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Complete Atrioventricular Block Revealing Extensive Periannular Destruction in Fulminant Pneumococcal Endocarditis: A Case Report

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Abstract

Streptococcus pneumoniae infective endocarditis is a rare but highly aggressive infection characterized by rapid valvular destruction, extensive periannular complications, and substantial mortality. We report the case of a previously healthy 25-year-old woman presenting with fever, profound asthenia, and complete atrioventricular block with a ventricular escape rhythm of 40 beats/min. Despite initially preserved blood pressure and left ventricular ejection fraction (62%), clinical signs of peripheral hypoperfusion were present. Urgent transthoracic and transesophageal echocardiography revealed extensive aortic valve destruction with severe aortic regurgitation, concomitant mitral valve involvement, and a large aortic root abscess (45 × 27 mm) extending toward the interatrial septum, with an additional vegetation identified in the right atrium. Blood cultures were obtained, and empirical intravenous antibiotic therapy was promptly initiated. Emergency cardiac surgery with planned intraoperative epicardial pacing was indicated. However, approximately one hour after cardiac intensive care unit admission, the patient developed abrupt profound hypotension followed almost immediately by cardiac arrest with documented asystole. Despite advanced resuscitative efforts, she died approximately two hours after admission, before definitive intervention could be performed. Blood cultures subsequently identified *S. pneumoniae*, with microbiological confirmation becoming available only after death. This case highlights complete atrioventricular block as a critical warning sign of extensive periannular infection and demonstrates that preserved left ventricular systolic function and initially maintained blood pressure do not exclude impending cardiovascular collapse. Early recognition, immediate multidisciplinary coordination, and expedited surgical management are essential, as the therapeutic window in fulminant pneumococcal endocarditis may be exceptionally narrow.

Categories: Cardiology
Keywords: acute severe aortic regurgitation, aortic root abscess, complete atrioventricular block, infective endocarditis, streptococcus pneumoniae

Introduction

Infective endocarditis (IE) remains a life-threatening cardiovascular infection, associated with substantial morbidity and mortality despite advances in antimicrobial therapy, multimodality imaging, and cardiac surgery [1,2]. Periannular extension represents one of its most destructive complications, potentially leading to aortic root abscess formation, multivalvular involvement, and life-threatening conduction disturbances [3,4]. Owing to the close anatomical relationship between the aortic root and the atrioventricular conduction system, new-onset atrioventricular block is a critical warning sign of invasive infection. According to the 2023 European Society of Cardiology (ESC) guidelines, complete atrioventricular block is observed in only 2.8% of patients with IE at diagnosis, highlighting its rarity and clinical significance [3]. Early recognition of these complications through transesophageal echocardiography (TEE) is essential for timely surgical management [3,5].

We report an exceptionally fulminant case of *Streptococcus pneumoniae* IE in a previously healthy 25-year-old woman, characterized by extensive aortic valve destruction with severe aortic regurgitation, concomitant mitral valve involvement, a large periannular aortic root abscess extending into the interatrial septum, and vegetations protruding into the right atrium. This aggressive pneumococcal infection was associated with complete atrioventricular block and rapidly progressive cardiogenic shock, culminating in fatal cardiac arrest despite preserved left ventricular systolic function. This case underscores the devastating potential of invasive pneumococcal endocarditis and highlights how electrical instability may herald extensive intracardiac destruction and precede catastrophic hemodynamic collapse, even in the absence of ventricular systolic dysfunction.

Case Presentation

A 25-year-old woman with no significant past medical history presented to the emergency department with a two-day history of profound asthenia and progressive deterioration in her general condition. She denied

syncope, presyncope, or loss of consciousness. She had no history of congenital or acquired heart disease, previous cardiac surgery, prosthetic valve implantation, or intravenous drug use. No known drug allergies were reported. Her pneumococcal vaccination status was unknown.

Further history revealed an episode of fever approximately two weeks before admission, with a self-reported temperature reaching 38°C, without associated significant weight loss. No identifiable infectious source was apparent. The patient denied respiratory symptoms, including cough and dyspnea, as well as urinary or gastrointestinal complaints. There was no history of recent travel to tropical or endemic regions, and no apparent cutaneous portal of entry was identified.

On admission, the patient was conscious and initially normotensive despite marked bradycardia, with a heart rate of 40 beats/min, a blood pressure of 110/80 mmHg, and a body temperature of 38°C. She was clinically stable from a respiratory standpoint, with no dyspnea or orthopnea, a respiratory rate of 16 breaths/min, an oxygen saturation of 100% on room air, and unremarkable pulmonary auscultation.

Cardiovascular auscultation revealed a prominent grade 5/6 diastolic murmur, best heard over the aortic area, suggestive of severe aortic regurgitation. Additional murmurs consistent with mitral and tricuspid regurgitation were also detected. Despite initially preserved blood pressure, signs of peripheral hypoperfusion were present, including cold extremities and a prolonged capillary refill time of four seconds, without mottling or diaphoresis. Oral examination revealed good dental hygiene, with no apparent dental infection or poor dentition.

A meticulous peripheral examination was performed to identify classical manifestations of IE and potential infectious portals of entry. Cutaneous examination was unremarkable, with no visible wounds, skin lesions, or apparent cutaneous infectious focus. No Osler nodes or Janeway lesions were identified. Other peripheral manifestations, including splinter hemorrhages, Roth spots, petechiae, and peripheral embolic phenomena, were not documented in the available clinical findings.

A 12-lead electrocardiogram performed immediately after admission demonstrated complete (third-degree) atrioventricular block, characterized by atrioventricular dissociation and a slow, narrow-QRS ventricular escape rhythm at approximately 40 beats/min (Figure *1*).

