

Actinotignum-associated prosthetic aortic valve infective endocarditis complicated by postoperative mixed shock and dialysis-dependent acute kidney injury: a case report

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Case Report

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Abstract

Background:

Actinotignum spp. are facultatively anaerobic Gram-positive rods increasingly recognized as urogenital pathogens, particularly in older or urologically compromised patients. Clinically significant infections may be underrecognized because these organisms grow slowly and may require anaerobic or CO₂-enriched culture conditions. Infective endocarditis caused by *Actinotignum* spp. is uncommon, and prosthetic valve involvement appears especially rare. Species-level identification within this genus may remain challenging without molecular confirmation.

Case presentation:

A 71-year-old man with benign prostatic hyperplasia, recurrent urinary retention requiring repeated clean intermittent self-catheterization, horseshoe kidney, chronic kidney disease, and bioprosthetic aortic valve replacement 7 months earlier presented with low back pain, rapid weight gain, oliguria, dark-colored urine, severe acute kidney injury, hyperkalemia, and systemic inflammation. Initial evaluation suggested urinary tract infection complicated by acute kidney injury, and intermittent hemodialysis was started. Transthoracic echocardiography revealed a large mobile vegetation around the bioprosthetic aortic valve, and emergency surgery was performed before microbiological confirmation. Extensive vegetations and abnormal granulation tissue were debrided, whereas redo aortic valve replacement was avoided because valve structure and sewing ring stability were preserved, and surrounding tissues were severely friable. Blood and urine cultures later yielded *Actinotignum* spp.; the best matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) match was *Actinotignum sanguinis*, although species-level identification was not confirmed molecularly. Urine culture required anaerobic culture on Brucella HK agar for detection. The postoperative course was complicated by mixed shock with low-output physiology, vasoplegia, anuria, fluid overload, hypoalbuminemia, and reduced effective intravascular volume. Integrated antimicrobial, circulatory, fluid, and renal replacement management led to vegetation disappearance, preserved prosthetic valve function, hemodialysis discontinuation, and functional recovery.

Conclusions:

Actinotignum spp. should be considered in urologically compromised patients with bacteremia or infective endocarditis, particularly when prosthetic material is present. Discordance between Gram staining and routine culture should prompt anaerobic culture and extended incubation. MALDI-TOF MS is useful, but species-level interpretation requires caution without molecular confirmation. In prosthetic valve endocarditis, source control may need to proceed before microbiological confirmation, and postoperative deterioration may require management as mixed shock rather than isolated septic shock.

Background

The genus *Actinotignum* comprises facultatively anaerobic Gram-positive rods increasingly recognized as urogenital pathogens. *Actinotignum schaalii* has been most frequently reported in human infections, particularly urinary tract infections in older or urologically compromised patients, whereas *A. sanguinis* and other species remain less well characterized [1]. These organisms grow slowly and require enriched media with prolonged incubation under anaerobic or CO₂-enriched conditions, and they are difficult to identify by conventional phenotypic methods [1, 2]. As a result, clinically significant infections may be underrecognized. Although matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) has improved genus-level recognition, species-level discrimination within *Actinotignum*, particularly between *A. schaalii* and *A. sanguinis*, may remain challenging without molecular confirmation [3].

Although most commonly associated with the urinary tract, *Actinotignum* spp. can cause invasive infections, including bacteremia, spondylodiscitis, abscesses, and infective endocarditis [1, 4]. Endocarditis caused by *Actinotignum* spp. is uncommon, and prosthetic valve involvement appears especially rare. A recent report of subacute prosthetic aortic endocarditis due to *A. schaalii*, complicated by delayed recognition, recurrent embolic events, extensive periprosthetic destruction, and death, underscores the potential severity of cardiovascular infection involving prosthetic material [5].

We describe a case of *Actinotignum*-associated prosthetic aortic valve infective endocarditis in a 71-year-old man with a complex urogenital and renal history, including benign prostatic hyperplasia, recurrent urinary retention, repeated clean intermittent self-catheterization, horseshoe kidney, and chronic kidney disease. The best MALDI-TOF MS match from both blood and urine cultures was *A. sanguinis*, but species-level identification was not confirmed molecularly; the infection is therefore described as *Actinotignum*-associated. This case illustrates delayed recognition requiring anaerobic culture, echocardiography-driven source control before microbiological confirmation, risk-adapted valve-preserving debridement, and recovery from postoperative mixed shock with temporary hemodialysis dependence.

Case Presentation

A 71-year-old man presented with a 1-week history of low back pain, rapid weight gain, bilateral lower extremity edema, decreased urine output, and dark-colored urine. Seven months before admission, he had undergone bioprosthetic aortic valve replacement using a 21-mm INSPIRIS valve (Edwards Lifesciences, Irvine, CA, USA), with concomitant mitral valve suture annuloplasty, for aortic stenosis. His medical history included hyperlipidemia, horseshoe kidney, chronic kidney disease, and benign prostatic hyperplasia. During the preceding year, recurrent urinary retention had required repeated clean intermittent self-catheterization, and renal function had declined from a serum creatinine of 0.95 mg/dL 2 years before admission to 1.30 mg/dL at the preoperative evaluation for valve surgery.

