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Dual-Valve Infective Endocarditis Caused by Carbapenem-Resistant Acinetobacter baumannii in a Patient with Bicuspid Aortic Valve: A Case Report

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Case Report

Keywords: Infective endocarditis, Acinetobacter baumannii, Carbapenem resistance, Bicuspid aortic valve, Culture-negative endocarditis

Posted Date: November 10th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11430772/v1

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Additional Declarations: No competing interests reported.

Abstract

Background

Infective endocarditis (IE) caused by carbapenem-resistant *Acinetobacter baumannii* (CRAB) is rare and associated with substantial mortality. When it arises on a bicuspid aortic valve (BAV), simultaneously involves the aortic and mitral valves, and presents with persistently negative blood cultures, diagnosis and management become exceptionally challenging.

Case presentation

A 52-year-old man presented with a four-month history of fever and positive rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies, initially raising suspicion of rheumatoid arthritis. Transesophageal echocardiography revealed a BAV (right–left cusp fusion), a large mitral anterior leaflet vegetation (1.5 × 1.3 cm), and aortic valve vegetations. Splenic and cerebral infarctions were subsequently identified. Blood cultures remained repeatedly negative in the context of antecedent antimicrobial exposure. He underwent double mechanical valve replacement with vegetation debridement, and intraoperative valve tissue culture grew CRAB, prompting adjustment to a high-dose sulbactam-based regimen. He recovered and was discharged.

To our knowledge, this combination of culture-negative CRAB native-valve IE, BAV, dual-valve involvement, and diagnosis solely by intraoperative tissue culture has not previously been reported. This case illustrates a plausible anatomical mechanism linking BAV to concomitant aortic–mitral involvement, highlights the diagnostic value of intraoperative tissue culture and the importance of timely surgical evaluation, and underscores the potential for positive rheumatoid serology to complicate the diagnostic process.

1. Introduction

Infective endocarditis (IE) remains a life-threatening disease with an annual incidence of 3–10 per 100,000 population and an in-hospital mortality of 15–22% [1, 2]. Gram-positive cocci account for the vast majority of cases, whereas gram-negative bacteria are implicated in only 1–10% [1, 3]. *Acinetobacter baumannii* is a rare but clinically important cause of IE: a systematic review identified only 19 published cases with an overall mortality of 52.6% [4], and a second review of 37 cases reported a mortality of 32.4% [5]. When the pathogen is carbapenem-resistant (CRAB)—designated by the World Health Organization as a "critical priority" pathogen [6]—therapeutic options narrow considerably [7], further compounding the already substantial mortality risk.

Bicuspid aortic valve (BAV), the most common congenital cardiac anomaly (prevalence 0.5–2%), confers a markedly elevated IE risk—up to 12-fold higher than that of the general population [8, 9]. BAV-associated IE preferentially involves the aortic valve, but the eccentric aortic regurgitant jet typical of BAV is

frequently directed toward the anterior mitral leaflet, predisposing to concomitant aortic–mitral infection [10, 11].

To our knowledge, we found no previous report describing A. baumannii IE occurring in a patient with BAV. Here, we report a case of dual-valve native-valve IE caused by CRAB in a patient with BAV and persistently negative blood cultures, complicated by cerebral and splenic embolization, and successfully managed with early surgery and a sulbactam-based regimen.

2. Case Presentation

Initial presentation and diagnostic delay

A 52-year-old Han Chinese man from Inner Mongolia presented to the emergency department with a four-month history of intermittent fever (peak temperature 39.5°C), accompanied by malaise, loss of appetite, and a 10-kg weight loss. He had no prior hospitalization, invasive procedure, implanted device, or intravenous drug use. The complete clinical course is summarized in Table 1. Before admission, serological testing at outside hospitals in June 2026 yielded positive rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies, leading clinicians to initially consider possible rheumatoid arthritis. Despite the positive serology, he had no joint swelling, morning stiffness, or clinical evidence of synovitis. A brief trial of Tripterygium wilfordii was discontinued after five days because of persistent malaise and lack of clinical improvement. However, the prolonged febrile illness and 10-kg weight loss remained unexplained. Laboratory tests at the emergency department showed leukocytosis and elevated CRP, ESR, and D-dimer. A single 2-g dose of ceftriaxone was administered, and he was admitted to the inpatient ward two days later (July 9, 2026).