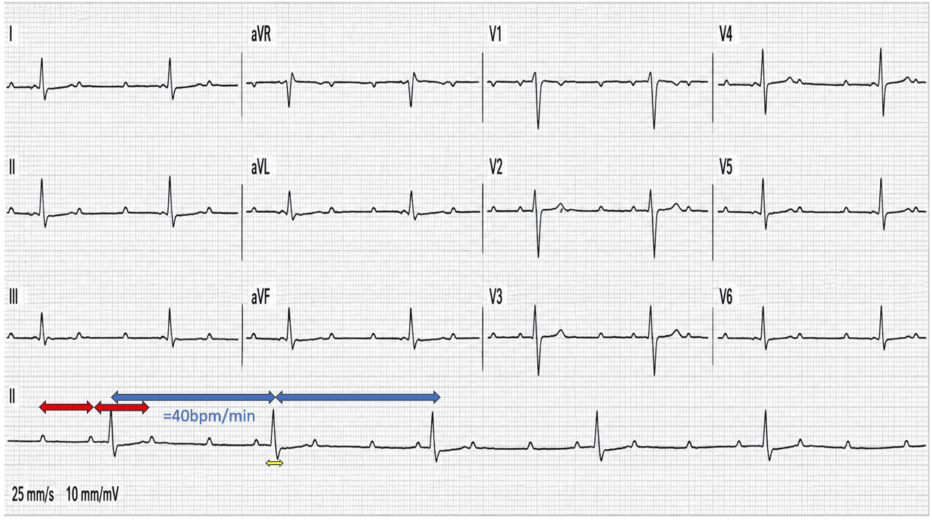


FIGURE 1: Twelve-lead electrocardiogram at admission demonstrating complete atrioventricular block.

The lead II rhythm strip demonstrates regular P waves occurring independently of the QRS complexes, indicating complete atrioventricular dissociation. The regular R-R intervals (blue arrows) demonstrate a slow ventricular escape rhythm of approximately 40 beats/min, with narrow QRS complexes (yellow arrow).

The chest radiograph obtained on admission was unremarkable, showing a normal cardiac silhouette without cardiomegaly, no evidence of pulmonary vascular congestion or hilar overload, and no parenchymal abnormalities.

Despite complete atrioventricular block with a ventricular escape rhythm of approximately 40 beats/min, the patient remained conscious and initially normotensive, with a blood pressure of 110/80 mmHg, although clinical signs of peripheral hypoperfusion were present. The association of fever, a prominent cardiac murmur, and complete atrioventricular block raised strong suspicion of IE with periannular extension.

Urgent transthoracic echocardiography (TTE), followed by TEE performed without intravenous sedation under continuous monitoring, was undertaken to characterize the valvular and periannular lesions, assess the extent of intracardiac involvement, and guide emergency surgical management. Emergency cardiac pacing equipment was immediately available in the echocardiography room throughout the examinations.

Urgent TTE revealed extensive destructive IE involving the aortic and mitral valves, with marked periannular infectious involvement and interatrial septal thickening. The left ventricle was non-dilated, with an end-diastolic diameter of 53 mm (indexed: 25 mm/m^2) and an end-systolic diameter of 35 mm (indexed: 16 mm/m^2). In the setting of extensive aortic valve destruction, the absence of left ventricular dilatation, together with a non-widened pulse pressure of 30 mmHg, supported the acute nature of the severe aortic regurgitation.

The tricuspid aortic valve exhibited severe structural destruction, with a large, highly mobile, multilobulated vegetation measuring 20 × 12 mm, predominantly involving the right coronary and noncoronary cusps. Flail of the right coronary cusp was associated with severe aortic regurgitation. Marked periannular echogenic thickening adjacent to the aortic root raised suspicion of extensive infectious extension (Figure *2*).

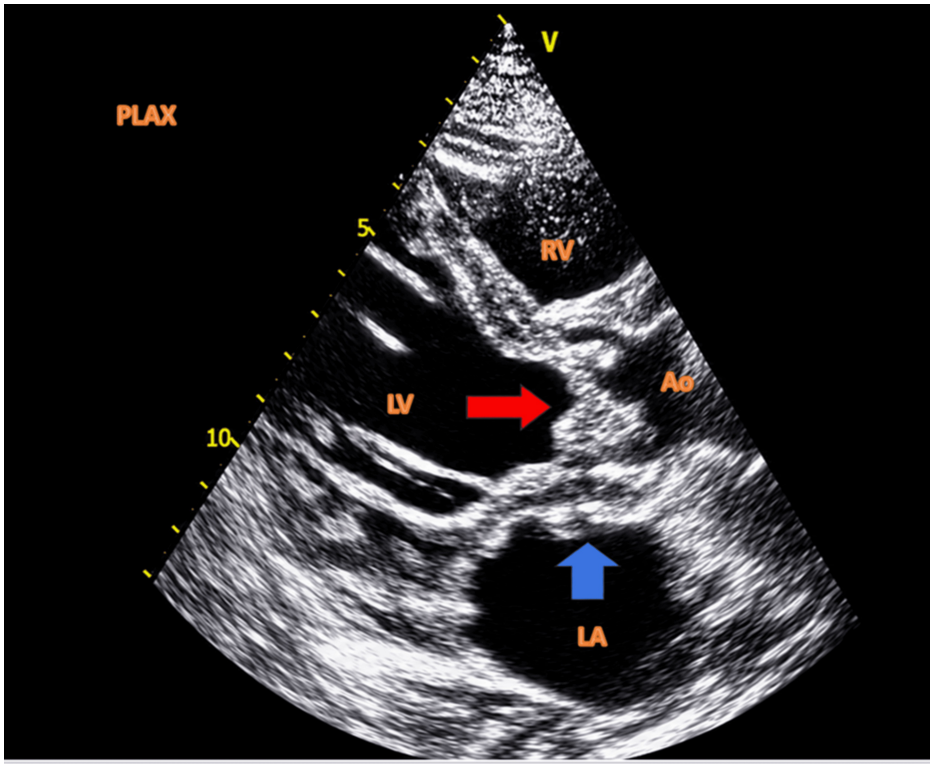


FIGURE 2: Transthoracic echocardiography demonstrating aortic valve vegetation and suspected periannular infectious extension.

Parasternal long-axis (PLAX) view showing an irregular echogenic mass involving the aortic valve, consistent with a vegetation (red arrow), and abnormal echogenic thickening adjacent to the aortic root (blue arrow), raising suspicion of periannular infectious extension.

