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CARDIAC TAMPONADE DUE TO MINOXIDIL USE: A CASE REPORT AND REVIEW OF THE LITERATURE

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ABSTRACT

Minoxidil is a direct-acting vasodilator that reduces systemic vascular resistance by opening adenosine triphosphate (ATP)-sensitive potassium channels, leading to arteriolar smooth muscle vasodilation. While commonly prescribed for resistant hypertension and androgenic alopecia, minoxidil is associated with fluid retention, and in rare cases, pericardial effusion that can progress to cardiac tamponade. In this report, we present a case of an 81-year-old male with a history of chronic kidney disease (CKD) who developed cardiac tamponade while on chronic minoxidil therapy for hypertension. He presented with progressive dyspnoea, lower extremity oedema, and fever. Imaging revealed an enlarged cardiac silhouette, and a confirmatory echocardiogram demonstrated a pericardial effusion with right ventricular collapse. The patient underwent urgent pericardiocentesis, yielding 545 ml of pericardial fluid. Infectious, malignant, and autoimmune aetiologies were ruled out, and minoxidil was identified as the likely cause. Following discontinuation of minoxidil, the patient's symptoms completely resolved, with no recurrence at 1-month follow-up. In addition, we reviewed 26 previously reported cases of minoxidil-induced pericardial effusion and/or cardiac tamponade, 17 of which are summarized in this report. This case demonstrates the rare but potentially fatal complication of therapy and emphasizes the importance of early detection and clinical vigilance, particularly in vulnerable populations such as patients with CKD.

KEYWORDS

Minoxidil, adverse drug effects, pericardial effusion-cardiac tamponade, pericardiocentesis

LEARNING POINTS

- This case highlights minoxidil-induced cardiac tamponade - a rare but fatal adverse effect requiring urgent intervention, especially underrecognized in clinical practice.
- Patients with chronic kidney disease are at increased risk for minoxidil-induced pericardial effusion due to altered fluid regulation and renin-angiotensin-aldosterone system activation.
- Clinicians should maintain a high index of suspicion and consider routine cardiac monitoring in minoxidil users, even for non-hypertensive indications such as hair loss.

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INTRODUCTION

Cardiac tamponade is a medical emergency caused by excessive fluid accumulation within the pericardial space[1]. The pericardial space normally contains 15-50 ml of serous fluid to help facilitate smooth beating of the heart. However, an increase in pericardial fluid volume raises intrapericardial pressures, causing pressure equalization in all cardiac chambers which disrupts venous return and stroke volume[1]. This leads to subsequent reductions in the pressure gradient between the cardiac chambers and between the right atrium and vena cava, impairing diastolic filling of the heart, which can lead to obstructive shock and death if not acutely managed[1,2]. This condition is commonly associated with malignancy, infection, uraemia, autoimmune diseases, and trauma. However, pharmacologic agents, such as minoxidil, have been implicated as rare causes of pericardial effusion and cardiac tamponade[3].

Minoxidil is a direct-acting vasodilator that lowers systemic vascular resistance (SVR) via the activation and opening of adenosine triphosphate (ATP)-sensitive potassium channels, leading to arteriolar smooth muscle vasodilation[4]. Although it is a third-line antihypertensive agent, its clinical use has increased in popularity for treating androgenic alopecia. However, minoxidil is known to promote fluid retention, peripheral oedema, and in rare cases, pericardial effusion and cardiac tamponade which are overlooked in clinical practice[5]. Several pathophysiologic mechanisms have been postulated for minoxidil-induced pericardial effusion and/or cardiac tamponade, including activation of the renin-angiotensin-aldosterone system (RAAS), vascular endothelial growth factor (VEGF) upregulation, and direct pericardial irritation[6]. Given the increased frequency and popularity of minoxidil use awareness of its cardiovascular adverse reactions is essential for timely intervention. Here, we present a case of minoxidil-induced cardiac tamponade, and discuss its potential mechanisms, and provide a comprehensive overview of published cases.

Original publications and case reports of minoxidil-related cardiac tamponade or pericardial effusion were identified from searching PUBMED, Ovid, Google Scholar and review of references of related articles. This search strategy located 26 well-documented reports relating to the topic. Details pertaining to minoxidil use and related cardiac complications were reviewed and extracted. Similarly, a review of the pharmacokinetics of minoxidil was performed and discussed in the article.

