Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Blood Culture-Negative Tricuspid Valve Infective Endocarditis with Septic Pulmonary Emboli after Urgent Surgery for Severe Abdominal Trauma: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i11.030 | **Received:** 17.08.2026 | **Accepted:** 26.10.2026 | **Published:** 07.11.2026

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Abstract | **Case Report**

Right-sided infective endocarditis (IE) most often affects the tricuspid valve and can be difficult to confirm when antimicrobial therapy begins before blood cultures are obtained. We report a 39-year-old man with no known structural heart disease or injection drug use who developed tricuspid valve IE after severe thoracoabdominal trauma, emergency abdominal surgery, and a complicated postoperative course. Serial echocardiography showed the new appearance and subsequent enlargement of two mobile tricuspid vegetations, from 15 × 5 mm and 16 × 6 mm to 24 × 4 mm and 22 × 5 mm, with severe tricuspid regurgitation. Blood cultures were repeatedly negative after prior antibiotic exposure. Computed tomography later showed bilateral peripheral pulmonary micronodules, several with cavitation, consistent with septic pulmonary emboli. The diagnosis was clinically considered blood culture-negative infective endocarditis and met the 2023 Duke-International Society for Cardiovascular Infectious Diseases definition of possible IE. The patient received ceftriaxone for 21 days with gentamicin during the first 14 days, followed by 42 days of vancomycin. At the end of treatment, the vegetations had partially regressed and the patient remained clinically stable. He was still asymptomatic two months later, with no documented recurrence. The case shows the value of serial echocardiography when the initial study is normal, the limitations of cultures obtained after antibiotics, and the importance of evaluating pulmonary embolic complications in suspected right-sided IE.

Keywords: Endocarditis, Bacterial; Tricuspid Valve; Pulmonary Embolism; Multiple Trauma; Blood Culture.

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INTRODUCTION

Right-sided infective endocarditis (IE) represents approximately 5%-10% of IE cases and most often involves the tricuspid valve. It is classically associated with injection drug use, cardiac implantable electronic devices, central venous catheters, and congenital heart disease. Because embolization from the right side of the heart reaches the pulmonary circulation, respiratory symptoms and fever may be more prominent than the peripheral stigmata typically associated with left-sided disease [1].

Blood culture-negative infective endocarditis (BCNIE) adds another layer of difficulty because microbiologic confirmation and targeted therapy may no longer be possible. The most common reason is antibiotic exposure before cultures are drawn, although fastidious or nonculturable organisms must also be considered [2,3]. We describe a case of native tricuspid BCNIE that developed during a prolonged hospitalization after major abdominal trauma and surgery in a patient without the usual risk factors for right-sided IE. The sequence was particularly informative: the valve was initially normal, new tricuspid lesions appeared on serial echocardiography, and later CT demonstrated pulmonary septic embolization. Organism-specific testing was not performed, and cardiothoracic surgical services were unavailable at the treating center.

CASE REPORT

A 39-year-old man with no known cardiac, renal, pulmonary, or systemic disease was admitted after a work-related fall from the third floor with major thoracoabdominal trauma. He did not smoke or use injection drugs and had no known allergy, pre-existing valvular disease, intracardiac device, or documented central venous catheter. Family history was

noncontributory. The clinical course is referenced to the trauma event (day 0).

Initial whole-body CT showed liver contusions and lacerations, a large hemoperitoneum with active contrast extravasation, pancreatic injury, transverse colonic perforation with pneumoperitoneum, and facial and rib fractures. Emergency laparotomy confirmed multiple hepatic fractures, pancreatic trauma, disruption of the transverse mesocolon root with middle colic artery injury, a perforated necrotic transverse colon, and massive hemoperitoneum with hemorrhagic shock. Management included hepatic packing, ligation of the middle colic artery, mesenteric repair, transverse colectomy with colostomy, abdominal lavage, and placement of two drains.