On admission (Day 1), he was alert and hemodynamically stable. Laboratory tests showed severe acute kidney injury, hyperkalemia, and systemic inflammation: serum creatinine 10.68 mg/dL, blood urea nitrogen 137.4 mg/dL, potassium 7.1 mEq/L, and C-reactive protein 17.05 mg/dL. Urinalysis after urethral catheterization revealed gross hematuria, proteinuria, occult blood, and bacteriuria. A urinary tract source of systemic infection complicated by severe acute kidney injury was suspected. Two sets of blood cultures and a urine culture were obtained before antimicrobial therapy, and intravenous

ceftriaxone 1 g/day was started. He was admitted to the high-care unit under nephrology care. On Day 2, worsening renal dysfunction and persistent hyperkalemia required urgent intermittent hemodialysis.

Magnetic resonance imaging of the spine raised concern for pyogenic spondylitis and spondylodiscitis as a possible infectious focus, but surgical intervention was not indicated. Linezolid 1200 mg/day was added on Day 3. That night, high-grade fever prompted further systemic evaluation. On Day 4, transthoracic echocardiography revealed a mobile echogenic mass measuring approximately 20 × 9 mm around the bioprosthetic aortic valve, adjacent to the right coronary cusp, with increased transprosthetic aortic valve gradients but without prosthetic valve dehiscence or paravalvular regurgitation (Fig. 1). Left ventricular ejection fraction was 51%. Although no causative organism had yet been identified, prosthetic valve infective endocarditis was diagnosed on clinical and echocardiographic grounds, and emergency surgical source control was judged necessary because of the large mobile vegetation and embolic risk. The patient was transferred to the intensive care unit, and emergency surgery was started at 22:00 on Day 4.

The planned surgical strategy included redo aortic valve replacement if prosthetic valve destruction, dehiscence, or sewing ring instability was present. Intraoperatively, vegetations were found on the leaflets of the bioprosthetic valve, and a large pedunculated vegetation was identified on the subvalvular aspect of the prosthesis, accompanied by abnormal granulation tissue. However, the prosthetic valve structure was preserved, with no leaflet destruction, sewing ring dehiscence, or suture disruption. Because the surrounding tissue was markedly indurated and friable, redo aortic valve replacement was considered to require extensive aortic root reconstruction with prohibitive operative risk. The prosthetic valve was therefore preserved, with debridement of all accessible vegetations and abnormal granulation tissue followed by copious irrigation with normal saline. During difficult dissection around the aortic root, the right coronary ostium was injured, requiring coronary artery bypass grafting with a saphenous vein graft to the proximal right coronary artery. Cardiopulmonary bypass time was 335 min, and aortic cross-clamp time was 315 min. Surgery was completed on the morning of Day 5, and the patient was transferred directly to the intensive care unit.

Intraoperative specimens were obtained from the perivalvular region, subvalvular tissue, and excised vegetations. Subsequent smear microscopy and aerobic and anaerobic cultures yielded no organisms. These negative results were interpreted cautiously because the specimens had been obtained after several days of antimicrobial therapy. A cream-colored turbid subcutaneous material encountered at sternotomy was also culture-negative.

On Day 5, blood cultures obtained on Day 1 were reported as positive. MALDI-TOF MS using a Bruker MALDI Biotyper system yielded *Actinotignum sanguinis* as the best match in two blood culture isolates, with score values of 2.02 and 2.10. On Day 6, the urine culture obtained on Day 1 was also reported positive; the best MALDI-TOF MS match was likewise *A. sanguinis*, with a score value of 2.31. Additional organisms identified from the urine culture included *Veillonella parvula* and *Peptoniphilus* sp. In the urine specimen, Gram staining had demonstrated Gram-positive and Gram-negative rods, whereas routine culture conditions without dedicated anaerobic incubation did not initially yield growth. Subsequent anaerobic culture led to detection of *Actinotignum* spp. Because 16S rRNA gene sequencing was not performed and *A. schaalii* appeared among lower-ranking MALDI-TOF MS matches, the organism was interpreted as *Actinotignum* spp., with *A. sanguinis* as the best MALDI-TOF MS match. Ceftriaxone and linezolid were continued initially because available minimum inhibitory concentration data suggested in vitro activity, although formal susceptibility category interpretation was limited.

Continuous hemodiafiltration was started on Day 5 for postoperative renal replacement therapy, but repeated circuit clotting prevented adequate solute clearance and ultrafiltration. The patient remained nearly anuric, and body weight increased rapidly from 66.2 kg on Day 5 to more than 80 kg by Day 10. On Day 8, he developed postoperative mixed shock requiring escalation of norepinephrine, dobutamine, epinephrine, and vasopressin (Fig. 4). The hemodynamic profile was not consistent with isolated septic shock, but rather with the coexistence of low-output physiology, vasoplegia, anuria, marked fluid overload, and reduced effective intravascular volume. Bedside assessment showed a central venous pressure of 5–8 mmHg on Day 8, an inferior vena cava diameter of approximately 10 mm, and mild intracardiac underfilling despite gross systemic fluid accumulation. Serum albumin was 1.8 g/dL. Intravenous 25% albumin was administered from Day 8 through Day 10 while vasoactive support was continued. Blood lactate peaked at 6.0 mmol/L on Day 8 and decreased to 3.3 mmol/L on Day 10.

On Day 9, antimicrobial therapy was changed from ceftriaxone and linezolid to ampicillin 6 g/day, based on the microbiological results and infectious disease consultation. The subsequent improvement was considered to reflect the combined effects of surgical source control, optimization of effective circulating volume, stabilization of renal replacement therapy, vasoactive support, and targeted antimicrobial therapy, rather than the effect of any single intervention. By Day 10, continuous hemodiafiltration circuit patency had stabilized after a change in anticoagulation strategy, allowing continuous operation.