Diagnostic assessment

On admission, the patient was afebrile with stable vital signs (T 36.5°C, BP 109/62 mmHg); physical examination showed a mitral systolic murmur. The electrocardiogram showed sinus rhythm with first-degree atrioventricular block. Laboratory tests revealed mild anemia (Hb 105 g/L), persistent inflammation (CRP 79.4 mg/L, ESR 81 mm/h), mildly abnormal liver function (ALT 66 U/L, AST 52 U/L, albumin 27.4 g/L), and elevated D-dimer (1028 ng/mL). Autoimmune screening revealed positive antinuclear antibody (ANA), RF-IgA, RF-IgM, and anti-centromere antibodies. Infectious screening (syphilis, HIV, hepatitis B/C, T-SPOT.TB, Brucella antibodies, (1,3)-β-D-glucan/galactomannan) was negative. Repeated blood cultures obtained on admission and thereafter were all negative.

The diagnostic reasoning progressively converged on IE. Transesophageal echocardiography (July 15) demonstrated a BAV of the right–left cusp fusion type, a mobile vegetation (1.5 × 1.3 cm) on the A3 segment of the mitral anterior leaflet with prolapse and moderate regurgitation, and aortic valve vegetations with mild regurgitation (Fig. 1). Brain MRI/MRA (July 17) showed acute–subacute cerebral infarction in the left occipital lobe, the occipital horn region of the left lateral ventricle, and the right frontal subcortical area (Fig. 2); neuroprotective therapy with butylphthalide and edaravone was initiated.

By the 2023 Duke–ISCVID criteria, the patient met one major criterion (typical echocardiographic vegetation) and at least three minor criteria (fever, predisposing BAV, and vascular phenomena), establishing definite IE despite negative blood cultures.

Surgical findings and microbiological diagnosis

Surgery was performed 14 days after admission (July 23, 2026). Intraoperative exploration revealed a BAV (right–left fusion) with extensive vegetations—predominantly on the left ventricular surface of the non-coronary cusp, leaving insufficient tissue after debridement—together with mitral A2/A3 vegetations with leaflet thickening, A3 chordal rupture, and a localized P3 vegetation. Aortic valve replacement (22-mm Medtronic AP360 mechanical valve), mitral valve replacement (26-mm Medtronic AP360 mechanical valve), vegetation debridement, and epicardial pacing lead placement were performed. Intraoperative valve tissue culture yielded CRAB.

Antimicrobial management and outcome

The antimicrobial course spanned the pre- and postoperative periods (Table 1). Preoperatively, empirical ceftriaxone 2 g daily was administered from July 10 to 16, followed by vancomycin 1 g every 12 h from July 16 to 22 after transfer to the cardiac surgery service. After surgical debridement and CRAB identification on tissue culture, therapy was initially escalated to empirical meropenem (0.5 g q6h) plus linezolid (600 mg q12h); tigecycline (100-mg loading dose, then 50 mg q12h) was subsequently added; and the regimen was ultimately optimized to piperacillin-sulbactam (5 g q8h) plus sulbactam (2 g q8–12h) plus linezolid, delivering high-dose sulbactam as the principal anti-Acinetobacter component. The patient experienced transient postoperative fever with elevated inflammatory markers and abdominal distension attributed to delayed gastrointestinal recovery; both resolved with continued therapy, and he was discharged in improved condition. At the most recent follow-up, he remained afebrile with stable mechanical-valve function on oral anticoagulation.

Table 1

Clinical course timeline: chronological summary of diagnostic, imaging, microbiological, and therapeutic milestones from symptom onset through postoperative recovery.