Table 1: Clinical timeline from trauma through the discharge laboratory assessment

Time after trauma	Event	Key findings or action
Day 0	Major trauma and surgery	Fall from height; hemorrhagic shock; emergency laparotomy, hepatic packing, transverse colectomy, colostomy, mesenteric repair, and drainage.
Day 1	Baseline TTE	Normal valves and right-sided chambers; LVEF 62%.
Day 9	Repeat TTE	Paradoxical septal motion, dilated dysfunctional RV, right atrial area 20 cm^2, and high probability of pulmonary hypertension; no significant valvular lesion.
Day 17	CT pulmonary angiography	No thrombotic pulmonary embolism; bilateral pleural effusions and passive atelectasis.
Day 38	Abdominal MRI	Large peripancreatic collection communicating with the main pancreatic duct at a complete disruption; imipenem and ciprofloxacin administered.
Day 57	Diagnostic TTE	Two mobile tricuspid vegetations (15 × 5 mm and 16 × 6 mm), severe tricuspid regurgitation, and right-sided chamber enlargement.
Day 58	Blood cultures	Three blood-culture pairs negative after imipenem and ciprofloxacin exposure.
Days 59-79	Initial antibiotic regimen	Ceftriaxone 2 g/day for 21 days; gentamicin 225 mg/day concurrently during the first 14 days.
Day 74	Serial TTE	Vegetations enlarged to 24 × 4 mm and 22 × 5 mm; severe tricuspid regurgitation persisted.
Day 80	Vancomycin initiation	Vancomycin 900 mg every 12 hours started after ceftriaxone and continued for 42 days.
Day 113	Staging CT and cultures	Multiple small peripheral pulmonary nodules, some cavitated, interpreted as septic emboli; two blood-culture pairs negative while receiving vancomycin.
Day 121	End-of-treatment TTE	Residual lesions 16 × 5 mm and 21 × 5 mm; LVEF 58%; preserved longitudinal RV function; persistent regurgitation; treatment completion.
Day 122	Discharge laboratory assessment	C-reactive protein 4.6 mg/L; hemoglobin 14.5 g/dL; leukocytes 10.09 × 10^9/L; platelets 270 × 10^9/L; renal and hepatic results within laboratory reference intervals.

Trauma was defined as day 0. Antibiotic course lengths are reported in elapsed days, inclusive of the first and final treatment days. Abbreviations: CT, computed tomography; LVEF, left ventricular ejection fraction; MRI, magnetic resonance imaging; RV, right ventricle; TTE, transthoracic echocardiography.

TTE on day 1 showed a nondilated, nonhypertrophied left ventricle with preserved global and regional systolic function (LVEF 62%), normal right-sided chambers and RV function, and no significant valvular abnormality. By day 9, repeat TTE showed paradoxical interventricular septal motion, RV dilation with impaired function, a right atrial area of 20 cm^2, and a high echocardiographic probability of pulmonary hypertension, described as an acute cor pulmonale pattern. No significant valvular lesion was seen. CT pulmonary angiography on day 17 excluded thrombotic pulmonary embolism but showed bilateral pleural effusions with adjacent passive atelectasis, greater on the right.

On day 34, the patient was transferred to a tertiary gastroenterology service for management of the pancreatic injury. Abdominal MRI on day 38 demonstrated a large peripancreatic collection crossing the pancreatic body and communicating with the main pancreatic duct at a site of complete ductal disruption, with small-volume intra- and retroperitoneal fluid. Imipenem and ciprofloxacin were started. No sample from the collection or the surgical drains was sent for culture. A temperature of 38.5°C was documented on day 44.

During preanesthetic assessment for planned endoscopic ultrasonography, TTE performed 57 days after the trauma revealed a new tricuspid valve abnormality. Two mobile masses compatible with vegetations measured 15 × 5 mm on the septal leaflet and 16 × 6 mm on the posterior leaflet (Figure 1A-B). Severe tricuspid regurgitation was present because of a central coaptation gap. The right atrium was enlarged, and the RV was dilated and hypertrophied, although longitudinal systolic function remained preserved. LVEF was 55%,

and the echocardiographic probability of pulmonary hypertension remained high. Three blood-culture pairs obtained the following day were negative; all were collected after exposure to imipenem and ciprofloxacin. No TEE, organism-specific serology, or molecular testing was performed. The echocardiographer confirmed that the lesions were mobile on dynamic examination.