From Day 11 onward, hemodynamics progressively improved, and net ultrafiltration became feasible. Body weight, which peaked around Day 13, began to decrease thereafter. Vasopressin was discontinued on Day 14. On Day 15, continuous hemodiafiltration was discontinued and renal replacement therapy was transitioned to intermittent hemodialysis combined with isolated ultrafiltration, allowing more efficient fluid management. The overall course of antimicrobial therapy, respiratory support, vasoactive support, renal replacement therapy, and fluid management is summarized in Figs. 3 and 4.

On Day 16, fever, tachypnea, leukocytosis, and increased purulent sputum raised suspicion for ventilator-associated pneumonia. Ampicillin was discontinued on Day 17, and piperacillin-tazobactam was started on Day 18; micafungin was added empirically because of the prolonged critical illness and broad-spectrum antimicrobial exposure. Sputum culture yielded *Pseudomonas aeruginosa*, whereas blood cultures were negative. All vasoactive agents were discontinued by Day 20. The patient was extubated on Day 21, discharged from the intensive care unit to the general ward on Day 22, and weaned from supplemental oxygen on Day 25.

Follow-up transthoracic echocardiography on Day 27 showed disappearance of the previously observed vegetation, preserved bioprosthetic valve function, no aortic or paravalvular regurgitation, no prosthetic dehiscence, and improved transprosthetic gradients (Fig. 2). Left ventricular ejection fraction was 50.7%, with improved wall motion. A repeat sputum culture reported on Day 27 identified piperacillin-resistant *P. aeruginosa*, and antimicrobial therapy was changed

to meropenem on Day 28, with dosing adjusted for dialysis-dependent renal function. A right pleural effusion required pleural drainage, which was removed on Day 33. Rehabilitation had been initiated during the intensive care unit stay, and by Day 28 the patient was able to participate in standing and gait training.

Spontaneous urine output was first observed around Day 33 and increased gradually thereafter. Meropenem was continued through Day 40, at which point all antimicrobial therapy was completed. Intermittent hemodialysis was continued approximately three times per week while native renal function was monitored. The final intermittent hemodialysis session was performed on Day 50. On Day 54, the nephrology team determined that hemodialysis could be discontinued, based on recovery of urine output and renal function. Urine output was 1373 mL/day on Day 54 and increased to 1622 mL/day by Day 57. On Day 60, the patient had achieved substantial functional recovery without apparent neurological or physical sequelae attributable to the critical illness and was transferred to a rehabilitation ward. He was subsequently discharged home from the rehabilitation ward, walking independently.

Patient perspective

During the consent interview for publication, the patient and his wife expressed appreciation for his recovery and wished to proceed with further treatment for urinary retention and benign prostatic hyperplasia after stabilization. Urological follow-up was arranged accordingly.

Discussion and Conclusions

This case highlights several educational points in the diagnosis and management of *Actinotignum*-associated prosthetic valve infective endocarditis (PVE). The patient had a clear urogenital background—benign prostatic hyperplasia, recurrent urinary retention, repeated clean intermittent self-catheterization, horseshoe kidney, and chronic kidney disease—and *Actinotignum* spp. was detected from both blood and urine cultures, supporting a urogenital portal of entry. Surgical source control was undertaken on the basis of echocardiographic findings before microbiological confirmation was available. Despite postoperative mixed shock and dialysis-dependent acute kidney injury, infection control, prosthetic valve preservation, hemodialysis discontinuation, and substantial functional recovery were ultimately achieved. We consider this case instructive primarily as an example of a potentially underrecognized urogenital pathogen capable of causing serious prosthetic material infection.

Reported cases of *Actinotignum*-associated infective endocarditis remain limited, and previously published cases are summarized in Table 1 [5–9]. These reports include both native and prosthetic valve infections, variable evidence of urinary tract involvement, different microbiological approaches, and heterogeneous outcomes. The recent report by Rallet et al. is particularly relevant, describing prosthetic aortic endocarditis due to *Actinotignum schaalii* complicated by delayed recognition, recurrent embolic events, extensive periprosthetic destruction, and death [5]. Other cases illustrate important diagnostic patterns, including coryneform Gram-positive rods requiring molecular identification, endocarditis following urological procedures, and urinary isolates detected only after anaerobic reinoculation [6, 7]. Compared with these reports, the present case is notable for concordant blood and urine culture findings, early echocardiographic recognition, individualized surgical source control, and recovery after severe postoperative mixed shock and temporary hemodialysis dependence. These features add to the limited literature without implying that optimal management can be inferred from a single case.

Table 1

Previously reported cases of *Actinobaculum*/*Actinotignum*-associated infective endocarditis and the present case