Ao: aorta; LA: left atrium; LV: left ventricle; RV: right ventricle

Additionally, a periannular cavity measuring 12 × 9 mm was identified adjacent to the aortic root. Color Doppler imaging demonstrated flow within this cavity, raising suspicion of communication with the aortic lumen and possible periannular pseudoaneurysm formation (Figure *3*).

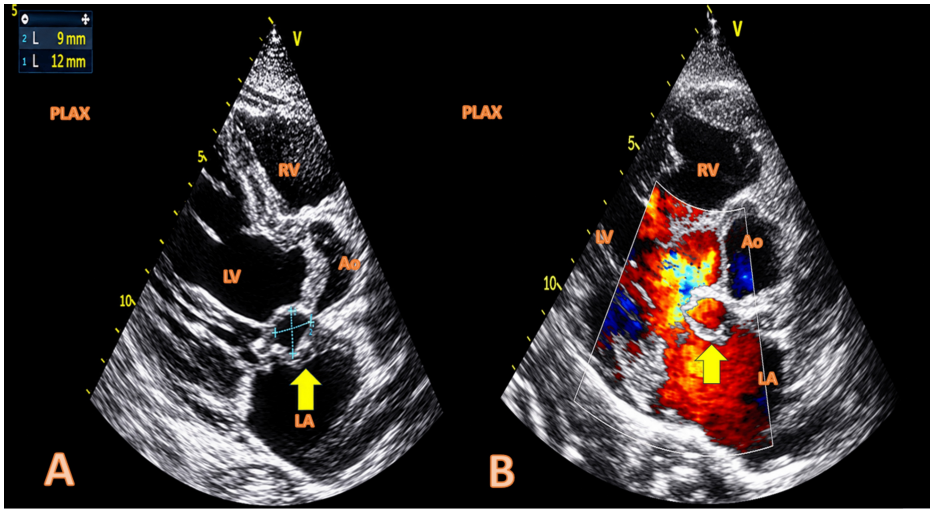


FIGURE 3: Transthoracic echocardiography in the parasternal long-axis (PLAX) view demonstrating a periannular cavity with color Doppler flow.

(A) Two-dimensional imaging showing a 12 × 9 mm periannular cavity adjacent to the aortic root (yellow arrow). (B) Corresponding color Doppler imaging demonstrating blood flow within the cavity (yellow arrow), raising suspicion of a communicating periannular pseudoaneurysm.

Ao: aorta; LA: left atrium; LV: left ventricle; RV: right ventricle

Particular attention was drawn to the interatrial septum, which appeared markedly thickened, predominantly at its basal portion adjacent to the aortic root, raising suspicion of extensive infectious infiltration. An additional vegetation was identified within the right atrium, adjacent to the interatrial septum, further highlighting the extensive intracardiac involvement (Figure *4*).

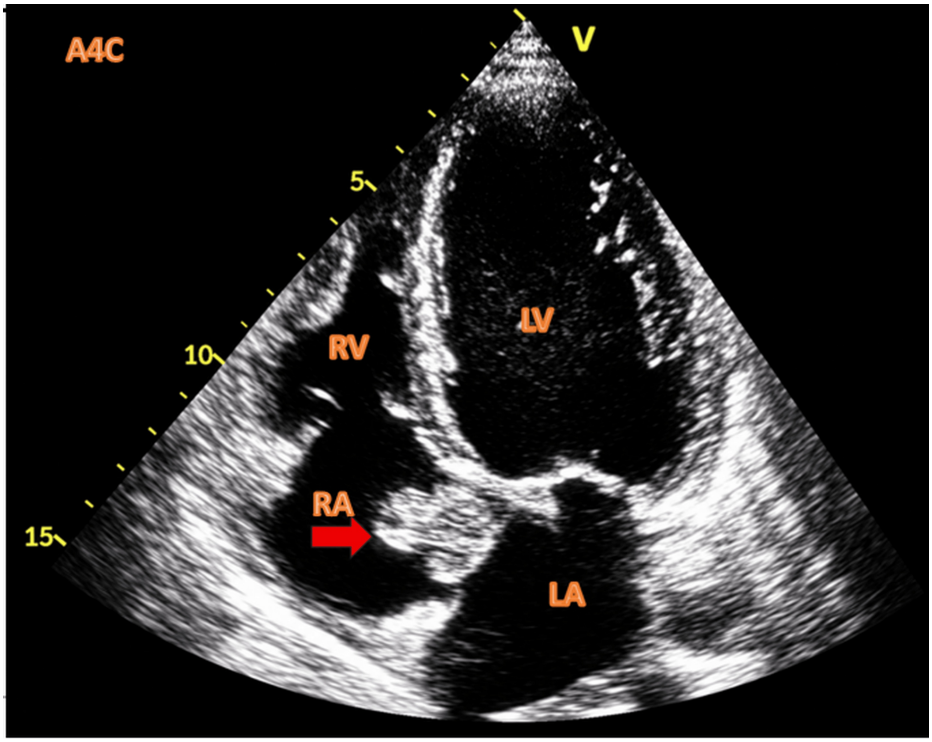


FIGURE 4: Transthoracic echocardiography in the apical four-chamber (A4C) view demonstrating a right atrial vegetation.

A4C view showing an irregular echogenic mass within the right atrium, adjacent to the interatrial septum (red arrow), consistent with a vegetation in the setting of extensive infective endocarditis.

RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle; IAS: interatrial septum

Color Doppler examination demonstrated severe aortic regurgitation, characterized by a broad, turbulent regurgitant jet extending into the left ventricular outflow tract. Continuous-wave Doppler assessment further demonstrated prominent holodiastolic flow reversal in the descending thoracic aorta, supporting the severity of aortic regurgitation (Figure *5*).