A repeat TTE 74 days after the trauma showed clear interval enlargement. The lesions were described as highly mobile and serpiginous, measuring 24 × 4 mm on the septal leaflet and 22 × 5 mm on the posterior leaflet (Figure 1C-D). Severe tricuspid regurgitation persisted. This study was performed during ceftriaxone therapy, two days after the final gentamicin dose and before vancomycin was started.

The patient was admitted to cardiology on day 106 while still receiving antimicrobial therapy. He denied recurrent fever, productive cough, dysuria, or chest pain. Blood pressure was 102/72 mmHg, heart rate 72 beats/min, respiratory rate 18 breaths/min, oxygen saturation 99% on room air, and temperature 37.6°C. He weighed 75 kg and was 173 cm tall (BMI 25.1 kg/m²). Cardiac examination showed regular heart sounds, a grade 2/6 murmur over the tricuspid area, and an RV heave. There was no jugular venous distention, hepatojugular reflux, peripheral edema, hypoperfusion, or other clinical evidence of right- or left-sided heart failure. The lungs were clear, the abdomen was soft, and the colostomy was functioning. Neurologic examination was normal. No petechiae, splinter hemorrhages, Janeway lesions, Osler nodes, or Roth spots were present. Oral hygiene was poor.

Electrocardiography showed sinus rhythm at 72 beats/min, left-axis deviation, and left anterior fascicular block without acute repolarization changes. Chest radiography showed no global cardiomegaly, a bilateral interstitial pattern, and a small right pleural effusion. Selected laboratory findings are summarized in Table 2. Twenty-four-hour urine protein excretion was 0.41 g, without hematuria, and rheumatoid factor was negative. Renal and hepatic function remained preserved during treatment.

Table 2: Selected laboratory results at admission and discharge

Analyte (unit)	Gastroenterology (day 34)	Cardiology (day 106)	Discharge (day 122)	Reference interval
Hemoglobin (g/dL)	11.7	13.1	14.5	13.0–18.0
Mean corpuscular volume (fL)	84.8	82.7	81.0	78–98
Leukocytes (× 10^9/L)	9.57	7.48	10.09	4.0–11.0
Neutrophils (× 10^9/L)	7.50	3.60	6.00	1.4–7.7
Platelets (× 10^9/L)	325	290	270	150–400
C-reactive protein (mg/L)	70.8	8.2	4.6	<5
Procalcitonin (ng/mL)	—	0.03	—	<0.50
Creatinine (µmol/L)	52	60	62	59–103
Urea (mmol/L)	3.50	3.66	5.00	2.83–8.16
Aspartate aminotransferase (U/L)	23	28	25	<50
Alanine aminotransferase (U/L)	15	18	28	<50
Albumin (g/L)	29.3	—	—	35–50
Sodium (mmol/L)	136	137	136	136–145
Potassium (mmol/L)	4.0	4.6	4.8	3.5–5.1
Chloride (mmol/L)	—	—	103	98–107

The first two columns refer to gastroenterology and cardiology admission assessments; discharge specimens were collected one day after treatment completion.

Contrast-enhanced cerebral, thoracic, abdominal, and pelvic CT performed 113 days after the trauma showed multiple small peripheral micronodules in both lungs, several of them cavitated; the largest measured approximately 5 mm. One solid nodule was surrounded by a ground-glass halo. In the clinical context, these findings were interpreted as septic pulmonary emboli. The examination also showed bilateral basal atelectatic bands, a moderate left pleural effusion, a small right pleural effusion, and right-chamber cardiomegaly. Hepatomegaly was present, with contrast reflux into the inferior vena cava and hepatic veins, an imaging sign compatible with elevated right-sided pressures (Figure 2A-D). No mycotic aneurysm or acute cerebral lesion was identified. The previously noted peripancreatic collection and intraperitoneal fluid had resolved, and the pancreas and main pancreatic duct had returned to an unremarkable appearance. Two additional blood-culture pairs obtained during the same evaluation remained negative while the patient was receiving vancomycin; sputum and urine cultures were sterile.