CASE PRESENTATION

An 81-year-old male with a past medical history of hypertension, stage III chronic kidney disease, asthma, and chronic obstructive pulmonary disease presented with progressive dyspnoea on exertion and bilateral lower extremity swelling over the past 2 weeks. Three days prior to presentation, he developed a fever, with a recorded temperature of 38.3°C, associated with a productive cough. An electrocardiogram (ECG) showed sinus rhythm with normal voltage, no evidence of electrical alternans, and no other significant findings. However, an enlarged cardiac silhouette was appreciated on the chest X-ray *(Fig. 1)*.



Figure 1. Chest X-ray showing an enlarged cardiac silhouette.

Initially, the patient was managed with intravenous furosemide and intravenous antibiotics (azithromycin and ceftriaxone) for possible community-acquired pneumonia. Two-dimensional transthoracic echocardiography (2D TTE) on admission demonstrated an ejection fraction of 55%, left ventricular hypertrophy, left atrium dilation and grade II diastolic dysfunction. A computed tomography (CT) scan of the chest confirmed a moderate pericardial effusion without signs of constriction. On day 5 of hospitalization, the patient experienced worsening dyspnoea, jugular venous distension, and hypotension, prompting a repeat 2D TTE, which showed right ventricular collapse, consistent with cardiac tamponade *(Fig. 2)*.

The patient underwent interventional radiology guided pericardiocentesis with an initial drainage of 160 ml at the time of insertion. A total of 545 ml of pericardial fluid was removed over 48 hours. A repeat 2D echocardiogram after the procedure was negative for fluid reaccumulation. Pericardial fluid analysis was negative for infection, malignancy, or autoimmune markers. Given the lack of other causative factors and the patient's chronic use of minoxidil for hypertension, it was suspected as a possible aetiology and immediately discontinued. The patient's symptoms resolved completely, and he was later discharged in stable condition with outpatient cardiology follow-up. No recurrent effusion was noted on echocardiogram at his 1-month follow-up appointment. No recurrence was noted for one year after discontinuing minoxidil.

DISCUSSION

Cardiac tamponade is a life-threatening hemodynamic condition characterized by excessive fluid accumulation within the pericardial space, resulting in elevated intrapericardial pressure that restricts ventricular filling and significantly reduces cardiac output, potentially leading

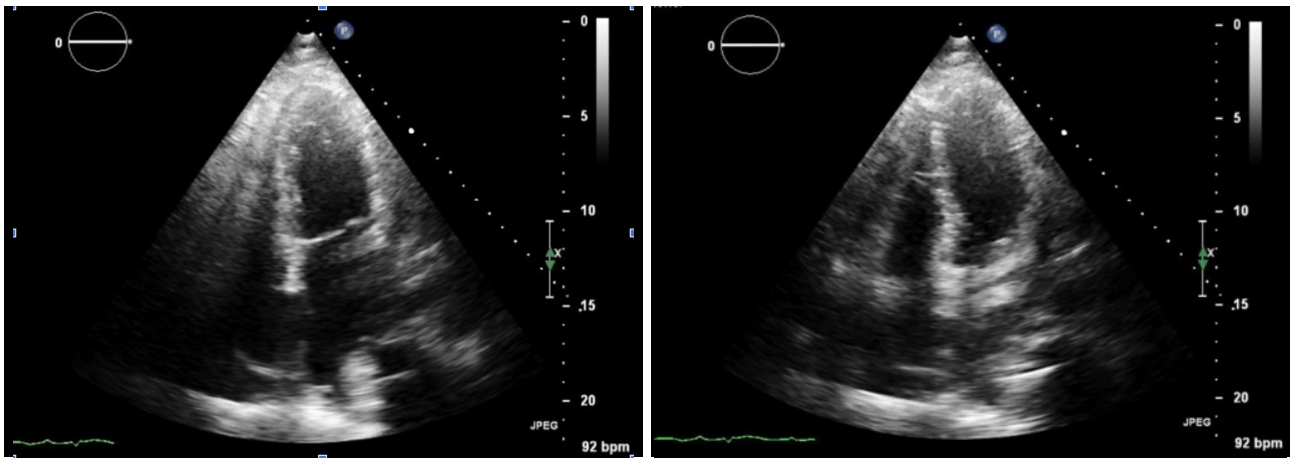


Figure 2. Echocardiogram showing right ventricle collapse.

to circulatory collapse. While tamponade is classically associated with trauma, malignancy, or inflammatory conditions, its association with pharmacologic agents - particularly minoxidil - has received increased attention.

