IJPSR (2025), Volume 16, Issue 7 **(Case Study)**

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 19 April 2025; received in revised form, 10 June 2025; accepted, 13 June 2025; published 01 July 2025

SINUS BRADYCARDIA IN SUPER VASMOL POISONING: A RARE BUT SIGNIFICANT CLINICAL MANIFESTATION – REPORT OF TWO CASES

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Keywords:

Super Vasmol, Paraphenylenediamine, Hair dye poisoning, Sinus bradycardia, Cardiotoxicity

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ABSTRACT: Introduction: Super Vasmol, an emulsion-based hair dye containing paraphenylenediamine (PPD), is an emerging cause of suicidal poisoning in developing countries, commonly presenting with cervicofacial oedema, rhabdomyolysis, and acute kidney injury (AKI). Cardiac involvement, though rare, typically manifests as tachycardia or myocarditis. This report highlights sinus bradycardia as an unusual presentation. **Case Presentation:** Two adult women with Super Vasmol poisoning developed marked sinus bradycardia during hospitalization. Case 1, a 34-year-old woman, presented with mild dysphagia and developed asymptomatic sinus bradycardia (heart rate [HR] 38–50/min) on day 3, resolving by day 6. Case 2, a 30-year-old woman, had mild cervicofacial edema and symptomatic bradycardia (HR 40–50/min) with giddiness and breathlessness on day 3, which normalized by day 6. Both patients recovered with conservative management. **Conclusion:** Sinus bradycardia is a rare but significant manifestation of Super Vasmol poisoning, potentially linked to PPD-induced cardiotoxicity or other components. Clinicians should recognize this atypical presentation to optimize management and reduce mortality in resource-limited settings where such poisoning is prevalent.

INTRODUCTION: Super Vasmol, an emulsion-based hair dye containing paraphenylenediamine (PPD), is increasingly implicated in deliberate self-harm in developing nations due to its accessibility [1, 2]. PPD poisoning is characterized by severe clinical manifestations, including cervicofacial oedema, rhabdomyolysis, acute kidney injury (AKI), and, less commonly, cardiac complications [3, 4]. These effects are attributed to PPD's oxidative properties, generating reactive oxygen species and causing multi-organ dysfunction [5, 6].

QUICK RESPONSE CODE

DOI: 10.13040/IJPSR.0975-8232.16(7).2034-38

This article can be accessed online on www.ijpsr.com

DOI link: https://doi.org/10.13040/IJPSR.0975-8232.16(7).2034-38

The rising incidence of Super Vasmol poisoning, particularly as a suicidal method, underscores its public health significance in low-resource settings [7, 8]. Cardiac involvement in PPD poisoning, though uncommon, significantly impacts morbidity and mortality. Reported cardiac manifestations include tachycardia and myocarditis, often linked to systemic inflammation and PPD's direct cardiotoxic effects [9, 10]. Myocarditis is a severe complication associated with poor outcomes [11].

However, the full spectrum of cardiac abnormalities in Super Vasmol poisoning remains underexplored, with bradyarrhythmias rarely reported. Sinus bradycardia, a heart rate below 60 beats per minute with normal P-wave morphology, is an uncommon finding in PPD poisoning. Unlike tachycardia, which aligns with the hyperadrenergic state of acute poisoning, sinus bradycardia suggests

distinct pathophysiological mechanisms, such as myocardial depression, autonomic dysregulation, or electrolyte imbalances secondary to rhabdomyolysis or renal dysfunction [12, 13]. This case report presents two cases of sinus bradycardia as a novel manifestation of Super Vasmol poisoning. It aims to enhance awareness of cardiac complications and emphasize the need for comprehensive cardiovascular monitoring **Table 1.**

Case Presentation:

Case 1: A 34-year-old woman with no prior medical history presented to the Emergency Department (ED) 30 minutes after ingesting approximately 50 mL of Super Vasmol in a suicide attempt. On admission, she was conscious, with blood pressure (BP) 120/80 mmHg, pulse rate 88 bpm, and oxygen saturation (SpO2) of 98% on room air. Physical examination revealed no cervicofacial oedema, with normal cardiovascular and respiratory findings, except for mild dysphagia. A gastric lavage was performed, and she was admitted to the Intensive Care Unit (ICU). She remained asymptomatic for 48 hours. On day 3, an electrocardiogram (ECG) revealed asymptomatic sinus bradycardia (HR 38 bpm) **Fig. 1B**. Atropine (0.6 mg IV) was administered, but HR ranged between 38–50 bpm over the next 48 hours without further intervention. Laboratory tests showed normal renal function (serum creatinine 0.9 mg/dL) and electrolytes (serum potassium 4.5 mEq/L). ENT evaluation, including laryngoscopy, ruled out laryngeal oedema. By day 6, HR normalized to 60 bpm **Fig. 1C**, and she was discharged on day 7 after psychiatric counselling.

