative explanation for the ST-elevation EKG changes and left ventricular apical akinesis seen in the present case. However, as stated in the ESC Expert Consensus Document on Tako-Tsubo syndrome (TTS), in TTS peak troponin values are substantially lower compared with the classical acute coronary

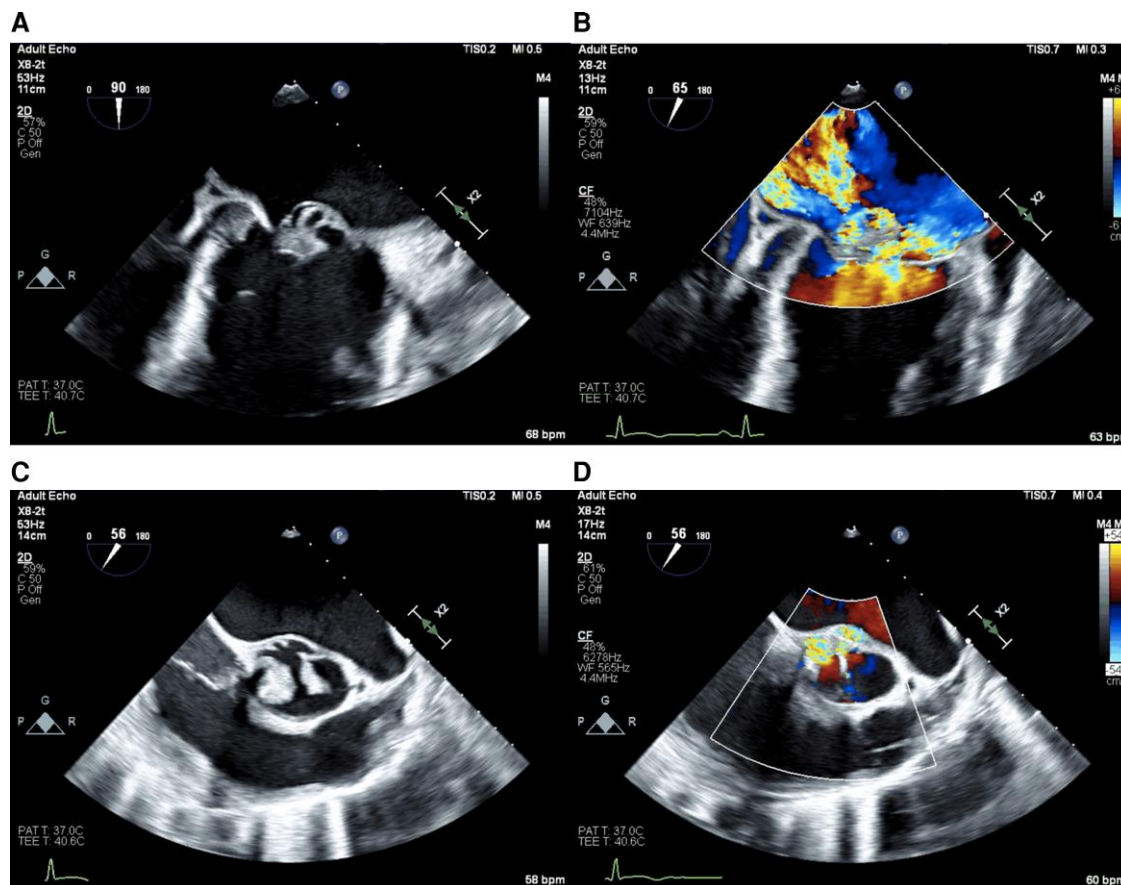


Figure 5 Transesophageal echocardiogram. Aorto-mitral abscess (A) with an anterior mitral aneurysm, leaflet perforation, and severe mitral regurgitation (B). Bicuspid aortic valve endocarditis (C) with pseudo-aneurysm (D).

syndrome, and the extent of LV regional wall motion impairment greatly exceeds that of associated myocardial necrosis.¹⁵ The InterTAK diagnostic score was 18 points in our patient, and predicted probability of TTS in patients with <30 points was <1%.

Identifying the IE related mycotic aneurysms and SAH in a timely manner is crucial for stratifying patient prognosis and planning management. Clinicians should consider IE as an underlying aetiology of SAH, particularly in the presence of fever, a new regurgitant cardiac murmur, and concomitant risk factors for IE. Multidisciplinary collaboration contributes to optimal patient care and decision making.

Lead author biography



Francesca Romana Prandi graduated as MD at La Sapienza University of Rome, Italy. She started her Cardiology Fellowship training at University of Rome Tor Vergata, Italy and is currently a Postdoc Research Fellow in Structural Cardiovascular Imaging at Mount Sinai Hospital, New York, USA.

Supplementary material

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

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We acknowledge the important contribution from all the health care personnel involved in the treatment of the patient.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The patient reported in this case is deceased. Despite the best efforts of the authors, they have been unable to contact the patient's next-of-kin to obtain consent for publication. Every effort has been made to anonymise the case. This situation has been discussed with the editors.

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Declaration

All authors have seen and approve the paper. The article is the original work of all the authors listed. The article has not been published elsewhere and is not under consideration elsewhere.

Data availability

The data underlying this article are available in the article and in its online [supplementary material](#).

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