Minoxidil is a potent, direct-acting arteriolar vasodilator primarily used as a third-line agent in the management of hypertension, and more recently, in lower doses for androgenic alopecia. Its mechanism of action involves activation of ATP-sensitive potassium channels in vascular smooth muscle cells, leading to potassium efflux, membrane hyperpolarization, and reduced calcium influx through voltage-gated channels[4]. The resultant decrease in intracellular calcium induces arteriolar smooth muscle relaxation, thereby reducing SVR and afterload, which in turn causes a drop in blood pressure[5]. In addition to its vasodilatory effects, minoxidil has been shown to increase vascular endothelial growth factor expression, enhancing capillary permeability and angiogenesis[6].

Despite its potential efficacy in resistant hypertension, minoxidil has been linked to several adverse effects including hypotension, reflex tachycardia, sodium and water retention, peripheral oedema, and heart failure. In the literature rare but serious complications such as pericardial effusion and cardiac tamponade have been documented with minoxidil use[3-11]. These complications are especially concerning given the expanding dermatologic use of minoxidil in the management of hair loss, generally without strict cardiovascular monitoring. The incidence of minoxidil-induced pericardial effusion has been reported up to 3% in some populations[7]. While patients with CKD or those undergoing dialysis are at a greater risk due to abnormal fluid and electrolyte regulation, studies have also documented cardiac tamponade in healthy individuals with a normal renal function[3,7].

While the exact pathophysiologic mechanism of minoxidil-induced pericardial effusion or tamponade remains enigmatic, several theories have been proposed. One in particular proclaims that arteriolar vasodilation leads to reduced SVR and arterial pressure, triggering RAAS activation, resulting in sodium and water retention[6]. This would essentially put an individual at risk of becoming volume overloaded, in juxtaposition with increased hydrostatic pressure, which may potentially promote transudation of fluid into the pericardial space. Additionally, VEGF upregulation may increase capillary leakage from the pericardial vasculature, further augmenting the risk of effusion formation[6]. Another postulated mechanism includes minoxidil-induced pericarditis[4,7].

In our patient, the development of pericardial effusion was likely multifactorial-mediated by dose-dependent hormonal alterations and renal hemodynamic perturbation. Since minoxidil opens potassium channels, the activity of the Na+/2Cl-/K+ transporter in the ascending limb of the renal tubules may be enhanced, exerting antidiuretic and anti-natriuretic effects. Likewise, its stimulation may enhance aldosterone levels via RAAS axis, exacerbating fluid retention and volume overload[8,9]. These effects are specifically amplified in patients with renal impairment, as seen in our patient with CKD.

Several case reports have been published demonstrating both acute and delayed onset of pericardial effusion following minoxidil therapy, with tamponade in some of the patients[6,10]. Reports of pleuropericardial effusion have been documented, adding further complexity to the clinical picture[11]. Some patients develop a pericardial effusion within weeks of initiating treatment, while others remain asymptomatic for months, complicating timely diagnosis[5,12]. Given that symptoms such as dyspnoea, hypotension, and tachycardia are nonspecific and may be attributable to other causes, a high index of suspicion is required. In rare cases, large effusions are clinically silent, emphasizing the importance of screening and monitoring for cardiovascular abnormalities in patients taking minoxidil[13].

Cardiac tamponade usually manifests with nonspecific findings of muffled heart sounds, hypotension, elevated jugular venous pressure (Beck's triad), tachycardia, and pulsus paradoxus[14]. Doppler echocardiogram remains crucial for identification and diagnostic confirmation of tamponade[9]. Point-of-care ultrasound has proven particularly effective in emergent settings for rapid detection, prompting immediate intervention to prevent further hemodynamic compromise[15]. Echocardiogram findings of pericardial fluid,

Table 1. Summary of reported cases of minoxidil-associated pericardial effusion and tamponade: patient characteristics, clinical course, and outcomes.