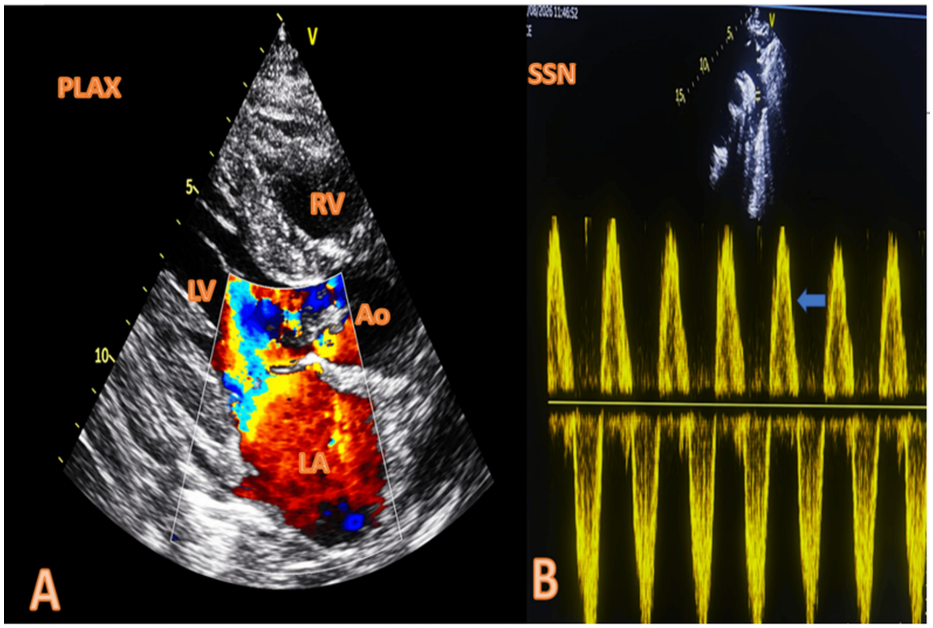


FIGURE 5: Transthoracic echocardiography in the parasternal long-axis (PLAX) and suprasternal notch (SSN) views demonstrating severe aortic regurgitation.

(A) PLAX view with color Doppler showing a broad, turbulent aortic regurgitant jet extending into the left ventricular outflow tract. (B) Suprasternal notch view with continuous-wave Doppler demonstrating prominent holodiastolic flow reversal in the descending thoracic aorta (blue arrow), supporting the diagnosis of severe aortic regurgitation.

Ao: aorta; LA: left atrium; LV: left ventricle; RV: right ventricle

Urgent TEE, performed without intravenous sedation under continuous monitoring, further characterized the extent of valvular and periannular destruction. A large aortic root abscess measuring approximately 45 × 27 mm was identified, extending posteriorly and superiorly toward the interatrial septum. Mid-esophageal short-axis imaging demonstrated extensive aortic valve destruction associated with severe aortic regurgitation and periannular infectious involvement. The mid-esophageal long-axis view further demonstrated a large aortic valve vegetation measuring 20 × 12 mm (Figure *6*).

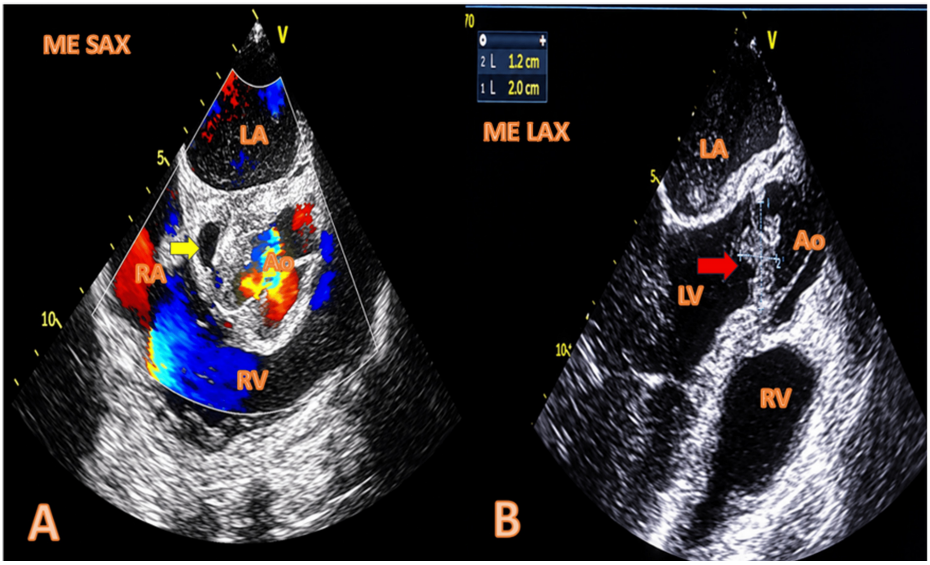


FIGURE 6: Transesophageal echocardiography demonstrating aortic valve destruction, severe aortic regurgitation, and periannular abscess formation.

(A) Transesophageal echocardiography in the aortic valve short-axis view with color Doppler showing extensive aortic valve destruction associated with severe aortic regurgitation and a periannular abscess (yellow arrow). (B) Mid-esophageal three-chamber view demonstrating a large aortic valve vegetation measuring 20 × 12 mm (red arrow).

ME SAX: mid-esophageal short-axis view; ME LAX: mid-esophageal long-axis view; LA: left atrium; RA: right atrium; RV: right ventricle; LV: left ventricle; Ao: aorta

TEE also revealed concomitant mitral valve involvement, with a large vegetation measuring 13 × 9 mm associated with moderate mitral regurgitation. The mid-esophageal bicaval view demonstrated an additional vegetation measuring 18 × 10 mm within the right atrium, adjacent to the interatrial septum, further highlighting the extensive intracardiac involvement of the infection (Figure 7).

