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An Unusual Presentation of Cardiac Tamponade: Transient Bilateral Vision Loss after Aortic Valve Replacement

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Abstract | **Case Report**

Cardiac tamponade is a serious clinical condition in which accumulation of fluid within the pericardial space increases intrapericardial pressure and restricts normal cardiac filling. Pericardial effusion is a known postoperative complication after cardiac surgery, including valve replacement procedures. Although tamponade classically presents with hypotension, dyspnea, and elevated jugular venous pressure, atypical presentations may occur. Transient visual disturbance represents an unusual manifestation and may result from reduced cerebral perfusion. We report the case of a 67-year-old man who developed recurrent episodes of transient bilateral vision loss two weeks after mechanical aortic valve replacement. Diagnostic evaluation revealed a large circumferential pericardial effusion with echocardiographic findings consistent with tamponade physiology. Urgent pericardiocentesis yielded hemorrhagic pericardial fluid with rapid clinical improvement. Anticoagulation therapy was temporarily withheld and subsequently restarted with bridging anticoagulation given the presence of a mechanical valve. This case highlights the importance of considering pericardial effusion and tamponade in postoperative cardiac patients presenting with unexplained neurologic or visual symptoms.

Categories: Cardiology, Internal Medicine, Cardiac/Thoracic/Vascular Surgery
Keywords: Amaurosis Fugax, Cardiac Tamponade, Cerebral Hypoperfusion, Hemorrhagic Pericardial Effusion, Mechanical Aortic Valve, Pericardial Effusion, Postoperative Complication, Transient Bilateral Vision Loss, Warfarin.

INTRODUCTION

Cardiac tamponade is a life-threatening condition that occurs when accumulation of fluid within the pericardial sac increases intrapericardial pressure and impairs normal cardiac filling. As pressure surrounding the heart rises, ventricular diastolic expansion becomes progressively restricted, resulting in decreased stroke volume and reduced cardiac output. The clinical impact of a pericardial effusion depends not only on the total volume of fluid but also on the rate of accumulation, with rapidly developing effusions capable of producing hemodynamic compromise even at relatively small volumes [1, 2].

Pericardial effusion is a recognized complication following cardiac surgery, including valve replacement procedures. Postoperative effusions may arise from inflammation, bleeding, or anticoagulation therapy, and while many remain clinically insignificant, a subset may progress to clinically significant tamponade requiring intervention [4-9]. Because symptoms may be nonspecific, imaging—particularly transthoracic echocardiography—plays a central role in detecting pericardial effusion and tamponade physiology [3-11].

Transient visual loss represents a symptom with a broad differential diagnosis and may arise from ocular, neurologic, or systemic causes. Monocular visual loss is most associated with transient ischemia of the retinal circulation, often related to embolic or vascular disease [7]. In contrast, binocular visual disturbances may occur in conditions associated with systemic hypotension or transient cerebral hypoperfusion, in which both visual cortices are affected simultaneously [8]. Recognition of the underlying cause is critical, particularly in patients with recent cardiac surgery, where hemodynamic abnormalities may contribute to atypical neurologic manifestations.

We present a case of hemorrhagic cardiac tamponade occurring shortly after mechanical aortic valve replacement that manifested primarily with transient bilateral vision loss. This unusual presentation

highlights the importance of considering hemodynamic causes, including pericardial effusion and tamponade, in postoperative cardiac patients presenting with unexplained visual or neurologic symptoms.

Case Presentation

A 67-year-old man with a recent history of mechanical aortic valve replacement two weeks prior to presentation presented with recurrent episodes of transient bilateral vision loss. The first episode occurred three days prior to presentation and lasted several seconds before resolving spontaneously. A second episode occurred on the day of admission while he was seated in a car and lasted approximately 30-45 seconds before complete resolution.

His past medical history was notable for severe aortic stenosis status post-surgical aortic valve replacement, paroxysmal atrial fibrillation, hypertension, chronic obstructive pulmonary disease, chronic kidney disease stage 3a, benign prostatic hyperplasia, and colon cancer in remission. His home medications included warfarin, aspirin, amiodarone, metoprolol, rosuvastatin, and tamsulosin.

The patient denied chest pain, dyspnea, syncope, focal weakness, or speech difficulty. On the morning of admission, he experienced palpitations and measured his blood pressure at home at approximately 145/90 mmHg.

On arrival to the emergency department his vital signs were blood pressure 113/77 mmHg, heart rate 66 beats per minute, respiratory rate 19 breaths per minute, temperature 97.7°F (36.5°C), and oxygen saturation 95% on room air.

Physical examination revealed mild jugular venous distention and distant heart sounds. No focal neurologic deficits were identified.

Laboratory evaluation on admission is summarized in Table 1. Notable findings included a supratherapeutic international normalized ratio (INR), anemia, mild hyponatremia and elevated creatinine. N-terminal pro-B- type natriuretic peptide (proBNP) was elevated at 1,928 pg/mL, while troponin I remained within normal limits.