Reference	Age/sex	Minoxidil indication	Minoxidil dose (mg/day)	Time to onset of symptoms	Comorbidities	Other aetiologies ruled out	Management	Minoxidil discontinued	Outcome	Recurrence
Reichgott[5]	70/M	Resistant HTN	10	4 months	CHF, CKD, stroke	Yes	-	Yes	Death (cardiac arrest)	-
Marquez-Julio et al.[9]	65/M	HTN	-	2 months	CKD	Yes	Pericardiocentesis/ window - 1 l	Yes	Resolved	No
Oye et al.[10]	48/M	HTN	-	4 weeks	CKD	Yes	Pericardiocentesis - 900 ml	Yes	Resolved	No
Akinleye et al.[11]	52/M	Resistant HTN	10	2 months	CKD-II	Yes	Pericardiocentesis	Yes	Resolved	No
Cilingiroglu et al.[12]	51/M	HTN	-	6 weeks	-	Yes	Pericardiocentesis - 450 ml	Yes	Resolved	No
Lundeen et al.[14]	35/M	Resistant HTN	20	3 years on minoxidil, dose increased 2 months prior	ESRD on HD, lupus nephritis, renal transplant	Yes	Pericardiocentesis/ window - 2.2 l + 550 ml	Yes	Resolved	No
Levy et al.[15]	56/M	Resistant HTN	15	7 years on minoxidil, had small effusion 5 years prior	ESRD on HD, bilateral RCC	Yes	Pericardiocentesis - 1.2 l	Yes	Resolved	No
Yasin et al.[16]	-/F	Androgenetic alopecia	1.25	-	-	-	-	-	-	-
Spodick et al.[17]	53/M	Resistant HTN	-	2 years	ESRD	Yes	Pericardiocentesis - 1.3 l	Yes	Resolved	No
Lew et al.[18]	70/M	Resistant HTN	20	-	-	Yes	Diuretics	Yes	Resolved	No
Trüeb et al.[19]	70/M	HTN	-	3 months	Thoracic aortic aneurysm	Yes	Pericardiocentesis - 700 ml	Yes	Resolved	No
Brondfield et al.[20]	40/F	Androgenetic alopecia	5% topical, 0.25 mg oral	3 weeks	-	Yes	Diuretics	Yes	Resolved	No
Gbadamosi et al.[21]	32/M	Resistant HTN	-	3 months	ESRD on HD, IgA nephropathy s/p renal transplant	Yes	Haemodialysis	Yes	Resolved	No
Dlova et al.[22]	62/M	HTN	30, increased from 15	8 months	CHF	Yes	Pericardiocentesis - 750 ml	No	Resolved	No, but continued minoxidil 20 mg/ day
Nautiyal et al.[23]	30/M	HTN	-	-	ESRD on HD, CHF	Yes	Pericardiocentesis - 1 l	Yes	-	-
Kolinsky et al.[24]	58/M	Resistant HTN	-	-	-	Yes	Symptomatic	Yes	Resolved	No
Webb et al.[25]	43/M	HTN	20	2 months	Left renal artery stenosis	Yes	Pericardiocentesis - 500 ml	Yes	Resolved	No

Abbreviations: HTN, hypertension; CHF, congestive heart failure; CKD, chronic kidney disease; ESRD, end-stage renal disease; HD, haemodialysis; RCC, renal cell carcinoma.

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right atrial and ventricular diastolic collapse is characteristic of tamponade[14].

While pericardiocentesis is the gold-standard for tamponade management in order to decompress the heart, spontaneous resolution of large effusions have been reported in select cases after discontinuing minoxidil[16].

A review of 26 published cases of minoxidil-induced pericardial effusion indicates the condition is more common in patients with CKD and end-stage renal disease. Seventeen of the cases are summarized in *Table 1*. Zarate et al. investigated the incidence of pericardial effusion in patients taking minoxidil, with a higher incidence in haemodialysis patients. The incidence of pericardial effusion in patients on dialysis was 81% (13/16), whereas in patients not on dialysis it was 23% (23/125), indicating increased vulnerability of patients with impaired renal function ($p<0.0005$)[3].

Although the overall incidence of minoxidil-induced pericardial effusion remains low, its potentially fatal nature warrants clinical vigilance, especially as off-label and cosmetic uses of minoxidil expand[16,17]. Clinicians should maintain a high degree of suspicion for cardiovascular adverse effects in patients taking minoxidil.

In our patient, pericardial effusion resolved following cessation of minoxidil, with no recurrence at 1-month follow-up echocardiogram. Common causes of tamponade - such as malignancy, infection, autoimmune disease, uraemia - were excluded, indicating minoxidil as the most plausible etiologic factor.

CONCLUSION

Minoxidil is a widely used antihypertensive and hair loss treatment, but its association with pericardial effusion and cardiac tamponade remains underrecognized. Clinicians should be attentive for cardiac symptoms in short and long-term minoxidil users, particularly those with CKD or volume overload states. Early recognition and timely discontinuation of minoxidil can prevent life-threatening outcomes. Routine monitoring with echocardiogram in high-risk patients may prove beneficial for early detection and mitigate severe complications. Evidence-based monitoring guidelines are essential to prevent fatal complications, especially in patients taking minoxidil for hair loss, where cardiovascular monitoring typically falls outside standard practice.

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