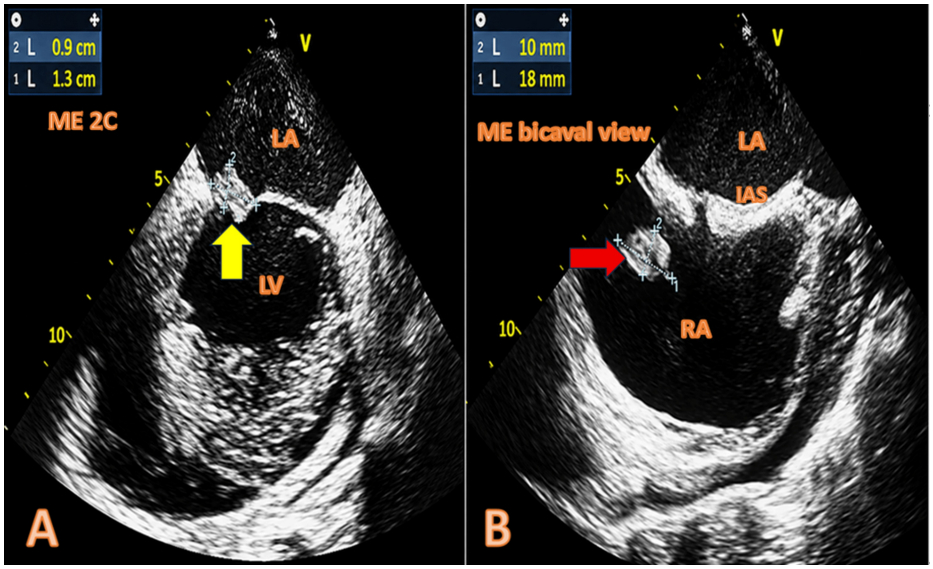


FIGURE 7: Transesophageal echocardiography demonstrating mitral valve and right atrial vegetations.

(A) Transesophageal echocardiography in the two-chamber view showing a large vegetation attached to the mitral valve, measuring 13 × 9 mm. (B) Bicaval view demonstrating a vegetation within the right atrium, measuring 18 × 10 mm, highlighting the extensive intracardiac spread of infective endocarditis.

ME 2C: mid-esophageal two-chamber view; LA: left atrium; LV: left ventricle; RA: right atrium; IAS: interatrial septum

Further assessment of the interatrial septum in the mid-esophageal bicaval view revealed marked echogenic thickening, predominantly involving its basal portion, suggestive of extensive infectious infiltration in the setting of periannular extension. Although these findings indicated widespread intracardiac involvement, the complete atrioventricular block observed on admission was more likely related to the extension of the aortic root infection toward the adjacent atrioventricular conduction system (Figure *8*).

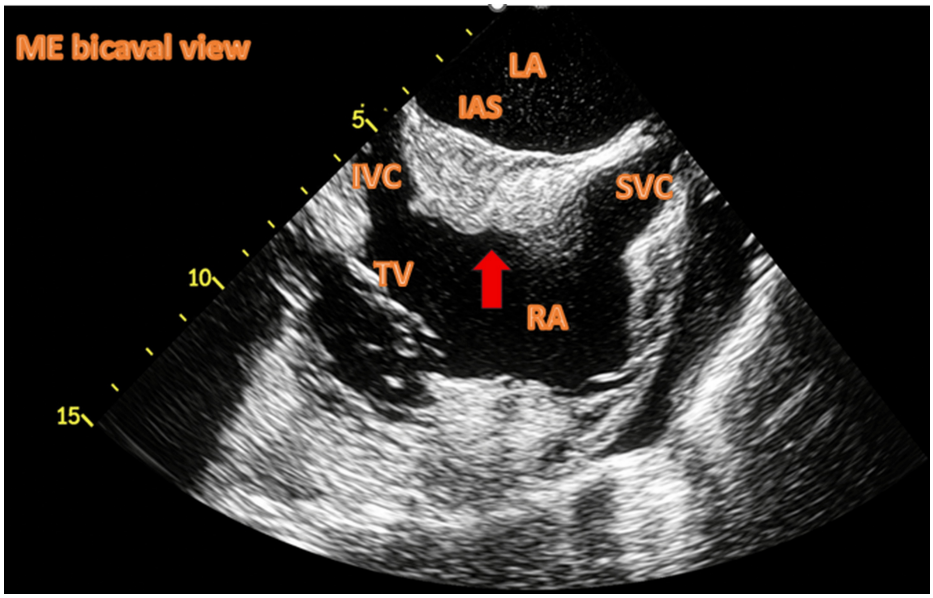


FIGURE 8: Transesophageal echocardiography (TEE) demonstrating interatrial septal thickening associated with atrioventricular conduction disturbances.

Mid-esophageal (ME) bicaval view showing marked, diffuse echogenic thickening of the interatrial septum (red arrow), suggestive of extensive infectious infiltration in the setting of destructive infective endocarditis with periannular extension.

IAS: interatrial septum; RA: right atrium; LA: left atrium; SVC: superior vena cava; IVC: inferior vena cava

Despite extensive valvular and periannular destruction, left ventricular systolic function remained preserved, with an estimated ejection fraction of 62%. Right ventricular systolic function was also preserved. The coexistence of a large aortic root abscess, multivalvular involvement, suspected infectious extension toward the interatrial septum, severe aortic regurgitation, and complete atrioventricular block reflected an exceptionally aggressive and advanced form of IE, warranting emergency surgical intervention.

Initial laboratory investigations revealed marked leukocytosis (20,000/µL), elevated C-reactive protein (200 mg/L), and increased procalcitonin (4 ng/mL), consistent with a severe systemic inflammatory response. Hemoglobin was 14 g/dL, and the platelet count was 250,000/µL. Renal function was preserved, with a serum creatinine concentration of 0.7 mg/dL and a blood urea level of 0.30 g/L. Serum potassium was 4.5 mmol/L, while aspartate aminotransferase and alanine aminotransferase levels were both 30 U/L. Serum lactate was 3 mmol/L, and N-terminal pro B-type natriuretic peptide (NT-proBNP) was elevated to 2,000 pg/mL. The laboratory findings are summarized in Table *1*.