Reference/year	Age/sex	Relevant background	Urogenital intervention/source	Valve involved	Major complications	Microbiological confirmation and diagnostic issue	Treatment
Hoenigl et al. / 2010	52/M	Mechanical AVR 2 years earlier; prior MCA stroke; gastric-duodenal angiodysplasia	None; source not identified	Prosthetic mechanical aortic valve	Prosthetic valve vegetation; fever, Janeway lesions, embolic stroke	Blood cultures positive; coryneform Gram-positive rods with difficult initial recovery/identification; confirmed by 16S rRNA; urine culture negative	No surgery; piperacillin-tazobactam followed by amoxicillin-clavulanate
Diallo et al. / 2018	63/M	BPH; recent transurethral resection of the prostate	Yes; transurethral resection of the prostate; presumed urogenital source	Native aortic and tricuspid valves	Large tricuspid vegetation; severe aortic regurgitation, acute heart failure	Blood culture identified <i>A. schaalii</i> ; stored urine positive only after anaerobic reinoculation; valve 16S rRNA PCR positive despite negative valve cultures	Aortic and tricuspid valve replacement; amoxicillin plus gentamicin
Loiez et al. / 2018	89/M	Prostatism; no urinary symptoms	None; urogenital source suspected but not microbiologically confirmed	Native aortic valve	Large aortic vegetation; heart failure; postoperative diaphragmatic paralysis requiring ventilatory assistance	Blood cultures and valve culture positive; identification by MALDI-TOF MS and 16S rDNA sequencing; coryneform Gram-positive rods initially noted	Biological AVR; amoxicillin-based therapy with gentamicin
Premkumar et al. / 2022	86/F	Advanced dementia/frailty	None; urinary source possible but not clearly established	Native mitral valve	Mitral vegetation; peripheral IE stigmata; fatal nosocomial pneumonia during treatment	Blood and urine cultures positive for <i>A. schaalii</i> ; limited microbiological details reported	No surgery; culture-directed antibiotic therapy including ceftriaxone
Rallet et al. / 2025	54/M	Prior Bentall procedure with mechanical aortic valve and vascular prosthesis; prior reintervention for coronary ostial pseudoaneurysm; recent tooth extraction	None; source not identified; urine cultures sterile	Prosthetic aortic valve / Bentall conduit	Recurrent embolic strokes; perivalvular abscess, fistulization, pseudoaneurysm, complete conduit dehiscence, aorto-mitral trigone destruction	Blood cultures positive after prolonged incubation; Gram-positive bacilli initially misidentified as <i>Corynebacterium</i> spp.; later confirmed as <i>A. schaalii</i> ; PET/CT and cardiac CT were diagnostically important	Amoxicillin-based therapy; redo surgery attempt but reconstruction deemed uneas
Present case / 2026	71/M	BPH, horseshoe kidney, CKD, hyperlipidemia, bioprosthetic AVR 7 months earlier	Yes; repeated clean intermittent self-catheterization; presumed urogenital source	Prosthetic bioprosthetic aortic valve	Large vegetation, subvalvular vegetation, abnormal granulation tissue; severe AKI, hyperkalemia, postoperative mixed shock	Blood and urine cultures positive; best MALDI-TOF MS match was <i>A. sanguinis</i> ; routine culture conditions without dedicated anaerobic incubation initially yielded no growth despite organisms on Gram stain; anaerobic culture positive; no 16S rRNA sequencing	Valve-preserving debridement and SVG-RCA CABG; ceftriaxone/linezolid → ampicillin; later broad-spectrum therapy for postoperative <i>P. aeruginosa</i> infection
Abbreviations: AKI, acute kidney injury; AVR, aortic valve replacement; BPH, benign prostatic hyperplasia; CABG, coronary artery bypass grafting; CKD, chronic kidney disease; IE, infective endocarditis; MALDI-TOF MS, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry; MCA, middle cerebral artery; PCR, polymerase chain reaction; PET/CT, positron emission tomography/computed tomography; SVG, saphenous vein graft.							
For the present case, species-level identification was not confirmed by 16S rRNA gene sequencing; therefore, the organism is described as <i>Actinotignum</i> sp. with <i>Actinotignum sanguinis</i> reported as the best MALDI-TOF MS match.							
The antimicrobial duration in the present case indicates the total systemic antimicrobial course; later broad-spectrum therapy was administered for postoperative <i>P. aeruginosa</i> infection.							

A central diagnostic challenge in this case was the fastidious nature of *Actinotignum* spp. These organisms are increasingly recognized as urogenital pathogens, particularly in older patients with urological abnormalities or instrumentation, but may be overlooked because of slow growth, fastidious culture requirements, and resemblance to commensal coryneform organisms [1, 2, 4, 10]. In the present case, Gram-positive and Gram-negative rods were visible on urine Gram staining, yet routine culture conditions without dedicated anaerobic incubation yielded no growth; *Actinotignum* spp. was recovered only after anaerobic culture on Brucella HK agar. This sequence is clinically important: urinary tract infection may be suspected early while the causative organism remains unidentified. Similar diagnostic difficulties have been described previously, including delayed blood culture positivity, initial misidentification as

Corynebacterium spp., and culture-negative valve specimens despite molecular confirmation [5–7]. When Gram staining and routine culture results are discordant, particularly in patients with urinary tract abnormalities, extended incubation and anaerobic culture should be considered.

The microbiological interpretation of this case requires caution. MALDI-TOF MS provided concordant *Actinotignum* identification from both blood and urine isolates, with *A. sanguinis* as the best match in all three cultures. However, closely related species, including *A. schaalii*, also appeared among lower-ranking matches, and confirmatory molecular testing such as 16S rRNA gene sequencing was not performed. Recent genomic analyses have shown that clinical isolates assigned to *A. schaalii* or *A. sanguinis* by MALDI-TOF MS may include cryptic species-level diversity, and that current commercial databases may not reliably discriminate species within this genus [3]. We therefore used *A. sanguinis* only to describe the MALDI-TOF MS best match, and *Actinotignum* spp. when discussing pathogenesis and clinical implications. This approach reflects a broader diagnostic principle: MALDI-TOF MS can be highly valuable for genus-level recognition, but it still requires cautious interpretation at the species level in taxonomically complex or underrepresented organisms.