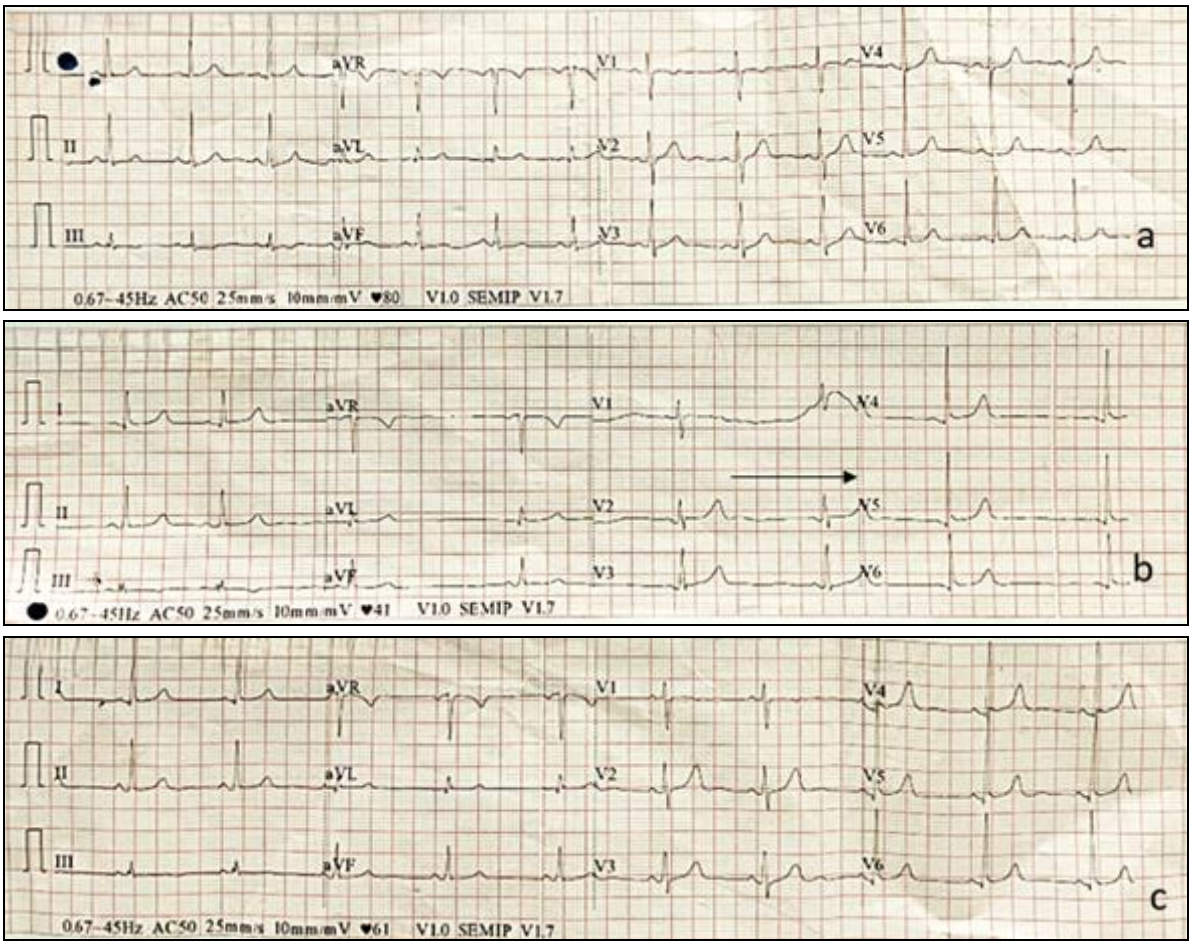

FIG. 1: ECG SHOWING (A) NORMAL SINUS RHYTHM ON DAY 1 WITH HR 75/MIN (B) SEVERE SINUS BRADYCARDIA ON DAY 3 WITH HR VARYING FROM 38 TO 60/MIN AND (C) NORMAL SINUS RHYTHM ON DAY 6 WITH HR 60/MIN

Diagnostic Assessment: ECG confirmed sinus bradycardia. No rhabdomyolysis (normal creatine phosphokinase [CPK]) or AKI was detected.

Case 2: A 30-year-old woman with no significant medical history presented to the ED 2 hours after ingesting an unknown quantity of Super Vasmol

with suicidal intent. Initial vital signs were BP 130/90 mmHg, HR 80 bpm, and SpO2 96% on room air. Examination was unremarkable except for mild cervicofacial edema and dysphagia on day 2. After gastric lavage, she was admitted to the ward.