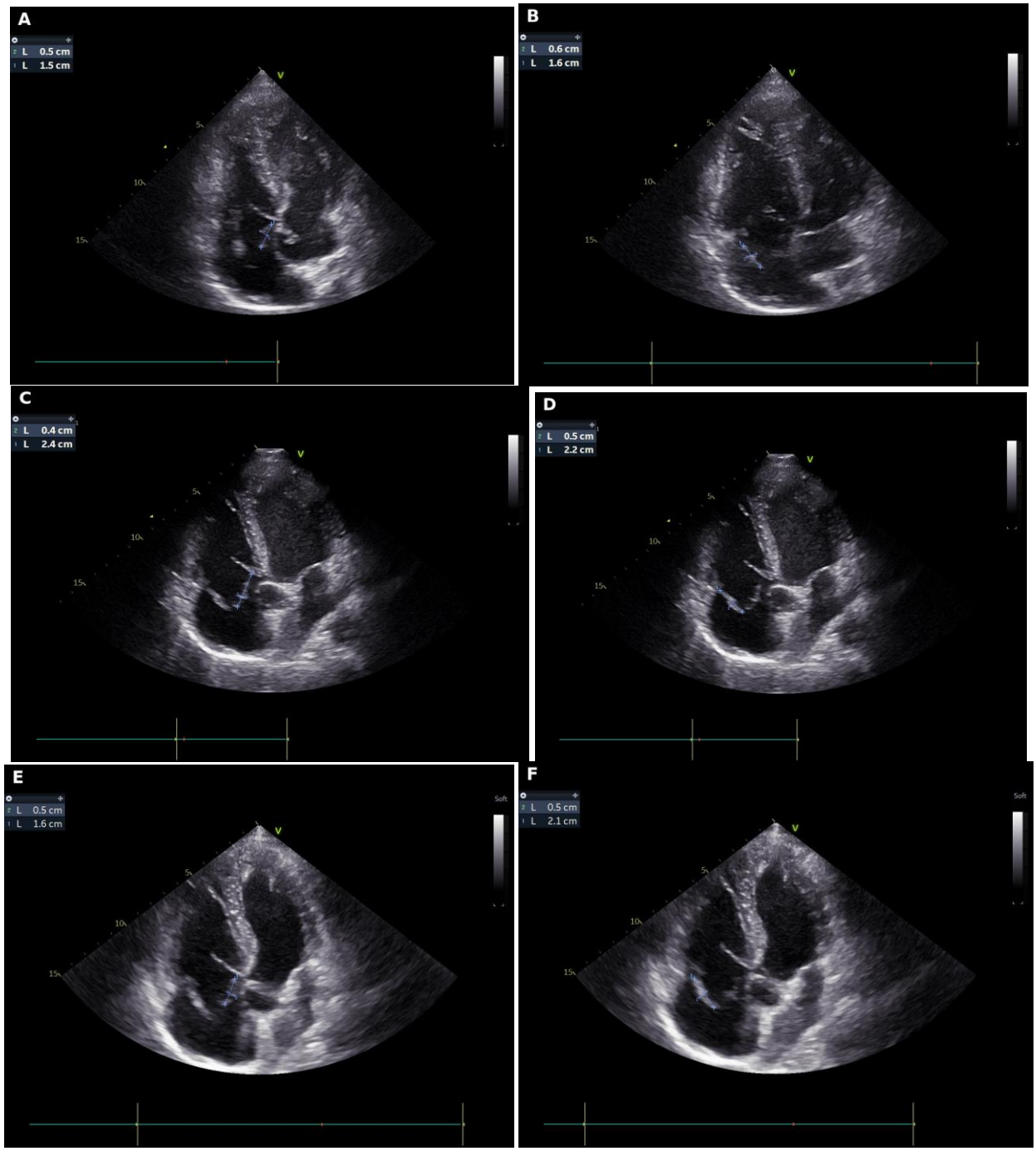


Figure 1: Serial two-dimensional transthoracic echocardiography of the tricuspid lesions. Apical four-chamber views from the initial diagnostic TTE (day 57 after trauma; A-B), the repeat TTE (day 74; C-D), and the end-of-treatment TTE (day 121; E-F). Displayed calipers measure 15 × 5 and 16 × 6 mm, 24 × 4 and 22 × 5 mm, and 16 × 5 and 21 × 5 mm, respectively. Left panels correspond to the reported septal lesion and right panels to the reported posterior lesion. Original on-screen measurements are in centimeters. Vegetation mobility was confirmed by the echocardiographer during dynamic examination