Although the urogenital tract was the most plausible portal of entry, this interpretation requires qualification. The patient had repeated self-catheterization, chronic urinary retention, bacteriuria, and concordant *Actinotignum* spp. detection from both blood and urine cultures, all strongly supporting a urogenital source. However, the urine culture was polymicrobial, with *Veillonella parvula* and *Peptoniphilus* sp. also identified, likely reflecting a chronically altered urogenital milieu related to urinary retention and instrumentation rather than a simple monomicrobial urinary tract infection. Because *Actinotignum* spp. was the sole organism recovered from blood cultures, its concordant detection in both blood and urine most strongly supported *Actinotignum*-associated bacteremia with a urogenital portal of entry. Nevertheless, the precise microbial ecology of the urinary infection and the possible contribution of coexisting anaerobes to local inflammation could not be fully resolved.

The timing and nature of source control also deserve emphasis. In PVE, surgical intervention may be required for large mobile vegetations, embolic risk, uncontrolled infection, or destructive perivalvular involvement, and decision-making often cannot await complete microbiological clarification [11, 12]. In this case, emergency surgery was performed before organism identification because TTE demonstrated a large mobile vegetation on the bioprosthetic aortic valve in a patient with systemic inflammation and a plausible urogenital source. Redo AVR was included in the planned operative strategy; however, intraoperative findings showed preserved prosthetic valve structure, no sewing ring dehiscence, and no suture disruption, whereas the surrounding tissues were markedly indurated and friable. Proceeding with redo valve replacement was considered to require extensive aortic root reconstruction at unacceptably high operative risk. The surgical team therefore selected valve-preserving debridement as a risk-adapted individualized source-control strategy, not as a proposed alternative to standard PVE management. This strategy reflected a balance between the need for surgical source control and the prohibitive risk of extensive redo aortic root reconstruction. The favorable outcome in this patient suggests that this strategy was followed by apparent infection control in this specific context, but it does not establish the general efficacy or safety of valve-preserving debridement for PVE.

The postoperative course illustrates the complexity of shock management in severe prosthetic valve infection. Although infection was central, the shock state was not adequately explained by septic physiology alone. The patient had low cardiac output after prolonged cardiopulmonary bypass, aortic cross-clamping, and complex redo cardiac surgery including SVG–RCA bypass, together with vasoplegia, anuria, severe hypoalbuminemia, and massive fluid accumulation from unstable CHDF. The intraoperative right coronary ostial injury and the subsequent need for bypass grafting prolonged the cardiopulmonary bypass time and may have contributed to additional myocardial ischemia, both of which could have affected postoperative cardiac function. Simultaneously, bedside assessment indicated reduced effective intravascular volume, with low central venous pressure, a small inferior vena cava, and intracardiac underfilling despite gross systemic fluid overload. This constellation is consistent with a mixed cardiogenic–vasodilatory shock phenotype, in which cardiac dysfunction and vasodilatory failure coexist and may produce apparently contradictory signs of congestion and intravascular depletion [13]. Recovery required integration of guideline-recommended principles [14]: albumin administration to restore effective circulating volume, vasoactive support, stabilization and eventual transition from CHDF to intermittent hemodialysis allowing controlled decongestion, and ongoing antimicrobial management. Failure to maintain effective renal replacement therapy amplified both circulatory and renal dysfunction, whereas its stabilization enabled progressive fluid removal, recovery of urine output, and eventual hemodialysis discontinuation [15]. This case underscores that, in severe postoperative infection, fluid overload and effective intravascular hypovolemia may coexist, and management requires repeated hemodynamic reassessment rather than reliance on a single shock category.

This case has several limitations. First, species-level identification was not confirmed by molecular methods; the organism is therefore described as *Actinotignum* spp., with *A. sanguinis* reported only as the best MALDI-TOF MS match. Second, intraoperative cultures were negative, possibly influenced by preceding antimicrobial therapy, and definitive microbiological confirmation from valve tissue was not obtained. Third, although a urogenital portal of entry was strongly supported, the polymicrobial urine culture indicates that the urinary microbial ecology could not be fully resolved. Fourth, it was not possible to quantify how prolonged cardiopulmonary bypass, aortic cross-clamping, right coronary ostial injury, and infection-related vasoplegia each contributed to the postoperative mixed shock state. Finally, the favorable outcome after valve-preserving debridement cannot establish the efficacy or generalizability of this approach for PVE.

In conclusion, *Actinotignum* spp. should be considered in patients with urinary tract abnormalities or instrumentation who develop bacteremia, systemic infection, or infective endocarditis, particularly when prosthetic material is present. Negative or discordant routine urine cultures should not exclude *Actinotignum* infection when organisms are observed on Gram stain; anaerobic culture, extended incubation, and MALDI-TOF MS may be essential for recognition. Species-level identification by MALDI-TOF MS should be interpreted cautiously when molecular confirmation is unavailable. In PVE, source control may need to proceed on clinical and echocardiographic grounds before microbiological confirmation, and surgical strategy must be individualized according to intraoperative findings and risk. Finally, postoperative deterioration in severe prosthetic valve infection may reflect mixed shock rather than isolated septic shock, and recovery may depend on integrated infection control, circulatory support, renal replacement therapy, and careful fluid management.

Abbreviations

ABPC, ampicillin;

AVR, aortic valve replacement;

CHDF, continuous hemodiafiltration;

CTRX, ceftriaxone;

HD, hemodialysis;

ICU, intensive care unit;

LZD, linezolid;

MALDI-TOF MS, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry;

MCFG, micafungin;

MEPM, meropenem;

PCV, pressure-controlled ventilation;

PSV, pressure support ventilation;

PVE, prosthetic valve infective endocarditis;

RCA, right coronary artery;

RRT, renal replacement therapy;

SVG, saphenous vein graft;

TAZ/PIPC, piperacillin-tazobactam;

TTE, transthoracic echocardiography.