On day 3, she reported giddiness and breathlessness, with BP escalating to 170/100 mmHg and HR dropping to 48 bpm. ECG confirmed sinus bradycardia **Fig. 2**. Furosemide (40 mg IV) was administered for hypertension, and she was transferred to the ICU. Continuous cardiac monitoring showed HR fluctuating between 40–50 bpm over 48 hours. Intravenous hydrocortisone and antihistamines resolved cervicofacial edema by day 4.

Renal function remained normal (serum creatinine 1.0 mg/dL; urine output >1 mL/kg/h), with mild hyperkalemia (serum potassium 5.2 mEq/L) correcting spontaneously. By day 6, HR stabilized at 65 bpm, and she was discharged.

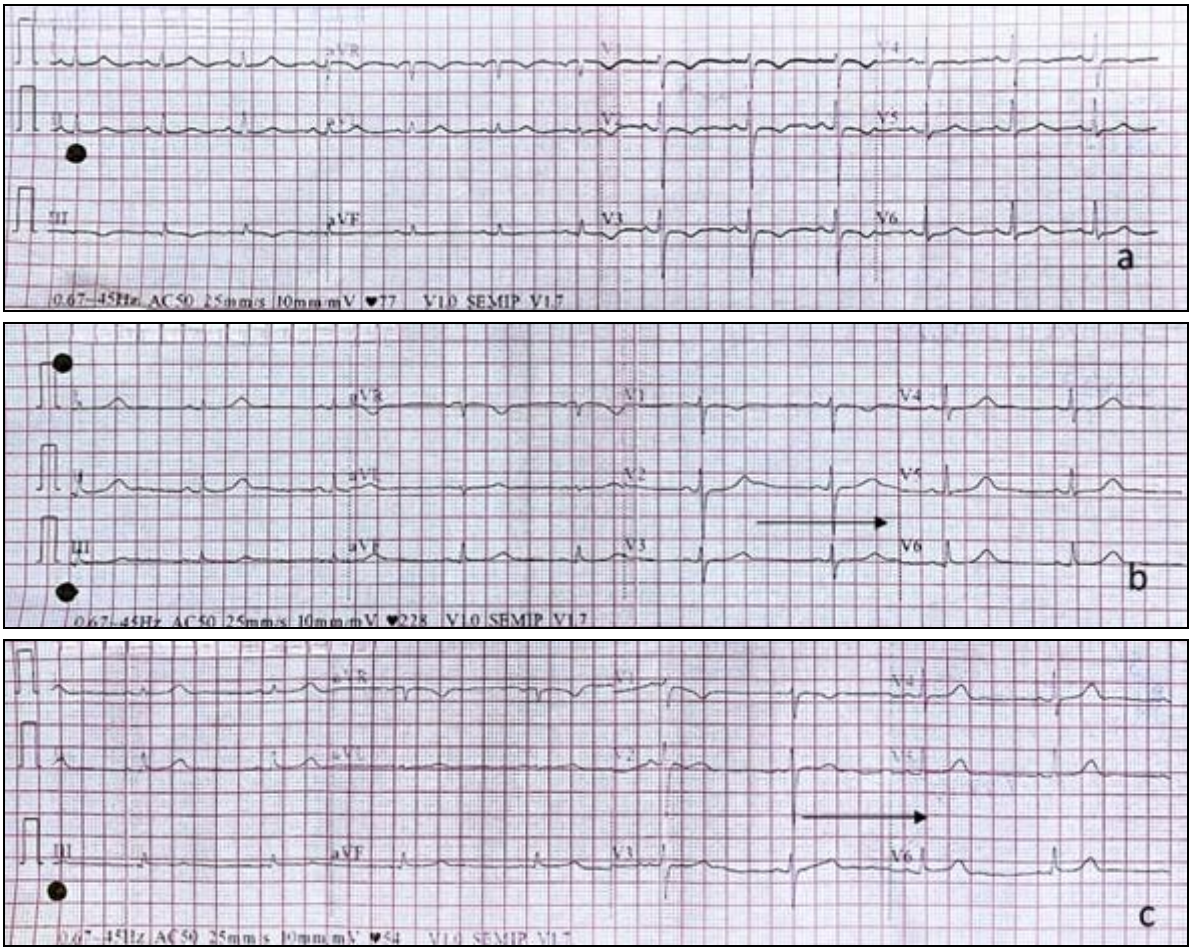

FIG. 2: ECG OF PATIENT 2. ECGS ON DAY 2 (A), DAY 3 (B), AND DAY 5 (C) OF ADMISSION. THE ECGS (B AND C - MARKED WITH ARROWS) SHOWED SINUS BRADYCARDIA

Diagnostic Assessment: ECG confirmed sinus bradycardia. Mild rhabdomyolysis (CPK 800 U/L) was noted without progression to AKI.

