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CASE REPORT

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# Unraveling a rare presentation of bivalvular infective endocarditis using POCUS

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**Key Clinical Message**

Infective endocarditis (IE) is rare, and involvement of two valves is rarer yet. We present a case of a 22-year-old male with liver failure who was found to have bivalvular IE. This case sheds light on the association between bivalvular IE and seemingly unrelated symptoms, emphasizing the need for early recognition.

**KEYWORDS**

bivalvular IE, infective endocarditis, liver failure, POCUS

## 1 | INTRODUCTION

Infective endocarditis (IE) is a rare but potentially lethal infection, with an estimated mortality of 14%–37%.[1] The annual incidence of IE is estimated to be 3–10 per 100,000 people. Still, the epidemiology of the disease has been significantly affected by variations in its predisposing factors, including the increase in intravenous (IV) drug use and the increased number of patients with prosthetic valves.[2,3] Most cases of IE are left-sided and involve a single valve. Bivalvular involvement in IE has not been extensively studied, however available data suggests an incidence ranging from 12% to 30% and more frequently requires surgical intervention.[4] Timely diagnosis of IE is imperative to prevent complications and improve mortality; however, the presentation of IE is highly variable from the timing (i.e., acute versus subacute) to the symptoms. Common symptoms of IE include fatigue, fevers, night sweats, tachycardia, arthralgias, new heart murmur, and dyspnea.[3,5] Presentation with fulminant liver failure and hematemesis is extremely rare. Evaluation of risk factors, including structural cardiac defects, prostatic valves, IV drug use, chronic dialysis, and poor dental hygiene, can aid in diagnosis.[3] Here, we describe an unusual presentation of bivalvular IE, emphasizing the crucial role of early recognition and the diagnostic utility of Point of care ultrasound (POCUS) to facilitate appropriate timely intervention.

## 2 | CASE PRESENTATION

A 22-year-old male was transferred to our tertiary care intensive care unit (ICU) due to fulminant liver failure and upper gastrointestinal (GI) bleeding. He initially presented to an outside emergency department with hematemesis and dyspnea which started the day prior. He reported symptoms of lethargy, fevers, and night sweats

for 4 weeks. His initial blood pressure was 117/40 mmHg, heart rate was 102/min, respiratory rate was 37/min, oxygen saturation was 100% on room air, and temperature was 94.8 F. Electrocardiogram (EKG) revealed sinus tachycardia without significant conduction abnormalities. His laboratory analysis revealed mild leukocytosis, anemia, elevated D-dimer, elevated potassium, lactic acidosis, and elevated creatinine (Table 1). He had a normal platelet count and fibrinogen but significantly elevated INR, aminotransferases, along with low albumin. A computed tomography (CT) angiography of the lungs revealed bilateral ground glass opacities and pleural effusions, but no pulmonary embolus. CT of the abdomen was unremarkable except for hepatomegaly.

## 3 | DIFFERENTIAL DIAGNOSIS, INVESTIGATIONS, AND TREATMENT

Given the recent use of acetaminophen and significantly elevated liver enzymes, he was suspected of having acute acetaminophen toxicity and was started on IV N-Acetylcysteine (NAC) protocol. He was also given empiric antibiotics, fluid resuscitation, packed red blood cells and transferred to our ICU for further management. On admission, he was noticed to have a wide pulse pressure and a faint Grade II/VI diastolic murmur over the left upper sternal border.

POCUS, immediately followed by a transthoracic echocardiogram (TTE), revealed aortic valve vegetations, torrential aortic regurgitation jet hitting the anterior mitral valve leaflet (AMVL), AMVL perforation, and severe mitral regurgitation (Figure 1). Blood cultures were drawn, and the patient was empirically started on IV vancomycin and ceftriaxone. Cardiothoracic surgery was consulted, and he was emergently taken to the operating room. Intraoperative transesophageal echocardiogram confirmed the TTE finding (Figure 2). He underwent aortic valve replacement, repair of the AMVL, and repair of penetrating ulcer on the noncoronary sinus. Peri-operatively, the patient required placement on veno-arterial extracorporeal membrane oxygenation (VA-ECMO). His blood and intra-operative tissue cultures grew *Streptococcus viridans* and antibiotic therapy was tailored according to the sensitivity report. The source was determined to be likely a dental filling done a few months prior to his presentation.