Table 1: Initial Laboratory Findings on Admission. This table summarizes the patient's laboratory values at presentation

Parameter	Patient Value	Reference Range
Sodium	132 mmol/L	135–145 mmol/L
Creatinine	1.94 mg/dL	0.7–1.3 mg/dL
eGFR	37 mL/min/1.73m²	>60 mL/min/1.73m²
Hemoglobin	9.1 g/dL	13.5–17.5 g/dL
White Blood Cell Count	13.8 ×10³/µL	4.0–11.0 ×10³/µL
Platelet Count	457 ×10³/µL	150–400 ×10³/µL
NT-proBNP	1,928 pg/mL	<125 pg/mL
Troponin I	<0.30 ng/mL	<0.30 ng/mL
Prothrombin Time	43.4 sec	11–13.5 sec
INR	4.4	0.8–1.2
Abbreviations: eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal pro–B-type natriuretic peptide; INR, international normalized ratio		

Electrocardiography demonstrated low-voltage QRS complexes (Figure 1). Chest radiography revealed an enlarged cardiac silhouette in the setting of prior sternotomy without overt pulmonary edema (Figure 2).

Given his recent cardiac surgery and ongoing anticoagulation, transthoracic echocardiography was performed and demonstrated a large circumferential pericardial effusion measuring up to 2.16 cm in maximal separation (Figure 3). Left ventricular systolic function was preserved with an estimated ejection fraction of 60%. Echocardiographic findings consistent with cardiac tamponade physiology were present, including right atrial systolic collapse and significant respirophasic variation in mitral valve inflow.

The patient underwent urgent pericardiocentesis with drainage of 610 ml hemorrhagic fluid, resulting in immediate clinical improvement and complete resolution of visual symptoms.

Given the hemorrhagic nature of the effusion and supratherapeutic INR, warfarin was held during acute management. Following pericardial drainage and clinical stabilization, anticoagulation was cautiously resumed with warfarin and bridging low-molecular-weight heparin due to the high thromboembolic risk associated with a mechanical aortic valve.

A follow-up transthoracic echocardiogram performed six weeks after the episode of cardiac tamponade demonstrated normal left ventricular systolic function with an estimated ejection fraction of 55%. The mechanical aortic valve prosthesis was well seated and functioning normally and no evidence of paravalvular

regurgitation. No recurrent pericardial effusion was identified.