Laboratory parameter	Patient's value	Reference range
White blood cell count	20,000/µL	4,000-10,000/µL
Hemoglobin	14 g/dL	12-16 g/dL
Platelet count	250,000/µL	150,000-400,000/µL
C-reactive protein	200 mg/L	<5 mg/L
Procalcitonin	4 ng/mL	<0.05 ng/mL
Fibrinogen	5 g/L	2-4 g/L
High-sensitivity cardiac troponin	15 ng/L	<35 ng/L (female)
NT-proBNP	2,000 pg/mL	<125 pg/mL
Serum creatinine	0.7 mg/dL	0.5-1.1 mg/dL
eGFR (CKD-EPI 2021)	123 mL/min/1.73 m^2	≥90 mL/min/1.73 m^2
Blood urea	0.30 g/L	0.15-0.45 g/L
Sodium	135 mmol/L	135-145 mmol/L
Potassium	4.5 mmol/L	3.5-5.0 mmol/L
INR	1.0	0.8-1.2
Complement C3	100 mg/dL	90-180 mg/dL
Complement C4	30 mg/dL	10-40 mg/dL
Lactate	3 mmol/L	0.5-2.0 mmol/L
AST	30 U/L	10-35 U/L
ALT	30 U/L	7-35 U/L

TABLE 1: Laboratory findings on admission.

Initial laboratory investigations revealed marked leukocytosis, elevated inflammatory markers, increased NT-proBNP levels, and mild hyperlactatemia, consistent with a severe systemic inflammatory response. Renal function, liver enzymes, coagulation parameters, serum complement levels, and high-sensitivity cardiac troponin remained within their respective reference ranges. Reference ranges may vary according to the laboratory.

ALT: alanine aminotransferase; AST: aspartate aminotransferase; eGFR: estimated glomerular filtration rate; INR: international normalized ratio; NT-proBNP: N-terminal pro-B-type natriuretic peptide; CKD-EPI: Chronic Kidney Disease Epidemiology Collaboration

Multiple sets of blood cultures were obtained before the initiation of empirical intravenous antibiotic therapy consisting of ampicillin (12 g/day), cloxacillin (12 g/day), and gentamicin (3 mg/kg/day).

Following echocardiographic evaluation, the patient was admitted to the cardiac intensive care unit for continuous electrocardiographic and hemodynamic monitoring. Although she remained conscious and initially normotensive, clinical signs of peripheral hypoperfusion were present. Complete atrioventricular block with a ventricular escape rhythm of approximately 40 beats/min raised significant concern for further conduction system deterioration and sudden hemodynamic compromise. Given the extensive aortic and mitral valve destruction, severe aortic regurgitation, and large periannular aortic root abscess, emergency cardiac surgery was indicated. In view of the mobile right atrial vegetation and the potential risk of vegetation disruption during transvenous lead manipulation, intraoperative epicardial pacing was planned as part of the emergency surgical management.

Additional investigations for extracardiac infectious and embolic complications, including contrast-enhanced thoraco-abdomino-pelvic computed tomography, were considered but could not be performed because of the rapidly evolving clinical course. These investigations were not intended to delay emergency surgical intervention.

Approximately one hour after admission to the cardiac intensive care unit, the patient developed abrupt profound hypotension, with a blood pressure of 50/30 mmHg, followed almost immediately by cardiac arrest with documented asystole. Advanced cardiopulmonary resuscitation was promptly initiated, including

external chest compressions and intravenous epinephrine administration. Despite sustained resuscitative efforts, return of spontaneous circulation could not be achieved. No repeat TTE was performed during or immediately after the cardiovascular collapse because of the rapid deterioration and ongoing resuscitation. The patient was pronounced dead approximately two hours after admission to the cardiac intensive care unit, before emergency cardiac surgery or the planned intraoperative epicardial pacing could be performed.

Blood cultures subsequently yielded *S. pneumoniae*, with antimicrobial susceptibility testing demonstrating susceptibility to all antibiotics tested (Table 2). Blood culture positivity was reported approximately four hours after admission to the cardiac intensive care unit, nearly two hours after the patient's death. Thus, microbiological confirmation was obtained post-mortem, precluding the initiation of pathogen-directed antimicrobial therapy.

Microbiological parameter	Result
Blood culture result	Positive
Identified microorganism	*Streptococcus pneumoniae*
Reported time to positivity	Approximately four hours after admission to the ICU
Antibiotic sensitivity	Sensitive to all tested antibiotics

TABLE 2: Blood culture findings and microbiological identification of Streptococcus pneumoniae.

Blood cultures identified *S. pneumoniae* approximately four hours after the patient's admission to the cardiac intensive care unit, following the patient's death.

Antimicrobial susceptibility testing demonstrated that the isolated *S. pneumoniae* strain was susceptible to all antibiotics tested, with low minimum inhibitory concentrations (MICs), particularly for penicillin G, amoxicillin, cefotaxime, and ceftriaxone (Table 3). Blood cultures were reported positive approximately four hours after admission to the cardiac intensive care unit, nearly two hours after the patient's death. Microbiological confirmation was therefore obtained post-mortem, precluding the initiation of pathogen-directed antimicrobial therapy.

Antibiotic	MIC (mg/L)	Interpretation
Penicillin G	0.016	Sensitive (S)
Ampicillin	0.032	Sensitive (S)
Amoxicillin	0.032	Sensitive (S)
Cefotaxime	0.032	Sensitive (S)
Ceftriaxone	0.032	Sensitive (S)
Vancomycin	0.25	Sensitive (S)
Erythromycin	0.064	Sensitive (S)
Clindamycin	0.064	Sensitive (S)
Levofloxacin	0.5	Sensitive (S)

TABLE 3: Antimicrobial susceptibility profile of Streptococcus pneumoniae.

Minimum inhibitory concentrations (MICs) are presented in mg/L; S denotes sensitivity. Interpretations are reproduced from the reported laboratory findings.

No primary infectious focus was identified. The patient reported no respiratory, urinary, gastrointestinal, or cutaneous symptoms suggestive of a specific portal of entry. The admission chest radiograph showed no evidence of pulmonary infection, and urine culture was negative. Although pneumococcal IE may be associated with respiratory tract infections, invasive pneumococcal infection can occur in the absence of clinically apparent pneumonia. Further investigations to identify the primary infectious source could not be

completed because of the rapidly fatal clinical course.