Date	Clinical course / event	Key findings & interventions
March 2026	Symptom onset	Intermittent fever (peak 38.5°C), malaise, and anorexia.
June 6,2026	Referring hospital (Inner Mongolia), visit 1	ALT 135 U/L, AST 48.9 U/L, CRP 4.3 mg/L, PCT 0.57 ng/mL, and ESR 77 mm/h; HIV, syphilis, and HBV serologies negative; T-SPOT.TB negative; Brucella antibody negative; rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies positive; sacroiliac joint degeneration.
June 9,2026	Referring hospital (Inner Mongolia), visit 2	HLA-B27 negative; antinuclear antibody (ANA), RF-IgA, and RF-IgM positive; anti-centromere antibody positive.
June 20–25,2026	Trial of Tripterygium wilfordii	Diagnosed with “possible rheumatoid arthritis”; discontinued after 5 days owing to persistent malaise and anorexia.
Early July 2026	Emergency department, index hospital	White blood cell count 11.31 × 10^9/L (elevated), neutrophil count 8.6 × 10^9/L (elevated), hemoglobin 119 g/L (low), C-reactive protein 132.8 mg/L (elevated), ESR 74 mm/h (elevated), and D-dimer 1204 ng/mL (elevated); a single 2-g dose of ceftriaxone was administered.
July 9,2026	Admission	Temperature 36.5°C, pulse 69 bpm, respiration 18 breaths/min, blood pressure 109/62 mmHg; systolic murmur at the mitral area.
July 15,2026	Transesophageal echocardiography (TEE) — pivotal study	Bicuspid aortic valve (right–left cusp fusion); mobile vegetation (1.5 × 1.3 cm) on the A3 segment of the mitral anterior leaflet; moderate mitral regurgitation; mild aortic regurgitation.
July 16,2026	Transfer to cardiac surgery	Antibiotic regimen: ceftriaxone 2 g once daily (July 10–16) switched to vancomycin 1 g every 12 h (July 16–22).
July 17,2026	Brain MRI/MRA	Acute–subacute cerebral infarctions (left occipital lobe, left lateral ventricular occipital horn, right frontal subcortical region); suspected aneurysm at the C5 segment of the right internal carotid artery; butylphthalide and edaravone initiated.
July 23,2026	Surgery	Aortic and mitral valve replacement, vegetation debridement, and epicardial pacemaker lead placement.
July 26,2026	Tissue culture result	Intraoperative valve tissue culture grew carbapenem-resistant Acinetobacter baumannii (CRAB).
July 23–30,2026	Postoperative antibiotics	Meropenem 0.5 g every 6 h plus linezolid 600 mg every 12 h (July 23–27) → tigecycline 50 mg every 12 h added (July 27) → piperacillin–sulbactam 5 g every 8 h plus sulbactam 2.0 g every 8 h plus linezolid 600 mg every 12 h (from July 29).

Date	Clinical course / event	Key findings & interventions
Follow-up	Recovery	Stable vital signs, improving inflammatory markers, no recurrent fever.

3. Discussion

To our knowledge, this is the first report of CRAB native-valve IE diagnosed exclusively by intraoperative tissue culture in a culture-negative setting, occurring on a BAV with concomitant aortic and mitral involvement and systemic embolization. The discussion is organized along the clinical decision pathway—predisposition, diagnosis, complications and treatment.

A rare convergence: CRAB IE on a bicuspid aortic valve

IE caused by *A. baumannii* is uncommon [4, 5], and cases attributable to CRAB represent an even smaller and therapeutically more demanding subset of these rare infections [7, 12]; individual case reports continue to describe native-valve involvement with high mortality [13]. Currently, the available evidence is limited to small collections of reported cases (e.g., n = 19 and n = 37) [4, 5]. In the Salvati series, native valves were involved in 9 of 19 cases and multi-valve involvement was uncommon [4], while the Ioannou series recorded multi-valve involvement in only 12.9% [5]. In addition, the prognosis is poorer for native than prosthetic valves—reported mortality approximately 33% versus 16.6% [13].

Previous reports have documented culture-negative CRAB IE involving prosthetic valves and diagnosed by blood mNGS (Lou et al. [12]), as well as CRAB IE involving native valves with positive blood cultures (Lahmidi et al. [13]). Other reported cases have involved isolated-valve infection or presented as a right atrial mass (e.g., Bhagan-Bruno et al. [14], Kunhi et al. [15]). In contrast, our patient had extensive destruction of both the native aortic and mitral valves requiring double-valve replacement. To our knowledge, the combination of culture-negative CRAB IE, native-valve involvement, a BAV substrate, and concomitant aortic–mitral involvement, with the pathogen identified solely by intraoperative tissue culture, has not previously been reported.