Declarations

Ethics approval and consent to participate

This case report was prepared in accordance with the principles of the Declaration of Helsinki. According to the institutional ethics committee policy of Daiyukai General Hospital, ethics approval was not required for this single-patient case report using anonymized clinical information.

Consent for publication

Written informed consent for publication of the clinical details and clinical images was obtained from the patient.

Use of artificial intelligence-assisted tools

Artificial intelligence-assisted tools, including ChatGPT, were used for language editing, manuscript organization, and refinement of English expressions. No artificial intelligence-assisted tool was used to generate or modify clinical data, microbiological data, references, figures, or clinical images. The authors reviewed and verified all content, clinical interpretations, and references, and take full responsibility for the final manuscript.

Availability of data and materials

The data supporting the findings of this case report are included in this article and its supplementary files. Additional data are not publicly available because they contain patient-related clinical information, but may be made available from the corresponding author upon reasonable request and to the extent permitted by the patient's consent and institutional regulations.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

NS was involved in the intensive care management of the patient, collected and organized the clinical data, reviewed the literature, and drafted the manuscript. AS was involved in the intensive care management of the patient, assisted with data organization, and critically reviewed the manuscript. TM was involved in the intensive care management of the patient, supervised the clinical interpretation, and critically reviewed the manuscript. All authors read and approved the final manuscript.

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Figures

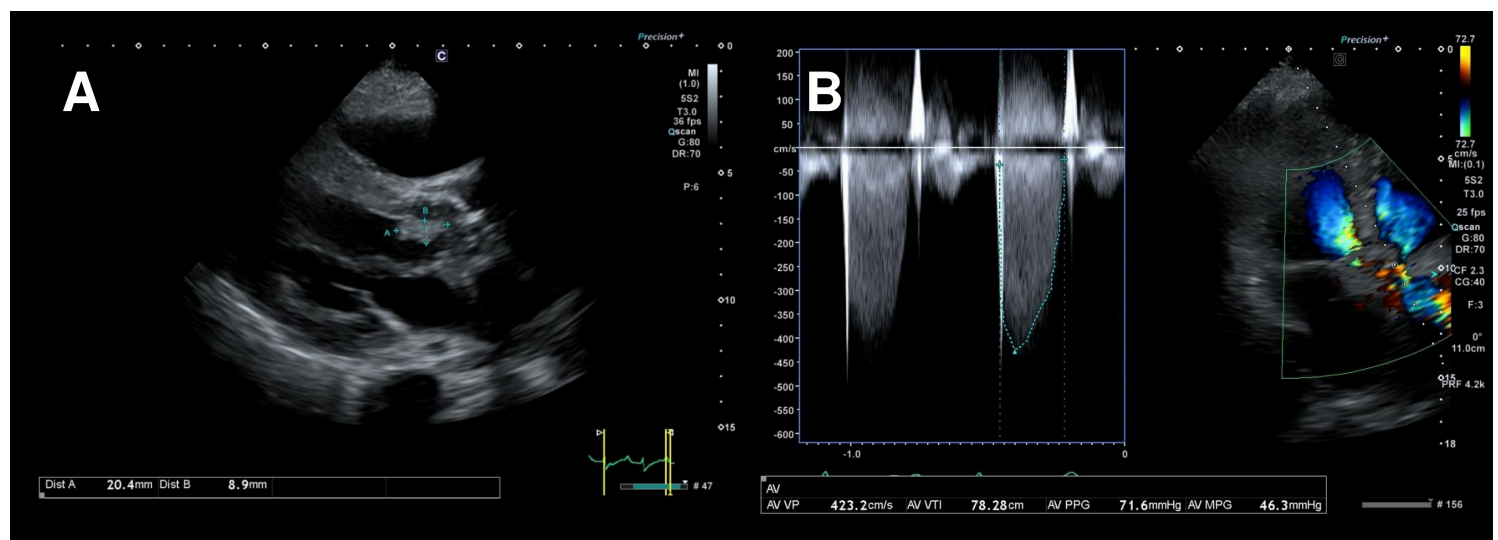


Figure 1

Preoperative transthoracic echocardiography.

(A) Parasternal long-axis transthoracic echocardiographic image showing an echogenic mass around the prosthetic aortic valve, compatible with vegetation, measuring 20.4 × 8.9 mm. (B) Color and spectral Doppler imaging showed an increased peak transprosthetic aortic valve velocity of 4.23 m/s, with peak and mean pressure gradients of 71.6 and 46.3 mmHg, respectively.