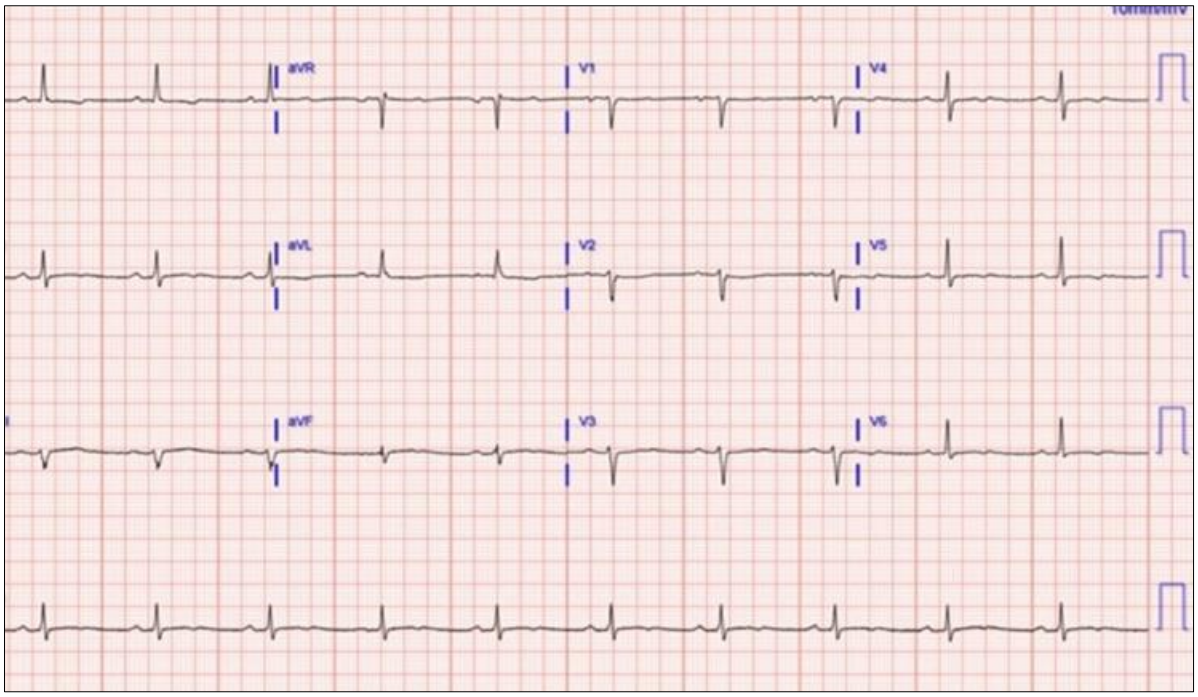

Figure 1: Twelve-lead electrocardiogram demonstrating low-voltage QRS complexes

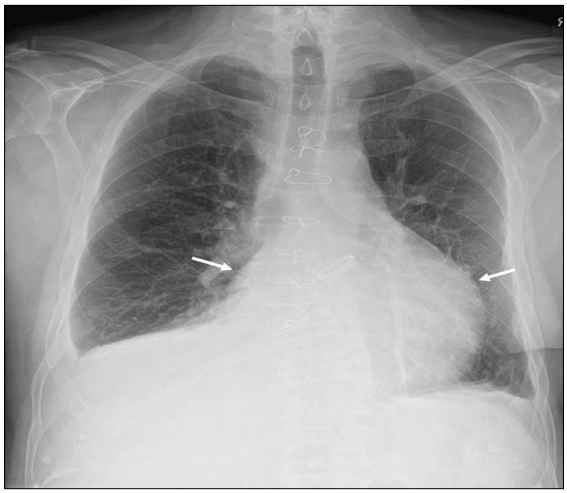

Figure 2: Posteroanterior chest radiograph demonstrating enlarged cardiac silhouette in a patient with prior sternotomy (sternal wires visible). No overt pulmonary edema is present. Arrows indicate the enlarged cardiac silhouette, which is suggestive of a large pericardial effusion

DISCUSSION

Cardiac tamponade results from the accumulation of fluid within the pericardial space leading to elevated intrapericardial pressure and impaired ventricular filling during diastole. As diastolic filling becomes restricted, stroke volume and cardiac output decline, producing the hemodynamic consequences characteristic of tamponade physiology [1, 2]. The severity of symptoms depends largely on the rate of fluid accumulation, as rapid increases in pericardial pressure can compromise cardiac function even when the total volume of effusion is relatively small.

Pericardial effusion is a recognized postoperative complication following cardiac surgery. Studies have demonstrated that a proportion of patients undergoing cardiac procedures develop pericardial fluid collections, with some progressing to clinically significant effusions that require drainage or additional intervention [4, 5]. Reviews of pericardial disease emphasize that careful monitoring and timely imaging are essential in postoperative patients presenting with concerning symptoms [9].

Echocardiography plays a central role in the diagnosis of pericardial effusion and cardiac tamponade.

Typical echocardiographic findings include right atrial systolic collapse, right ventricular diastolic collapse, and respiratory variation in transvalvular flow velocities, which reflect the hemodynamic consequences of increased pericardial pressure [3-11].

Prompt recognition of these findings allows for timely intervention, most commonly with pericardiocentesis, which can result in rapid clinical improvement.

Transient visual loss may occur as either monocular or binocular visual disturbance depending on the underlying mechanism. Monocular episodes are most frequently related to transient retinal ischemia or embolic disease affecting the ophthalmic circulation [7]. In contrast, binocular visual symptoms are more often associated with systemic hypotension or transient reductions in cerebral perfusion that affect the occipital cortices [8]. In the setting of cardiac tamponade, reduced cardiac output and impaired systemic perfusion may contribute to transient cerebral hypoperfusion, providing a potential explanation for the bilateral visual symptoms observed in this patient.

Patients with mechanical aortic valve prostheses require lifelong anticoagulation to prevent thromboembolic complications, most commonly with warfarin therapy [6]. However, supratherapeutic anticoagulation may increase the risk of bleeding complications, including hemorrhagic pericardial effusion. In such cases, temporary interruption of anticoagulation may be necessary during acute management, followed by cautious reinitiation once the patient has stabilized.

This case underscores the importance of maintaining a broad differential diagnosis when evaluating transient visual disturbances, particularly in patients with recent cardiac surgery. Although visual symptoms are more commonly attributed to neurologic or ophthalmologic causes, clinicians should also consider hemodynamic conditions such as cardiac tamponade, as early recognition and prompt treatment are critical to preventing serious complications.

CONCLUSIONS

Cardiac tamponade may occur as a postoperative complication following cardiac surgery and can present with atypical or non-specific symptoms. This case illustrates that transient bilateral vision loss may be an unusual presenting manifestation of tamponade physiology in patients with recent cardiac surgery. Early recognition and prompt echocardiographic evaluation are essential to establish the diagnosis and guide appropriate management. Clinicians should maintain a high index of suspicion for pericardial effusion and tamponade in postoperative cardiac patients presenting with unexplained neurologic or visual symptoms.

Additional Information

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

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Disclosures

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