



## REVIEW ARTICLE

# PARADOXICAL HYPERTENSION COMPLICATING POSTOPERATIVE CARDIAC TAMPONADE: A DIAGNOSTIC CHALLENGE AFTER MINIMALLY INVASIVE ASD CLOSURE

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### ABSTRACT

This report details a 54-year-old female who presented with severe hypertension (170/100 mmHg) and acute dyspnea approximately six weeks after minimally invasive cardiac surgery (MICS) for atrial septal defect (ASD) closure. The initial presentation, characterized by significant hypertension and elevated NT-proBNP, mimicked hypertensive heart failure, prompting diuretic administration. However, a bedside echocardiogram confirmed a significant pericardial effusion, leading to emergent pericardiocentesis. Subsequent investigations revealed a large, organized hemopericardium causing cardiac tamponade, despite the atypical hypertensive state. This case highlights the diagnostic challenge posed by cardiac tamponade when it presents with paradoxical hypertension, a phenomenon reported in a notable subset of tamponade cases. It underscores the potential pitfalls of initial management if the underlying tamponade is masked and emphasizes the need for a high index of suspicion for pericardial complications even after MICS. Surgical re-exploration for clot evacuation led to a favorable outcome.

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## INTRODUCTION

Pericardial effusion is a recognized sequela of cardiac surgery; for instance, a study in the *European Journal of Cardio-Thoracic Surgery* by Lehto et al. reported occurrences in 50-64% of patients, with cardiac tamponade developing in a smaller subset, approximately 0.8-6% (1). While minimally invasive cardiac surgery (MICS) aims to reduce surgical trauma, it is not without risk of such complications. Concurrently, hypertensive emergencies, with Zampaglione et al. noting in *Hypertension* that around 2% of hypertensive patients experience such events, frequently present with dyspnea (in ~22% of cases) or chest pain (~27%) (2), which can mimic other acute cardiac conditions. A particularly perplexing scenario arises when cardiac tamponade presents with paradoxical hypertension; Argulian et al. observed this in approximately 27-43% of patients with subacute tamponade in their study in the *American Journal of Cardiology* (8). This atypical presentation can significantly complicate diagnosis and initial management. We report a case of a 54-year-old female who, six weeks post-MICS for ASD closure, presented with severe hypertension and dyspnea, ultimately diagnosed as cardiac tamponade from an organized hemopericardium, illustrating these diagnostic and therapeutic challenges.

## CASE PRESENTATION

A 54-year-old female with a known history of chronic hypertension underwent MICS for a secundum ASD approximately six weeks prior to this presentation. Her initial postoperative course included a brief readmission (three days after initial discharge, lasting three days) for respiratory symptoms, possibly a lower respiratory tract infection. She was continued on aspirin 75mg daily from her previous discharge medications. She re-presented to the emergency department with a 6-7 day history of progressive cough, resting dyspnea, intermittent feverish sensations, and a syncopal episode three days before this admission. On arrival, she was alert but in significant respiratory distress. Her blood pressure was 170/100 mmHg, qualifying as severe hypertension (Stage 2) in this acute symptomatic setting. Heart rate was 92 beats/minute, respiratory rate 23 breaths/minute, and oxygen saturation 96% on ambient air. Laboratory investigations showed anemia (hemoglobin 8.5 g/dL), leukocytosis (11,060/mm<sup>3</sup>), and hypokalemia (3.0 mmol/L). NT-proBNP was markedly elevated at 4060 pg/mL. Cardiac Troponin assays were not performed. An electrocardiogram revealed sinus rhythm, an RSR' pattern in leads V1-V2, and low voltage precordial leads. Chest radiography demonstrated moderate cardiomegaly with significant bilateral pleural effusions. Given the severe hypertension and respiratory distress apparent on initial presentation, management in the emergency

department included intravenous furosemide for presumed hypertensive heart failure with fluid overload. However, due to persistent distress and high clinical suspicion for an alternative diagnosis, an urgent bedside echocardiogram was performed.



**Figure 1. Bedside echocardiogram revealing a large pericardial effusion. This finding prompted emergent pericardiocentesis, which yielded 650 mL of serosanguinous fluid**

This confirmed a significant pericardial effusion, and emergent pericardiocentesis was subsequently undertaken. This yielded 650 mL of serosanguinous fluid, and a pigtail catheter was left in situ. A more formal transthoracic echocardiogram performed after this drainage (pre-re-exploration) documented only mild residual pericardial effusion, generalized left ventricular hypokinesia (ejection fraction 50-55%), and Grade 1 diastolic dysfunction. Despite initial drainage, concerns for an organized hemopericardium causing persistent cardiac compression led to surgical re-exploration via median sternotomy two days after admission. Intraoperative findings confirmed a significant organized hemopericardium (clotted blood with fibrinous strands and adhesions) within the pericardial sac, particularly anterolaterally. The procedure involved adhesiolysis, meticulous evacuation of all clots and fibrinous material, suction of residual fluid, ensuring hemostasis, and placement of chest drains. The patient recovered well and was discharged approximately eleven days after admission on standard post-cardiac surgery medications.