**TABLE 1** Laboratory values on presentation.

| Blood tests | Result | Normal value |
|---|---|---|
| White blood cell count, k/μL | 14.7 | 4.50–11.0 |
| Hemoglobin, g/dL | 9.7 | 13.0–18.0 |
| Platelet, k/μL | 189 | 150–440 |
| INR | 2.5 | <1.1 |
| Albumin, g/dL | 2.3 | 3.5–5.2 |
| Fibrinogen mg/dL | 264 | 173–454 |
| D-dimer, ng/mL | 19,180 | <500 |
| Potassium, mEq/L | 5.7 | 3.5–4.5 |
| Creatinine, mg/dL | 2.2 | 0.72–1.25 |
| Lactic acid, mmol/L | 8.7 | 0.5–2.0 |
| AST, U/L | 8290 | 5–34 |
| ALT, U/L | 5552 | 0–55 |

## 4 | OUTCOME

The patient had an expectant response to IV antibiotics and surgical treatment. He was successfully decannulated off VA-ECMO. His liver and renal function recovered well. The patient was eventually transferred to a rehabilitation facility for neuromuscular recovery with a 6-week course of IV antibiotics.

## 5 | DISCUSSION

Bivalvular endocarditis involving the left-sided valves usually results from the infection of the mitral valve, which results from a "jet lesion" due to aortic regurgitation. The regurgitant jet can be directed towards the mitral valve, leading to the development of vegetations or perforation of the AMVL, resulting in significant mitral regurgitation and bivalvular endocarditis. Sometimes, the same aortic regurgitation "backward-jet" hitting the AMVL causes AMVL fluttering and turbulence in the left ventricle leading to a rumbling diastolic murmur called "Austin-Flint murmur" that is best heard at the apex.[6] Another important cause of bivalvular endocarditis may be the spread of infection into the adjacent mitral annulus, resulting in secondary destruction of the mitral annulus. If concomitant mitral regurgitation develops, it can exacerbate the effects of the aortic regurgitation.[7]

This case shows an unusual presentation of bivalvular IE that illustrates the significance of maintaining a broad differential and utilizing POCUS when seeing young patients presenting with fulminant liver failure. Patients with IE can display a wide variety of atypical symptoms, and many patients may not have clearly definable risk factors. A delay in diagnosis and treatment can lead to severe complications, including heart failure, septic embolization, stroke, renal infarction, and death.[3] Only a few studies have examined bivalvular endocarditis specifically, and recently it was reported to be frequent in acute left-sided IE, with

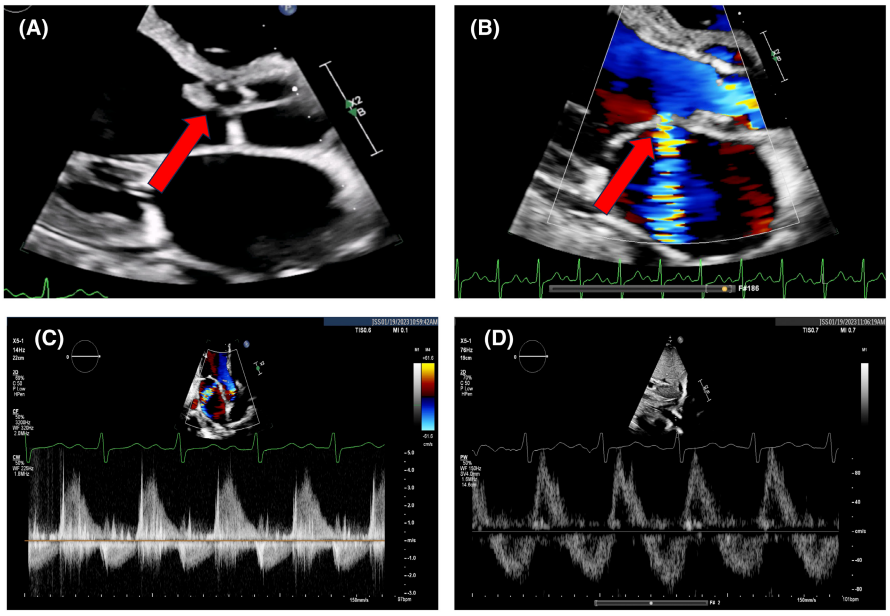


**FIGURE 1** Transthoracic echocardiogram (A). Parasternal long axis (magnified view) showing aortic valve leaflet vegetation and prolapse (red arrow) into the left ventricle (B). Apical 3-chamber (magnified view) showing severe eccentric mitral regurgitation through anterior mitral valve leaflet perforation (red arrow) (C). Apical 3-chamber continuous flow Doppler showing "jet reversal" at aortic valve (D). Abdominal aorta Doppler exam showing flow reversal.