This exceptionally fulminant presentation highlights the destructive potential of pneumococcal IE, characterized by extensive aortic root abscess formation, multivalvular involvement, suspected infectious extension toward the interatrial septum, and complete atrioventricular block. The rapid progression from an initially preserved blood pressure to profound circulatory collapse and refractory cardiac arrest, despite preserved left ventricular systolic function, illustrates the severity of the disease. Although acute severe aortic regurgitation, extensive periannular infection, and complete atrioventricular block may have contributed to the sudden deterioration, the precise mechanism of cardiovascular collapse could not be definitively established.

The chronological sequence of clinical presentation, diagnostic investigations, therapeutic decisions, and subsequent clinical deterioration is summarized in Table *4*.

Timing	Clinical events
Two weeks before hospital admission	Self-reported febrile illness (38°C).
Two days before hospital admission	Onset of profound asthenia.
Hospital admission	Fever (38°C), blood pressure 110/80 mmHg, complete atrioventricular block with a ventricular escape rhythm of 40 bpm.
Initial diagnostic assessment	Urgent TTE and TEE revealing extensive aortic and mitral valve involvement, severe aortic regurgitation, and a large aortic root abscess extending into the interatrial septum. LVEF 62%.
Initial management	Blood cultures collected and empirical intravenous antibiotic therapy initiated.
T0 - Cardiac ICU admission	Initially preserved blood pressure with clinical signs of peripheral hypoperfusion. Emergency cardiac surgery and intraoperative epicardial pacing planned.
T+1 hour	Abrupt profound hypotension (50/30 mmHg), followed almost immediately by cardiac arrest. Cardiopulmonary resuscitation initiated.
T+2 hours	Death despite advanced resuscitative efforts.
T+4 hours	Blood cultures reported positive for *Streptococcus pneumoniae*, approximately two hours after death.

TABLE 4: Clinical timeline of fulminant pneumococcal infective endocarditis: from initial presentation to fatal cardiovascular collapse.

This table summarizes the chronological sequence of clinical manifestations, diagnostic findings, therapeutic decisions, and subsequent deterioration. Despite initially preserved blood pressure, the patient developed abrupt circulatory collapse followed by cardiac arrest with documented asystole approximately one hour after admission to the cardiac intensive care unit. Death was pronounced approximately two hours after admission despite advanced resuscitative efforts. Blood cultures were reported positive approximately four hours after admission, nearly two hours after death, with microbiological confirmation becoming available only post-mortem.

TTE: transthoracic echocardiography; TEE: transesophageal echocardiography; ICU: intensive care unit; LVEF: left ventricular ejection fraction

Discussion

Pneumococcal IE is a rare but highly aggressive infection characterized by rapid valvular destruction, extensive intracardiac complications, and substantial mortality [6,7]. In a systematic review of 111 cases, native valves were involved in 93.7% of patients, predominantly the aortic valve (53.2%), while pre-existing cardiac disease was identified in only 16.2%. Pneumonia was present in 45.9% of cases, indicating that pneumococcal IE may occur without clinically apparent pulmonary infection [6]. Our case illustrates this uncommon presentation in a previously healthy young woman without an identifiable primary infectious focus.

Periannular extension represents one of the most severe complications of IE and may lead to abscess formation, extensive valvular destruction, and life-threatening conduction abnormalities [3,4]. Complete atrioventricular block is an important warning sign of invasive aortic root infection because of the close anatomical relationship between the aortic annulus and the atrioventricular conduction system. According to the 2023 ESC guidelines, third-degree atrioventricular block is observed in approximately 2.8% of patients with IE at diagnosis [3]. In our patient, complete atrioventricular block was associated with a large aortic

root abscess extending toward the interatrial septum, suggesting advanced periannular infection involving tissues adjacent to the cardiac conduction system. However, direct involvement of the conduction system could not be anatomically confirmed.

Early echocardiographic assessment is essential for identifying destructive complications, defining their anatomical extent, and guiding surgical management. TEE is particularly valuable for detecting periannular abscesses, evaluating multivalvular involvement, and identifying associated intracardiac complications [3,5]. In our case, urgent transthoracic echocardiography and TEE demonstrated extensive aortic valve destruction, severe aortic regurgitation, concomitant mitral valve involvement, and a large aortic root abscess extending toward the interatrial septum. An additional mobile vegetation was identified within the right atrium. Marked interatrial septal thickening suggested possible infectious infiltration, although definitive septal involvement could not be established. Despite complete atrioventricular block with a ventricular escape rhythm of approximately 40 beats/min, the patient remained conscious and initially normotensive. Nevertheless, cold extremities and a prolonged capillary refill time of four seconds indicated peripheral hypoperfusion. TEE was performed without intravenous sedation under continuous monitoring, with emergency pacing equipment immediately available.

The management of complicated pneumococcal IE requires prompt blood culture collection, immediate empirical intravenous antimicrobial therapy, continuous cardiac monitoring, and early multidisciplinary surgical assessment [3]. According to the 2023 ESC guidelines, treatment of IE caused by penicillin-susceptible *S. pneumoniae* generally follows the recommendations for susceptible streptococcal endocarditis, using intravenous penicillin G, amoxicillin, or ceftriaxone. A four-week treatment course is generally appropriate for native-valve infection, whereas shortened two-week regimens have not been adequately evaluated for pneumococcal IE. In cases of reduced penicillin susceptibility, antimicrobial selection should be guided by MICs. When meningitis is present, ceftriaxone or cefotaxime, with or without vancomycin according to the susceptibility profile, is preferred because of the need for adequate cerebrospinal fluid penetration [3].

In our patient, empirical intravenous ampicillin (12 g/day), cloxacillin (12 g/day), and gentamicin (3 mg/kg/day) were initiated after blood culture collection. Subsequent antimicrobial susceptibility testing demonstrated low MICs for penicillin G (0.016 mg/L), amoxicillin (0.032 mg/L), and cefotaxime (0.032 mg/L), consistent with a penicillin-susceptible isolate (Table *3*). These findings would have supported targeted intravenous beta-lactam therapy. However, blood cultures were reported positive approximately four hours after admission to the cardiac intensive care unit, nearly two hours after the patient's death. Microbiological confirmation was therefore obtained only post-mortem, precluding pathogen-directed antimicrobial treatment.