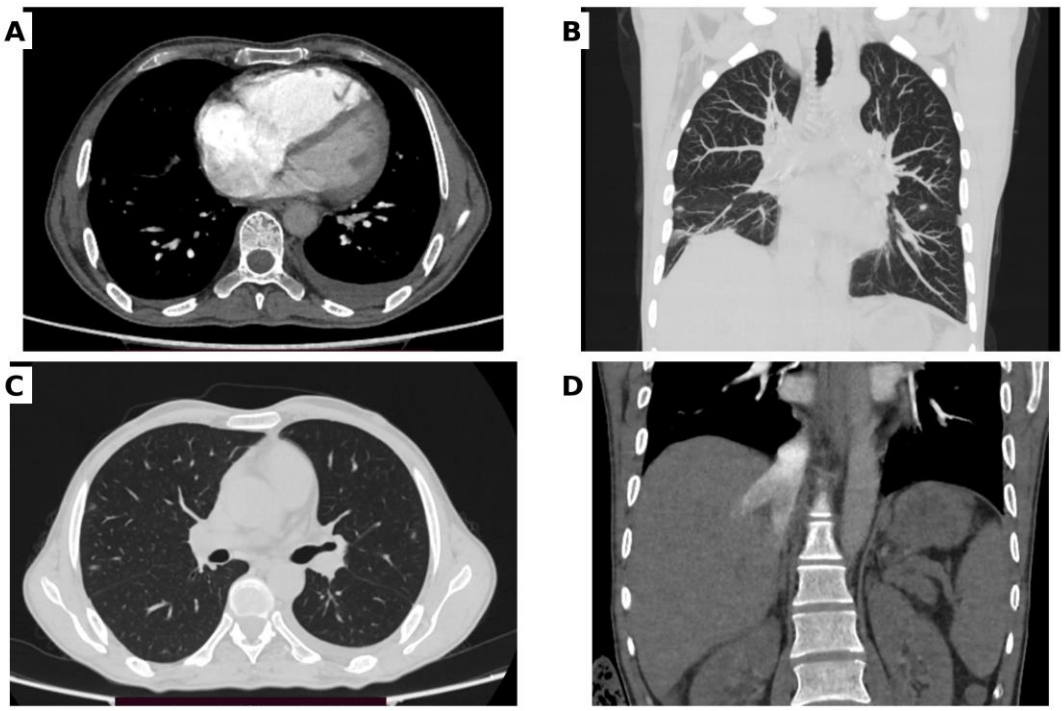


Figure 2: Contrast-enhanced CT findings 113 days after the trauma. (A) Axial thoracic image showing right-chamber cardiomegaly. (B) Coronal lung image showing bilateral peripheral pulmonary micronodules, interpreted in the clinical context as septic emboli. (C) Axial lung image showing additional bilateral micronodules. (D) Coronal abdominal image showing hepatomegaly with reflux of contrast into the inferior vena cava and hepatic veins, compatible with elevated right-sided pressures

The main diagnostic question was whether the tricuspid masses represented infective endocarditis, sterile thrombotic vegetations, or intracardiac thrombus. Several features favored infection: the lesions appeared after previously normal valve examinations, enlarged on serial TTE, were associated with new severe tricuspid regurgitation, and developed in the setting of fever and systemic inflammation. The later appearance of peripheral cavitated pulmonary nodules added further support. Under the 2023 Duke-International Society for Cardiovascular Infectious Diseases criteria, the patient had one major imaging criterion and two minor criteria—temperature >38°C and vascular dissemination to the lungs—meeting the clinical definition of possible IE [4]. Because blood cultures were drawn after antibiotics and no pathogen was identified by another method, the working diagnosis was clinically diagnosed BCNIE of the native tricuspid valve. The postoperative intra-abdominal process was considered a plausible portal of entry, although a direct causal link could not be established.