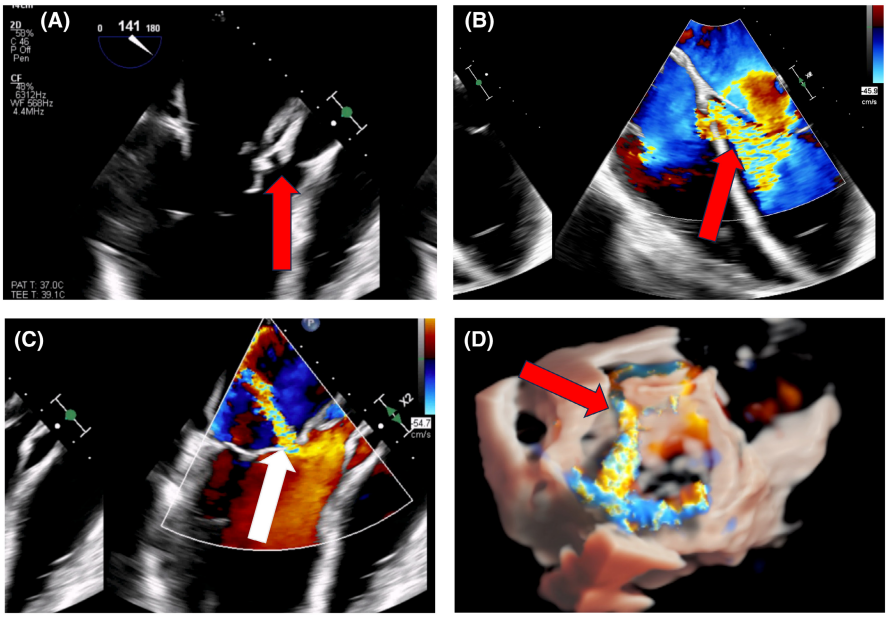


**FIGURE 2** Intra-operative transesophageal echocardiogram (A). Left ventricular outflow tract (LVOT) view showing aortic valve vegetation and prolapse (red arrow) into the left ventricle (B). Color Doppler 4-chamber view showing severe aortic regurgitation causing premature closure of anterior mitral valve leaflet (echocardiographic Austin-Flint Murmur) (red arrow) (C). LVOT view showing severe eccentric mitral regurgitation (white arrow) (D). Three-dimensional (3D) en-face view of the mitral valve. Color Doppler showing anterior mitral valve leaflet perforation (red arrow) and severe eccentric mitral regurgitation.

approximately 19% incidence.[8] They also reported higher rates of embolic events and heart failure in patients with bivalvular IE as compared to single valve IE.[8] Treatment of IE must be tailored to the pathogen, severity of infection, comorbidities, as well as the type of IE (i.e., native or prosthetic valve and single verse multi-valve involvement). As far as surgical intervention is concerned, the recommendations are predominantly based on observational studies thus far. Surgical intervention is indicated for IE complications such as heart block, aortic abscess, destructive penetrating lesions, or valve dysfunction resulting in heart failure.[1] Surgery is also recommended in patients with persistent infection despite antibiotic therapy and should be considered in cases of infection with fungal or highly resistant organisms.[1] A recent study found that early surgical intervention in patients with mitroaortic IE was associated with a reduced 30-day as well as long-term mortality, again highlighting the importance of early diagnosis.[8] Patients with

bivalvular IE require careful long-term follow-up to monitor for recurrence or worsening valve dysfunction.

To summarize, in patients exhibiting symptoms of liver failure, it is important to consider the possibility of bivalvular endocarditis when there is significant valvular pathology. Routine use of POCUS in ICU settings and a multidisciplinary approach are crucial for the successful and timely treatment of bivalvular IE.

## AUTHOR CONTRIBUTIONS

**Mariam Dvalishvili:** Conceptualization; project administration; writing – original draft; writing – review and editing. **Soban Ahmad:** Conceptualization; resources; supervision; validation; writing – review and editing. **Ahmed Hassaan Qavi:** Conceptualization; validation; writing – review and editing. **Sivakumar Ardhanari:** Supervision; validation; writing – review and editing.

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None.

## CONFLICT OF INTEREST STATEMENT

None of the authors of this manuscript have any conflicts of interest to declare.

## DATA AVAILABILITY STATEMENT

Data sharing not applicable—no new data generated.

## CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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