The anatomical basis for dual-valve involvement is well illustrated by recent BAV-IE data: the eccentric aortic regurgitant jet typical of BAV is directed toward the anterior mitral leaflet, facilitating bacterial seeding at that site [10, 11], and a Mayo Clinic series confirmed a significantly higher prevalence of isolated mitral and combined aortic–mitral IE in BAV native-valve IE [10]. In our patient the BAV was right–left fusion type—the most common morphology—and the large mitral A3 vegetation with chordal rupture is anatomically consistent with jet-mediated injury. This anatomical hypothesis is biologically plausible but cannot be confirmed by a single case; nevertheless, careful mitral-valve assessment is reasonable in BAV-associated IE, even when the aortic valve appears primarily affected.

Diagnostic challenges: culture-negative IE and serological mimicry of rheumatoid arthritis

Two diagnostic difficulties converged in this patient—microbiological and immunological—and together account for the four-month delay. Microbiologically, blood culture-negative infective endocarditis (BCNIE) accounts for 10–20% of IE and is most often caused by antibiotic administration before blood culture acquisition, which sterilizes the bloodstream without eradicating valvular infection [16, 17]. Our patient received ceftriaxone both in the emergency department and for six days after admission, followed by vancomycin, superimposed on months of intermittent outside-hospital antibiotic exposure, providing a plausible, although not definitive, explanation for the persistently negative blood cultures.

The 2023 Duke–ISCVID criteria nonetheless allowed a clinical diagnosis of definite IE from echocardiographic and embolic findings [18]. Intraoperative valve tissue culture established the etiology and enabled a transition from empirical broad-spectrum therapy to CRAB-targeted treatment. Mirroring the experience of Lou et al., in whom blood cultures were negative after prior antibiotic exposure and metagenomic next-generation sequencing (mNGS) of blood provided complementary etiologic evidence [12], current recommendations therefore support routine submission of valve tissue for histopathology, culture, and—where available—molecular methods such as 16S rRNA PCR or mNGS, which have demonstrable yield in culture-negative IE [12, 17]. Importantly, A. baumannii is not intrinsically unculturable and can be recovered from blood cultures with extended incubation (up to 5 days). Prolonged incubation, valve-tissue culture, and molecular approaches such as mNGS therefore constitute complementary diagnostic tools in culture-negative IE [12, 14]. Although mNGS was not performed in our patient, the successful recovery of CRAB from intraoperative valve tissue, together with the substantial antecedent antibiotic exposure, suggests that the persistently negative blood cultures were more likely attributable to prior antimicrobial therapy than to an intrinsically unculturable organism.

Immunologically, chronic intravascular infection can induce polyclonal B-cell activation and generate false-positive autoantibodies, which in this patient pointed the initial work-up toward rheumatoid arthritis. Rheumatoid factor is elevated in up to 36% of definite IE cases and was the only inflammatory marker showing a statistically significant difference between definite IE and non-IE controls [19], and IE may overtly masquerade as rheumatoid arthritis with markedly elevated rheumatoid factor titers [20]. Before immunosuppressive therapy is initiated, IE should be actively excluded in febrile patients with positive autoantibodies, particularly when constitutional symptoms such as weight loss are present in the absence of clear clinical features of inflammatory arthritis. This concern is particularly relevant because CRAB infection can progress rapidly in immunocompromised hosts; in an immunosuppressed renal-pancreas transplant recipient, CRAB caused fulminant, disseminated IE that was fatal within days [21]. Rheumatoid factor or anti-CCP positivity alone does not establish rheumatoid arthritis. The two diagnostic traps in this case are mechanistically linked—prior antibiotics obscure the microbiology, while infection-driven autoantibodies misdirect the differential—so both must be actively anticipated in prolonged febrile illness.