Treatment consisted of ceftriaxone 2 g intravenously once daily for 21 days, with gentamicin 225 mg intravenously once daily during the first 14 days. Vancomycin 900 mg intravenously every 12 hours was then given for 42 days. Altogether, the patient received nine weeks of antibiotic therapy, including six weeks of vancomycin. Vancomycin serum concentrations were not measured. Spironolactone 50 mg/day and furosemide 40 mg/day were also prescribed. A cardiothoracic surgery service was unavailable at the treating center, so no cardiac surgical team was consulted and the patient was managed medically. Throughout the admission, he remained hemodynamically stable without recurrent fever, respiratory failure, clinical heart failure, worsening conduction abnormality, or arrhythmia.

At treatment completion (day 121 after the trauma), TTE still showed two vegetations, measuring 16 × 5 mm on the septal leaflet and 21 × 5 mm on the posterior leaflet (Figure 1E-F), compared with 24 × 4 mm and 22 × 5 mm on the preceding study. The change was more evident in the septal lesion; the 1-mm difference in the second lesion was small and was not considered meaningful by itself. LVEF was 58%. Right atrial dilation and RV dilation and hypertrophy persisted, while longitudinal RV systolic function remained preserved. The inferior vena cava was not dilated and remained collapsible. Tricuspid regurgitation persisted on color Doppler. At discharge, one day after treatment completion, C-reactive protein was 4.6 mg/L, hemoglobin 14.5 g/dL, leukocytes 10.09 × 10^9/L, and platelets 270 × 10^9/L. Renal and hepatic laboratory values were within the stated reference intervals (Table 2). No post-treatment blood cultures were obtained. At the latest available review, two months after completion of therapy, the patient remained asymptomatic.

DISCUSSION

This patient developed a new native tricuspid valve process during a prolonged hospitalization despite having none of the usual right-sided IE risk factors: no injection drug use, intracardiac device, known structural valve disease, or documented central venous catheter. The sequence of events is important. The valve was normal on day 1 and no significant lesion was present on day 9; only later, after bowel perforation, major abdominal surgery, pancreatic duct disruption, and a peripancreatic collection, did two tricuspid masses appear and enlarge. That chronology makes an acquired healthcare-associated process plausible. Healthcare-associated native-valve IE, including culture-negative episodes, has been described in patients without injection drug use [5]. Still, because blood cultures and the presumed abdominal source were never microbiologically linked, the portal of entry remains uncertain.

The Duke-ISCVID classification also deserves careful wording. Echocardiographic vegetations satisfy a major imaging criterion, while fever above 38°C and septic pulmonary emboli count as minor clinical criteria. The isolated proteinuria of 0.41 g/24 h did not meet the 2023 Duke-ISCVID definition of immune complex-mediated glomerulonephritis because renal function was preserved, hematuria was absent, and there were no compatible biopsy findings [4]. It therefore could not be used as an additional minor criterion. The case consequently met one major and two minor criteria, supporting possible rather than definite IE [4]. Sterile thrombotic lesions remained a theoretical alternative, but the progressive mobility and enlargement of the masses, new severe tricuspid regurgitation, fever, inflammatory response, and compatible pulmonary embolic pattern made infection the leading clinical diagnosis.

The negative blood cultures are most readily explained by the fact that antibiotics had already been given before sampling. This is the leading cause of BCNIE, although fastidious pathogens cannot be excluded on that basis alone [2,3]. Current guidance supports a structured workup with repeat cultures when feasible, a careful exposure history, serologic testing for organisms such as Coxiella burnetii and Bartonella species, and targeted PCR or sequencing when appropriate tissue or other specimens are available [1,2,3]. None of these organism-specific tests was performed. As a result, therapy could not be directed toward a confirmed pathogen, and cultures obtained during vancomycin treatment could not demonstrate microbiologic clearance.