TABLE 1: TIMELINE OF CLINICAL EVENTS IN CASE 1 AND CASE 2

Day	Case 1 Events	Case 2 Events
1	Admission, gastric lavage, ICU observation	Admission, gastric lavage, ward admission
2	Asymptomatic, mild dysphagia	Mild cervicofacial edema, IV steroids
3	Sinus bradycardia (HR 38 bpm), atropine	Giddiness, breathlessness, bradycardia (HR 48 bpm), ICU transfer
4–5	Persistent bradycardia (HR 38–50 bpm)	Bradycardia (HR 40–50 bpm), edema resolution
6	HR stabilized (60 bpm)	HR normalized (65 bpm), discharge
7	Discharge after counseling	-

Interventions: Both patients underwent gastric lavage per local protocol, despite its controversial efficacy in PPD poisoning [6].

Case 1 received atropine for bradycardia with minimal response and supportive care for dysphagia. Case 2 was treated with IV hydrocortisone and antihistamines for cervicofacial edema, furosemide for hypertension, and ICU monitoring. No specific antidote for PPD poisoning exists; management focused on symptomatic relief and monitoring [7].

Follow-Up and Outcomes: Both patients recovered fully without sequelae. Case 1 was discharged on day 7 with normal HR (60 bpm) and no renal or respiratory complications. Case 2 was discharged on day 6 with resolved bradycardia (HR 65 bpm) and edema. One-month follow-up showed no symptom recurrence or long-term cardiac effects.

DISCUSSION: Super Vasmol poisoning is a growing public health concern in rural India, predominantly affecting young women due to socioeconomic factors [1, 8]. PPD, the primary toxin, causes early angioedema (within 6 hours) and delayed complications like rhabdomyolysis and AKI (days 2–5) [2, 9]. Cardiac involvement, though rare, includes myocarditis and tachycardia, with morbidity rates of 11–47% depending on ingested volume and time to presentation [4, 12].

These cases highlight sinus bradycardia as a rare cardiac manifestation. Unlike the expected hyperdynamic state from myocarditis or catecholamine excess, [12] both patients developed profound bradycardia (HR 38–50 bpm) on day 3, resolving by day 6. This contrasts with prior reports of tachycardia (76.9% prevalence) or ventricular arrhythmias [2, 11]. A rare case of bradycardia preceding cardiac arrest was attributed to hypoxia, unlike our cases [14].

PPD's aromatic structure may exert direct cardiotoxicity, potentially disrupting sinoatrial node function [15]. Propylene glycol, another component, is linked to arrhythmias (typically tachycardia) at high doses [16]. Resorcinol neurotoxicity or sodium EDTA-induced hypocalcemia could contribute, though serum calcium was normal in Case 1 and untested in Case 2.3 Autonomic dysregulation or vagal responses to systemic inflammation are plausible but unconfirmed due to lack of electrophysiological testing. Notably, neither patient developed severe angioedema, rhabdomyolysis, or AKI, common at doses >50 mL [9]. Case 1 ingested 50 mL without systemic toxicity, while Case 2's unknown dose caused mild edema and rhabdomyolysis (CPK 800 U/L). This variability suggests dose-dependent toxicity, with bradycardia possibly reflecting a distinct phenotype at lower exposures [17]. Management aligned with guidelines prioritizing airway protection and renal support [7]. Atropine's limited efficacy in Case 1 suggests a non-cholinergic mechanism. Recognizing bradycardia as a warning sign prevented unnecessary invasive interventions, unlike cases requiring tracheostomy or dialysis [2, 10].

Strengths and Limitations: This report introduces a novel cardiac complication of Super Vasmol poisoning, supported by ECG evidence **Fig. 1** and **2.** Limitations include lack of serum PPD levels, echocardiography, or Holter monitoring due to resource constraints in rural settings.

Patient Perspective: Both patients' suicidal intent underscores the need for integrated mental health support alongside medical care.

CONCLUSION: Sinus bradycardia is a rare but critical complication of Super Vasmol poisoning, potentially reflecting PPD or component-related cardiotoxicity. Clinicians should be vigilant for atypical cardiac presentations beyond tachycardia to optimize outcomes, particularly in resource-limited settings where PPD poisoning is prevalent. Further research is needed to elucidate mechanisms and refine management strategies.

ACKNOWLEDGMENTS: We thank the patients for consenting to this report and the hospital staff for their support.

Funding: None.

CONFLICT OF INTEREST: The authors declare no conflicts of interest.

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How to cite this article:
Sivamani K and Divyashanthi CM: Sinus bradycardia in super vasmol poisoning: a rare but significant clinical manifestation – report of two cases. Int J Pharm Sci & Res 2025; 16(7): 2034-38. doi: 10.13040/IJPSR.0975-8232.16(7).2034-38.