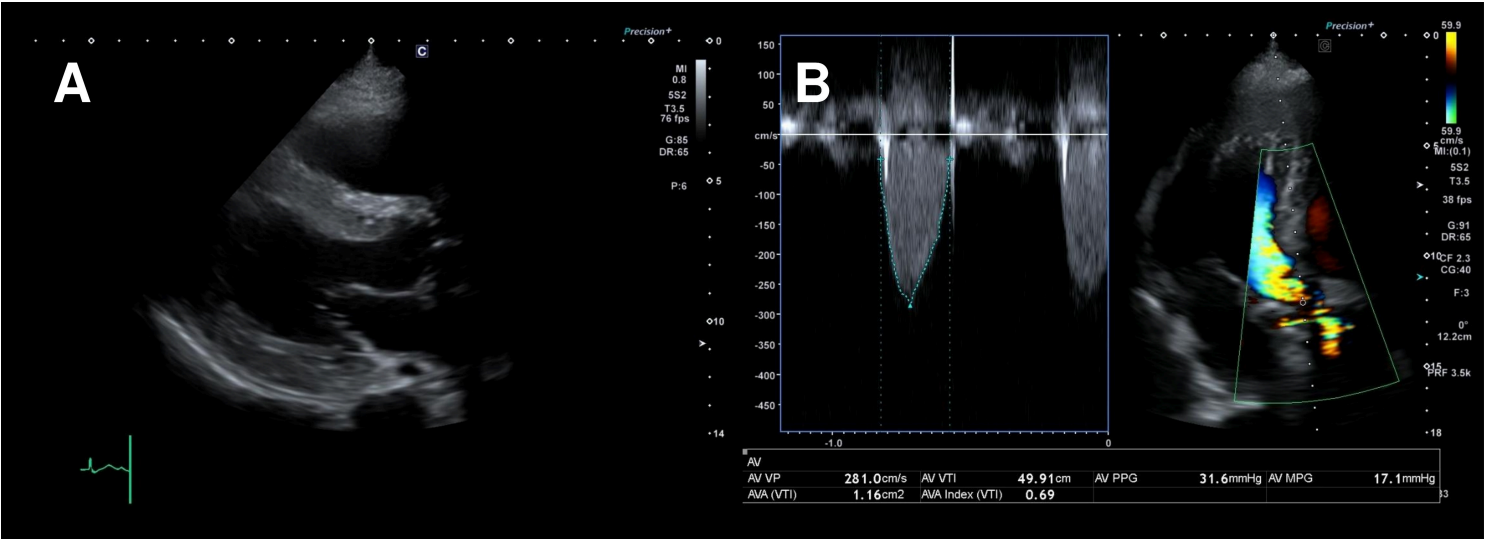


Figure 2

Follow-up transthoracic echocardiography.

(A) Follow-up transthoracic echocardiographic image showing disappearance of the previously observed prosthetic aortic valve vegetation. (B) Color and spectral Doppler imaging showed improvement in the peak transprosthetic aortic valve velocity to 2.81 m/s, with peak and mean pressure gradients of 31.6 and 17.1 mmHg, respectively.

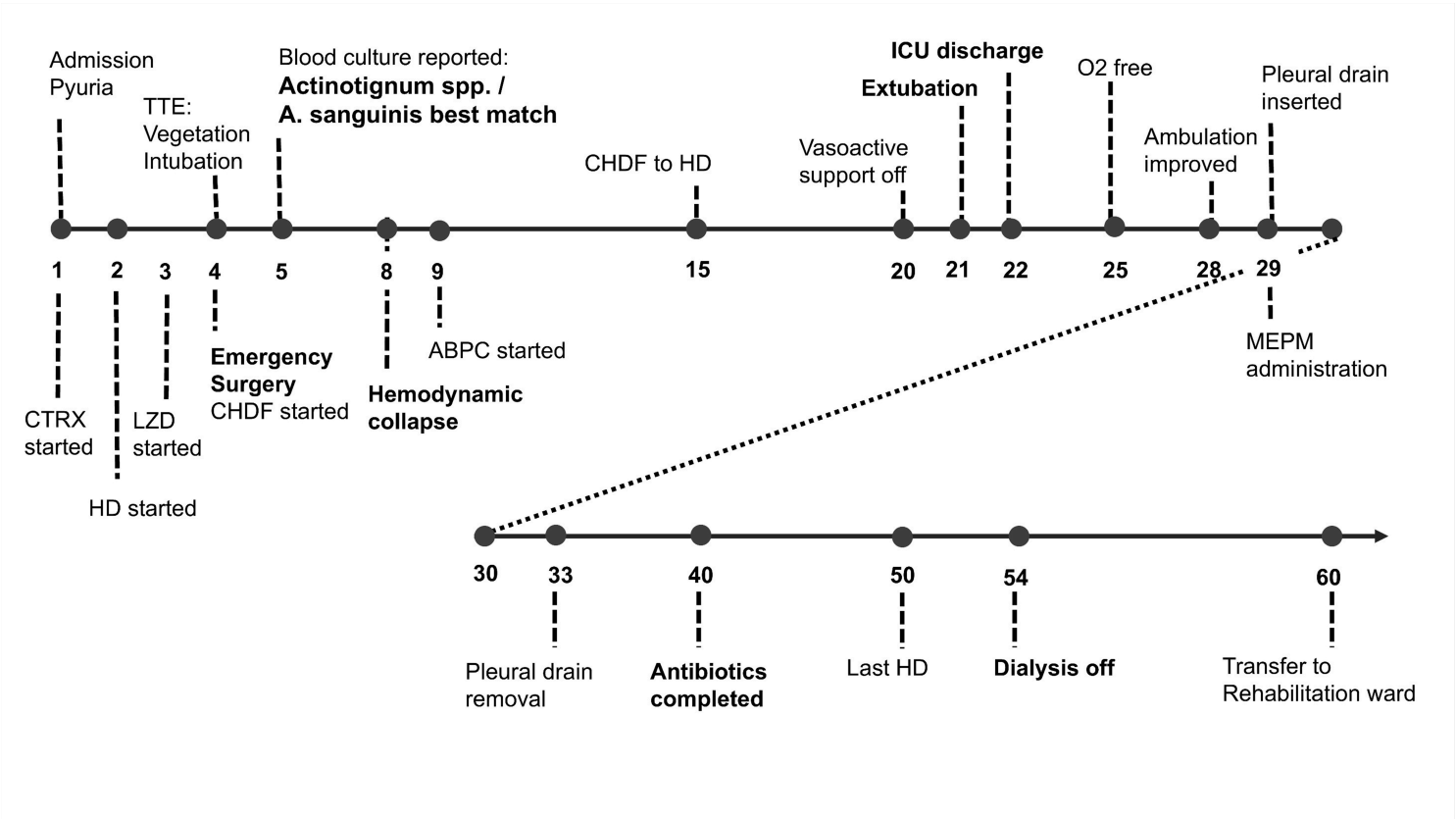


Figure 3

Overall clinical course from admission to transfer to the rehabilitation ward.