The CT findings were especially helpful because they connected the tricuspid lesions with a typical complication of right-sided IE. Multiple peripheral nodules, cavitation, and pleural involvement are recognized manifestations of septic pulmonary embolism [6]. In this patient, an earlier CT pulmonary angiogram had excluded thrombotic pulmonary embolism. The later development of peripheral, partly cavitated nodules alongside enlarging tricuspid vegetations therefore favored septic rather than bland embolization. The CT also showed right-chamber enlargement and systemic venous contrast reflux, supporting the presence of significant right-sided hemodynamic burden. The small size of the pulmonary lesions and preserved oxygenation likely explain why the patient did not develop overt respiratory failure.

Antibiotic treatment in this case was necessarily empirical because no organism was identified. The 2023 ESC guidelines recommend 4-6 weeks of therapy for most right-sided IE outside the narrowly defined uncomplicated methicillin-susceptible Staphylococcus aureus cases that may qualify for shorter treatment; a six-week vancomycin course falls within that range [1]. The preceding three weeks of ceftriaxone brought the total duration of antibiotic exposure to nine weeks, but this should not be read as a guideline-based nine-week regimen. The lack of pathogen identification and the absence of vancomycin therapeutic drug monitoring also limit how confidently antimicrobial adequacy can be judged. ESC guidance counts treatment from the first day of effective antibiotics, which could not be established microbiologically here [1]. Likewise, aminoglycosides are not routinely recommended for staphylococcal native-valve IE because of renal toxicity and lack of proven benefit [1]; the 14-day gentamicin course is therefore reported as part of the historical management, not as a recommended regimen.

Surgery also required consideration. Contemporary guidance recommends specialist surgical evaluation in right-sided IE when there is persistent bacteremia despite appropriate therapy, severe tricuspid regurgitation with RV dysfunction despite diuretics, recurrent pulmonary emboli requiring ventilatory support, large residual tricuspid vegetations >20 mm after recurrent embolization, or associated left-sided disease [1]. In this case, the vegetations exceeded 20 mm at their maximum size, severe tricuspid regurgitation was present, and CT documented septic pulmonary dissemination. These features would ordinarily prompt valve-center review, but a cardiothoracic surgery service was unavailable at the treating center. The patient had no persistent fever, clinical heart failure, respiratory failure, or left-sided involvement, and the lesions partially regressed by the end of therapy. His favorable short-term course is reassuring, but it does not prove definitive cure because residual masses remained and post-treatment microbiologic confirmation was unavailable.

Several limitations remain. TEE was not performed, so smaller lesions, periannular complications, and alternative diagnoses could not be assessed as completely as they might have been. Vancomycin levels were not monitored. No cultures were obtained from the suspected intra-abdominal

source, and no organism-specific serologic or molecular testing was performed. Post-treatment blood cultures were also not obtained. For these reasons, continued clinical follow-up and serial echocardiography remain important, particularly for recurrent pulmonary embolization, worsening tricuspid regurgitation, or signs of right-sided heart failure. A reduction in vegetation size alone cannot confirm eradication of infection.

CONCLUSION

This case shows how native tricuspid BCNIE can emerge during a complicated hospitalization even when the usual predisposing factors are absent. The combination of newly developed tricuspid masses, fever, severe regurgitation, and peripheral cavitated pulmonary nodules strongly supported the clinical diagnosis, while serial echocardiography demonstrated that the valve lesions were acquired and progressive.

The main practical lessons are straightforward: obtain adequate blood cultures before starting antibiotics whenever the patient's condition allows, pursue a systematic workup when cultures remain negative, and involve an Endocarditis Team or valve center early when large vegetations or septic pulmonary emboli are present. Clinical improvement and partial echocardiographic regression are encouraging, but residual vegetations and persistent tricuspid regurgitation still warrant microbiologic and echocardiographic surveillance.

Informed Consent

Written informed consent was obtained from the patient for publication of the de-identified case details and images.

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