Embolic complications and the imperative of cerebral imaging

Systemic embolization occurs in 20–50% of IE, with the brain and spleen the most common sites [17, 22], and concomitant splenic and cerebral embolization has been reported [23]. In our patient, the detection of splenic infarction prompted brain MRI/MRA, which revealed clinically silent multifocal cerebral infarctions and a possible intracranial mycotic aneurysm. This approach is guideline-endorsed: the American Association for Thoracic Surgery (AATS) consensus considers routine preoperative cerebral imaging justified in high-risk left-sided IE, even when asymptomatic, because subclinical cerebral lesions inform surgical timing and hemorrhagic risk on cardiopulmonary bypass [24]; routine cerebral MRI detects abnormalities in up to 80% of IE patients and can upgrade "possible" to "definite" IE [17]; and the 2023 ESC guideline classifies recurrent embolization as a Class I indication for early surgery [18]. Neurology consultation guided neuroprotective treatment and surgical timing in this case; however, the contribution of butylphthalide and edaravone to the favorable outcome cannot be determined from a single case.

Early surgery and antimicrobial management guided by current guidelines

This patient met several guideline-based indications for early surgery, most notably a large (> 10 mm) mobile vegetation with systemic embolization and infection by a highly resistant organism—both Class I indications under the 2023 ESC guideline [18]—together with extensive dual-valve destruction requiring surgical reconstruction. The 2023 ESC guideline and the 2020 ACC/AHA valvular heart disease guideline recommend early surgery—during the initial hospitalization and before completion of antibiotic therapy—for left-sided IE with vegetation ≥ 10 mm and another surgical indication or with recurrent embolization [18, 25]; the AATS consensus places large-vegetation/embolic IE in the urgent (3–5 day) category [24]. The survival benefit is substantial: in the Salvati series, surgical mortality was 16.7% (1/6) versus 69% (9/13) without surgery[4]. Our patient's favorable outcome—double-valve replacement with debridement combined with CRAB-targeted therapy—embodies the principle that for refractory, drug-resistant IE, prompt surgery and pathogen-directed antimicrobials are complementary, not alternative, strategies [12]. Regarding antimicrobials, CRAB IE lacks a standardized regimen; sulbactam possesses intrinsic activity against *A. baumannii* and forms the backbone of high-dose combination therapy [26], and current Infectious Diseases Society of America (IDSA) guidance recommends sulbactam-containing regimens, with sulbactam/durlobactam preferred where available [26]. Our stepwise escalation to a high-dose sulbactam regimen reflects the constraints of locally available agents and the principle of antimicrobial synergy. Novel combinations such as eravacycline–cefiderocol have recently shown promise in refractory CRAB IE [12] and represent a potential option should sulbactam-based therapy fail.

Limitations

This is a single-case report and cannot establish the incidence, causality or generalizability of the observations. Detailed antibiotic minimum inhibitory concentration data and serial long-term follow-up imaging were unavailable, limiting pharmacodynamic interpretation and assessment of long-term outcomes. The relatively short follow-up period limits our ability to assess long-term outcomes.

Nevertheless, the case provides, to our knowledge, the first integrated description of this rare clinical constellation and offers actionable guidance for diagnosis and management.

4. Conclusion

This case illustrates the diagnostic complexity of culture-negative, dual-valve IE in a patient with BAV after antimicrobial exposure. It highlights four practical lessons: (1) in patients with prolonged unexplained fever and positive autoimmune serology, the diagnosis should be reassessed when the overall clinical picture is not fully explained; (2) for suspected culture-negative IE, surgically excised valve tissue should be systematically submitted for microbiological and, when available, molecular testing; (3) systemic embolization should prompt careful assessment for cerebral involvement; and (4) early cardiac surgical consultation is warranted when large vegetations, embolic events, or extensive valvular destruction are present. Because the etiological diagnosis in this case relied on valve-tissue culture without molecular confirmation and long-term follow-up remains limited, conclusions regarding pathogen attribution and the optimal antimicrobial regimen should be interpreted cautiously.

Abbreviations

A. baumannii
Acinetobacter baumannii
BAV
bicuspid aortic valve
BCNIE
blood culture-negative infective endocarditis
CRAB
carbapenem-resistant *Acinetobacter baumannii*
CT
computed tomography
ESC
European Society of Cardiology
IE
infective endocarditis
MRI
magnetic resonance imaging
mNGS
metagenomic next-generation sequencing
RF
rheumatoid factor
TEE
transesophageal echocardiography
TTE

transthoracic echocardiography.