## DISCUSSION

**The Conundrum of Paradoxical Hypertension in Tamponade** This case's central challenge was the patient's severe hypertension (170/100 mmHg) coexisting with cardiac tamponade. This "paradoxical hypertension" is atypical, as tamponade classically impairs ventricular filling and reduces stroke volume, often leading to hypotension (4,5). However, as reported by Argulian et al., a hypertensive response can occur in 27-43% of patients with subacute cardiac tamponade, especially those with pre-existing hypertension, as was true for our patient (8). Their study also noted that relief of tamponade often results in a decrease in blood pressure (8). The hypertension in this patient was likely multifactorial: her chronic hypertensive state, an acute sympathetic stress response, and possibly an early compensatory increase in systemic vascular resistance, as discussed in guidelines by Ristić et al. and reviews by Spodick (3,4). This misleading hypertension, guided initial treatment towards diuretics. This intervention, while logical for isolated hypertensive fluid overload, risks worsening hemodynamics in tamponade by reducing preload (4,5). The subsequently returned NT-proBNP (4060 pg/mL) confirmed significant myocardial strain, consistent with either condition!

**Etiology of Organized Hemopericardium Post-MICS** The development of an organized hemopericardium six weeks post-MICS

ASD closure suggests a complex process. MICS, using smaller incisions (a right thoracotomy and femoral cannulation were used here), still involves intracardiac manipulation and carries risks of bleeding and inflammation (9,10). Potential contributors include:

1. **Postpericardiotomy Syndrome (PPS):** An inflammatory reaction occurring in up to 30% of cardiac surgery patients according to some literature, potentially causing serosanguinous effusions (1,4). The patient's intermittent feverishness is consistent with PPS.
2. **Hemorrhage:** The operative findings clearly indicated a hemorrhagic process. This could be due to slow oozing from surgical sites or exacerbated by continued aspirin therapy (75mg daily). Even minor persistent bleeding can accumulate. The combination of inflammation and bleeding likely led to the organization of the pericardial contents.

**Diagnostic and Management Considerations** The emergent pericardiocentesis, guided by bedside echocardiography, provided temporary relief. This case emphasizes that MICS does not eliminate the risk of serious pericardial complications (9,10). The absence of Troponin data was a limitation. Creating a pleuropericardial window, as suggested by Gürsoy et al. for surgical ASD closure, might reduce effusion rates (7).

## CONCLUSION

Cardiac tamponade can present atypically with paradoxical hypertension, particularly in patients with pre-existing hypertension, creating a significant diagnostic challenge. This case of organized hemopericardium post-MICS ASD closure underscores the need for a high index of suspicion for pericardial complications despite "minimally invasive" approaches. Prompt bedside echocardiography is crucial in guiding emergent interventions. Definitive surgical exploration is warranted for organized collections causing persistent cardiac compromise.

## REFERENCES

1. Lehto J, Gunn J, Karjalainen P, Airaksinen J, Bodegard J, Harjola VP, et al. Incidence and risk factors of postoperative pericardial effusion and cardiac tamponade. *Eur J Cardiothorac Surg*. 2004 Dec;26(6):1156-61.
2. Zampaglione B, Pascale C, Marchisio M, Cavallo-Perin P. Hypertensive urgencies and emergencies: prevalence and clinical presentation. *Hypertension*. 1996 Jan;27(1):144-7.
3. Ristić AD, Imazio M, Adler Y, Anastakis A, Badano LP, Brucato A, et al. Triage strategy for urgent management of cardiac tamponade: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2014 Aug 21;35(34):2279-84.
4. Spodick DH. Pericardial diseases. In: Zipes DP, Libby P, Bonow RO, Mann DL, Tomaselli GF, Braunwald E, editors. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 11th ed. Philadelphia, PA: Elsevier; 2019. p. 1716-49.
5. Schairer J, Akiyama M, Lelin J. Cardiac Tamponade. [Updated 2023 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-.
6. Gu X, Zheng Q, Zhang Z, Liu H, Li L, Zhuang J, et al. Incidence and predictors of asymptomatic pericardial effusion after transcatheter closure of atrial septal defect. *EuroIntervention*. 2013 May 17;9(1):132-6.

7. Gürsoy M, Hatemi AC, Karaduman M, Ünal Aksu H, Bakır İ. Pleuropericardial Window Prevents Pericardial Effusion Following Surgical Atrial Septal Defect Closure. *Anatol J Cardiol.* 2022 Oct;26(10):778-83.
8. Argulian E, Herzog E, Halpern DG, Messerli FH. Paradoxical hypertension with cardiac tamponade. *Am J Cardiol.* 2012 Oct 15;110(8):1066-9.
9. Mishra YK, Malhotra R, Kuberan A, Meharwal ZS, Trehan N. Minimally invasive atrial septal defect closure in adults. *Indian Heart J.* 2002 Jul-Aug;54(4):393-7.
10. Cleveland Clinic. ASD Closure: Procedure, Risks and Recovery [Internet]. Cleveland (OH): Cleveland Clinic; [updated 2022 Jan 18; cited 2024 May 7].

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