Antimicrobial therapy alone cannot reverse the mechanical consequences of extensive valvular destruction or eliminate the need for surgery in advanced periannular infection [3,4]. The 2023 ESC guidelines identify periannular abscess formation and new atrioventricular conduction abnormalities as indications for urgent surgical management, generally within three to five days. Emergency surgery within 24 hours is recommended when severe valvular dysfunction results in cardiogenic shock or refractory pulmonary edema [3]. In our patient, extensive aortic root destruction, severe aortic regurgitation, complete atrioventricular block, and clinical evidence of impaired peripheral perfusion prompted the decision to pursue emergency surgical management, given the perceived risk of imminent cardiovascular deterioration.

The management of complete atrioventricular block was further complicated by the presence of a mobile right atrial vegetation. Transvenous pacing lead manipulation was considered to carry a potential risk of vegetation disruption and septic pulmonary embolization. In view of the planned emergency cardiac surgery, intraoperative epicardial pacing was selected as an individualized strategy to avoid transvenous instrumentation. This approach was consistent with the 2023 ESC recommendation to consider immediate epicardial pacemaker implantation in patients undergoing surgery for valvular IE who have complete atrioventricular block and predictors of persistent conduction abnormalities, including aortic root abscess [3]. Nevertheless, the potential embolic risk of transvenous pacing in this particular setting remains uncertain, and temporary transvenous pacing may still be necessary when life-threatening bradycardia requires immediate treatment and alternative pacing approaches are not readily available. Unfortunately, the patient developed abrupt cardiovascular collapse and documented asystole before emergency surgery or intraoperative epicardial pacing could be performed.

An important feature of this case was the preservation of left ventricular systolic function despite extensive valvular destruction and rapid clinical deterioration. The estimated left ventricular ejection fraction remained 62%, illustrating that preserved systolic function does not necessarily indicate hemodynamic stability in acute severe aortic regurgitation. Acute regurgitant lesions may substantially reduce effective forward cardiac output before overt left ventricular systolic dysfunction becomes apparent [8].

The precise mechanism of sudden cardiovascular collapse remains uncertain and may have been multifactorial. Acute severe aortic regurgitation may have contributed to hemodynamic deterioration through increased left ventricular filling pressures and reduced effective forward cardiac output. The

absence of left ventricular dilatation, with an end-diastolic diameter of 53 mm (indexed: 25 mm/m^2) and an end-systolic diameter of 35 mm (indexed: 16 mm/m^2), together with a non-widened pulse pressure of 30 mmHg, was compatible with an acute presentation of severe aortic regurgitation [8]. Furthermore, progressive periannular infection involving tissues adjacent to the atrioventricular conduction system may have compromised the ventricular escape rhythm and contributed to the development of asystole. Infection-related circulatory dysfunction may also have played a role. Additional mechanical complications, including abscess rupture or fistulization, cannot be completely excluded. However, no repeat echocardiographic examination was performed during the cardiovascular collapse, and neither surgical exploration nor postmortem examination was available. Consequently, these proposed mechanisms remain hypothetical, and the exact cause of cardiac arrest could not be definitively established.

The prognosis of pneumococcal IE remains unfavorable, particularly in patients with extensive intracardiac complications. De Egea et al. reported an in-hospital mortality rate of 20.7%, with cardiac surgery associated with improved survival [6]. Similarly, a French multicenter study reported substantial early mortality and an association between surgical intervention and better outcomes [7]. Although these observational findings cannot establish a causal survival benefit for every individual patient, they emphasize the importance of early recognition, prompt surgical evaluation, and coordinated multidisciplinary management. The exceptionally rapid deterioration observed in our patient, from initially preserved blood pressure with signs of peripheral hypoperfusion to fatal cardiovascular collapse within hours, illustrates the narrow therapeutic window associated with fulminant pneumococcal IE.

Previous case reports have described severe conduction disturbances and destructive periannular complications associated with pneumococcal IE. Watanabe et al. reported a 62-year-old woman with pneumococcal aortic valve endocarditis presenting with complete atrioventricular block who survived following pacemaker implantation and aortic valve replacement [9]. Another report described pneumococcal endocarditis in a patient with previous cardiac surgery, illustrating the potential for severe intracardiac complications in this infection [10]. In contrast, our patient was a previously healthy 25-year-old woman presenting with complete atrioventricular block, extensive native-valve destruction, and a large aortic root abscess, followed by fatal cardiovascular collapse within hours. These comparisons emphasize the unusually aggressive course of the present case.

Beyond acute management, pneumococcal vaccination remains an important preventive strategy against invasive pneumococcal disease. Pneumococcal conjugate vaccines reduce vaccine-type invasive infections, including bacteremia, and may therefore indirectly reduce the occurrence of rare complications such as pneumococcal endocarditis. However, a specific protective effect against pneumococcal IE has not been established [11,12]. In our patient, pneumococcal vaccination status was unknown, and no causal relationship between vaccination history and the development of endocarditis can be inferred. Nevertheless, this case highlights the broader public health importance of pneumococcal vaccination according to established recommendations, particularly among individuals at increased risk of invasive pneumococcal disease [11,12].

Conclusions

This case highlights the devastating potential of pneumococcal IE, which may cause extensive intracardiac destruction and fatal cardiovascular collapse even in previously healthy young adults. Complete atrioventricular block should be recognized as a critical warning sign of invasive periannular infection, particularly in the setting of aortic valve involvement. Importantly, preserved left ventricular systolic function and initially preserved blood pressure should not be mistaken for a favorable prognosis when severe acute valvular dysfunction and extensive aortic root destruction coexist. Although acute severe aortic regurgitation and extensive periannular infection may have contributed to the sudden cardiovascular deterioration, the precise mechanism of collapse could not be definitively established. Early TEE, immediate readiness for cardiac pacing, prompt antimicrobial therapy, and expedited surgical evaluation are essential in managing this rapidly evolving condition. Beyond acute care, this case reinforces the importance of pneumococcal vaccination as a preventive strategy against invasive pneumococcal disease. Ultimately, in fulminant pneumococcal endocarditis, the window between initially preserved blood pressure and irreversible cardiovascular collapse may be alarmingly narrow.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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