Declarations

Ethics approval and consent to participate Not applicable.

Consent for publication Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief.

Competing interests: The authors declare that they have no competing interests.

Funding: This work was supported by the Peking University People's Hospital Scientific Research Development Funds (Project No. RDL2025-27).

Authors' contributions: HZ collected and analyzed the clinical data and drafted the manuscript. SW contributed to clinical data collection, case analysis, and manuscript revision. DW conducted the literature review and contributed to manuscript revision. ZC contributed to clinical case analysis, interpretation of clinical findings, and critical revision of the manuscript. YZ supervised the work, contributed to the interpretation of clinical findings, and critically revised the manuscript. All authors read and approved the final manuscript.

Availability of data and materials: All relevant data supporting the findings of this case report are included in the article. Additional patient-level clinical data are not publicly available due to privacy and confidentiality considerations.

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Figures

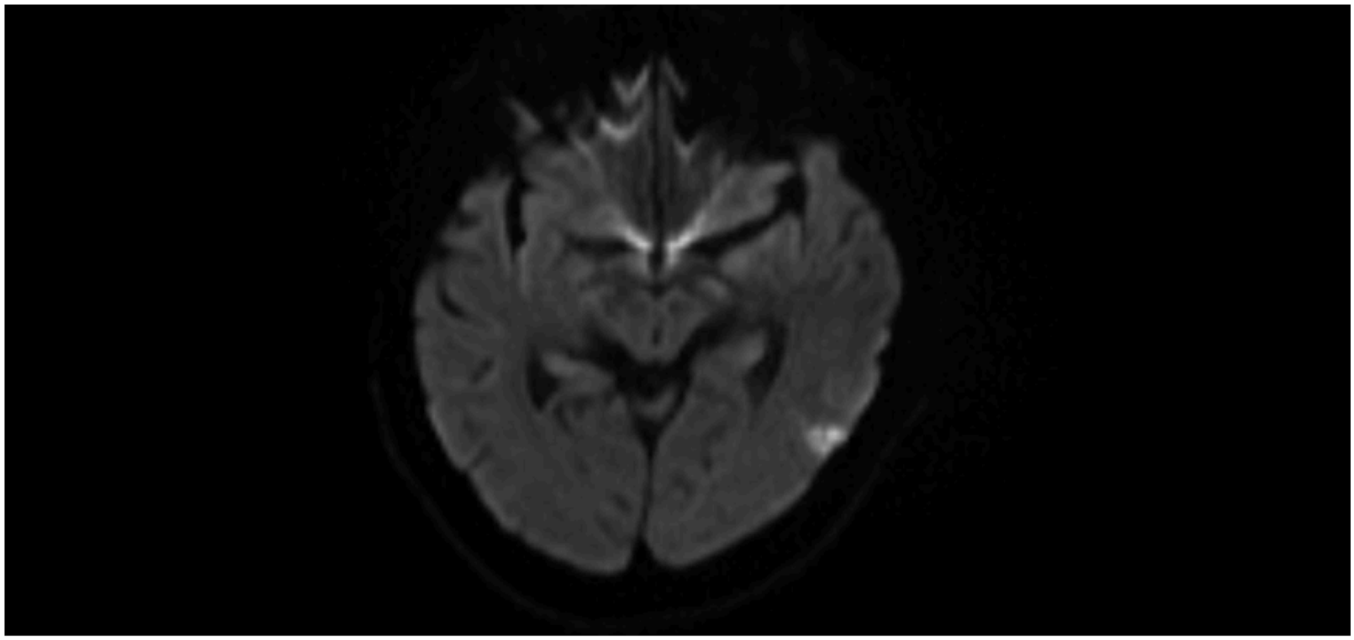


Figure 2

Brain MRI (diffusion-weighted imaging) showing acute-subacute cerebral infarction in the left occipital lobe, the left lateral ventricle occipital horn region, and the right frontal subcortical area.