The early ICU course is expanded for clarity. Major clinical events are shown, including diagnosis of prosthetic valve infective endocarditis, emergency surgery, microbiological reporting, antimicrobial transition, ICU discharge, ambulation recovery, pleural drainage, completion of antimicrobial therapy, discontinuation of hemodialysis, and transfer to the rehabilitation ward. TTE, transthoracic echocardiogram; HD, hemodialysis; CHDF, continuous hemodiafiltration; *A. sanguinis*, *Actinotignum sanguinis* as the best MALDI-TOF MS match; MALDI-TOF MS, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry; LZD, linezolid; CTRX, ceftriaxone; ABPC, ampicillin; MEPM, meropenem.

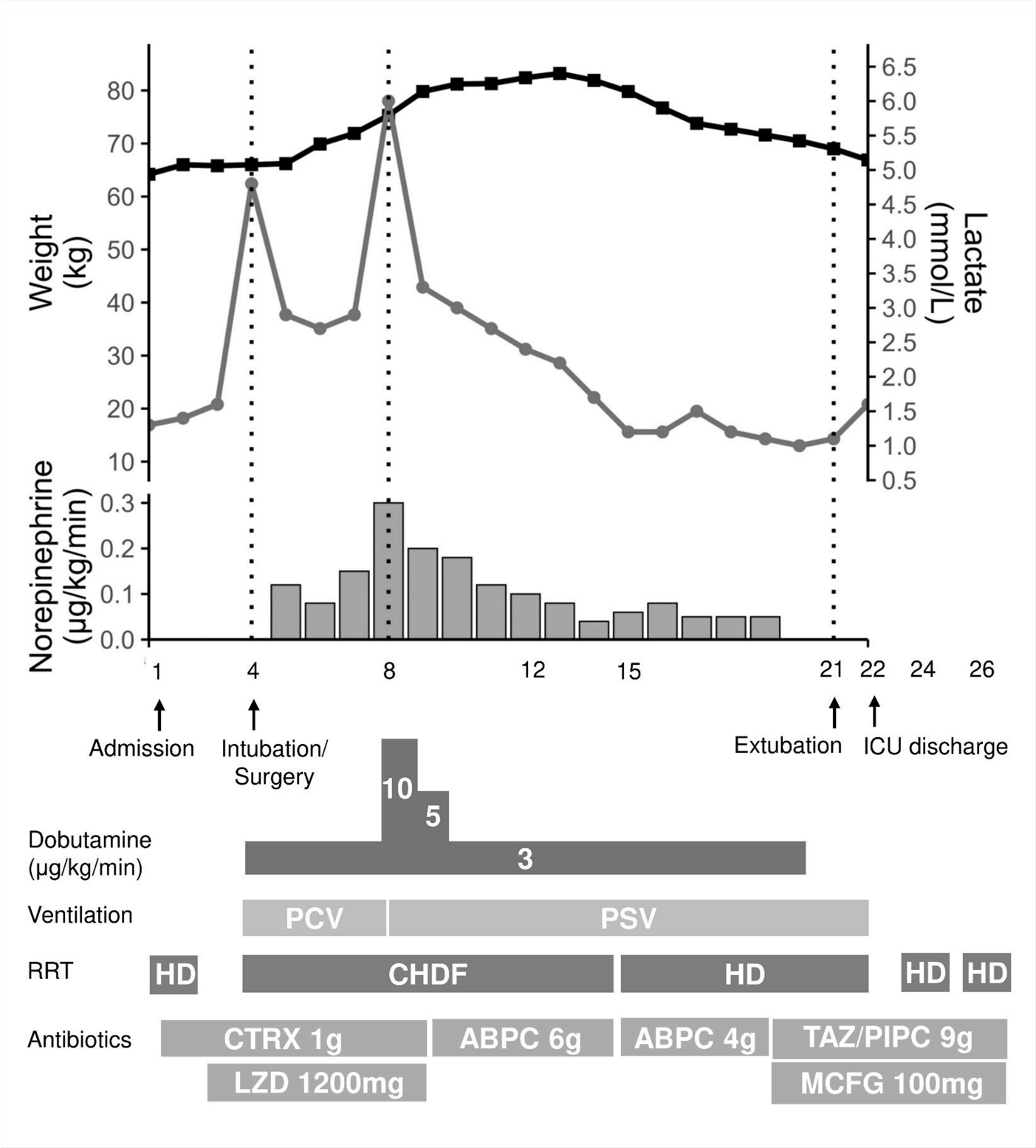


Figure 4

Trends in body weight, lactate, norepinephrine dose, and organ support during the early ICU course.

Body weight and lactate are shown as solid lines with square and circular markers, respectively. Gray bars indicate the maximum daily norepinephrine dose.

Dotted vertical lines mark intubation/emergency surgery, initiation of ampicillin therapy, and extubation. Lower bars indicate dobutamine support, ventilation mode, renal replacement therapy, and antimicrobial therapy. PCV, pressure-controlled ventilation; PSV, pressure support ventilation; RRT, renal replacement therapy; HD, hemodialysis; CHDF, continuous hemodiafiltration; LZD, linezolid; CTRX, ceftriaxone; ABPC, ampicillin; TAZ/PIPC, piperacillin-tazobactam; MCFG, micafungin.

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