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Life-Threatening Ventricular Tachycardia and Transient Brugada-Like Electrocardiographic Abnormalities Caused by Severe Hyperkalemia Due to Urinary Retention in a 55-Year-Old Man

Authors' Contribution:

Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Male, 55-year-old

Final Diagnosis: Acute kidney injury • hyperkalemia • urinary retention • ventricular tachycardia

Symptoms: Diaphoresis • dyspnea • palpitations

Clinical Procedure: —

Specialty: Cardiology

Objective: Unusual clinical course

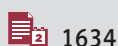
Background: Hyperkalemia is a potentially fatal electrolyte disturbance that typically manifests as progressive conduction abnormalities, bradyarrhythmias, and sine-wave electrocardiographic changes. In rare cases, however, severe hyperkalemia also presents with malignant ventricular tachyarrhythmias and transient electrocardiographic abnormalities involving the right precordial leads, potentially leading to diagnostic confusion with primary arrhythmic disorders.

Case Report: A 55-year-old man with no known cardiovascular disease presented with palpitations, dyspnea, and diaphoresis. Electrocardiography demonstrated sustained wide-QRS ventricular tachycardia accompanied by hemodynamic instability. Following electrical cardioversion, recurrent ventricular tachyarrhythmias occurred, requiring repeated defibrillation, cardiopulmonary resuscitation, intravenous amiodarone, and mechanical ventilation. Laboratory testing revealed severe hyperkalemia (7.6 mmol/L), profound metabolic acidosis (pH 7.059, HCO_3^- 13.8 mmol/L), and advanced renal dysfunction. Serum potassium was confirmed on repeat testing. Subsequent electrocardiography demonstrated marked QRS widening, terminal conduction delay, and transient right precordial ST-segment abnormalities involving leads V1-V2. Intravenous calcium gluconate and sodium bicarbonate rapidly improved the electrocardiographic abnormalities. Continuous hemodiafiltration and bladder catheterization were performed after postrenal acute kidney injury caused by urinary retention was identified. Correction of hyperkalemia together with treatment of metabolic acidosis resulted in complete resolution of the ventricular arrhythmias and electrocardiographic abnormalities without recurrence. No evidence of structural heart disease or persistent myocardial ischemia was identified during subsequent evaluation.

Conclusions: This case illustrates that severe hyperkalemia secondary to reversible postrenal acute kidney injury can simultaneously present with malignant ventricular tachyarrhythmia and transient electrocardiographic abnormalities. Recognition of this reversible metabolic condition is essential to avoid diagnostic error and facilitate prompt life-saving treatment.

Keywords: Hyperkalemia • Ventricular Flutter

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Introduction

Hyperkalemia is a potentially life-threatening electrolyte abnormality that can induce fatal ventricular arrhythmias by altering myocardial membrane excitability and conduction velocity [1,2]. Acute kidney injury (AKI) is a common cause of severe hyperkalemia, and postrenal AKI due to urinary tract obstruction is a reversible but often underrecognized etiology [3,4]. Urinary retention can cause obstructive nephropathy, resulting in impaired renal potassium excretion and potentially severe hyperkalemia, particularly when obstruction is prolonged or accompanied by marked renal dysfunction [3,5]. Hyperkalemia classically produces progressive electrocardiographic changes, including tall, symmetric peaked T waves, PR prolongation, loss of P waves, progressive QRS widening, and ultimately a sine-wave pattern that can precede ventricular fibrillation or asystole [6,7]. Although hyperkalemia classically presents with bradyarrhythmias and conduction disturbances, malignant ventricular tachyarrhythmias, including ventricular tachycardia (VT) and ventricular fibrillation (VF), can occur in severe cases [1,2,6]. Brugada syndrome is an inherited cardiac channelopathy characterized by coved-type ST-segment elevation in the right precordial leads (V1-V3) and an increased risk of ventricular fibrillation and sudden cardiac death [8,9]. Brugada phenocopy refers to a Brugada-like electrocardiographic pattern caused by an identifiable and reversible clinical condition

rather than congenital Brugada syndrome [10]. Several cases of hyperkalemia-associated Brugada phenocopy have been reported, including presentations associated with severe metabolic disturbances such as renal dysfunction and diabetic ketoacidosis [11-14].

The purpose of this report is to illustrate that severe hyperkalemia caused by reversible postrenal acute kidney injury can present with malignant ventricular tachyarrhythmia and transient electrocardiographic abnormalities involving the right precordial leads that resolve after potassium-directed treatment. This case highlights the importance of recognizing reversible metabolic electrocardiographic abnormalities that can mimic primary arrhythmic disorders in emergency clinical settings.

Case Report

A 55-year-old man with no known cardiovascular history was brought to the emergency department with acute palpitations, dyspnea, diaphoresis, and a sensation of heat. He lived alone and had not received regular medical care. There was no known family history of sudden cardiac death, unexplained syncope, or inherited arrhythmia syndrome. He was not taking sodium-channel-blocking drugs or other medications known to induce a Brugada-like electrocardiographic pattern, and there was no

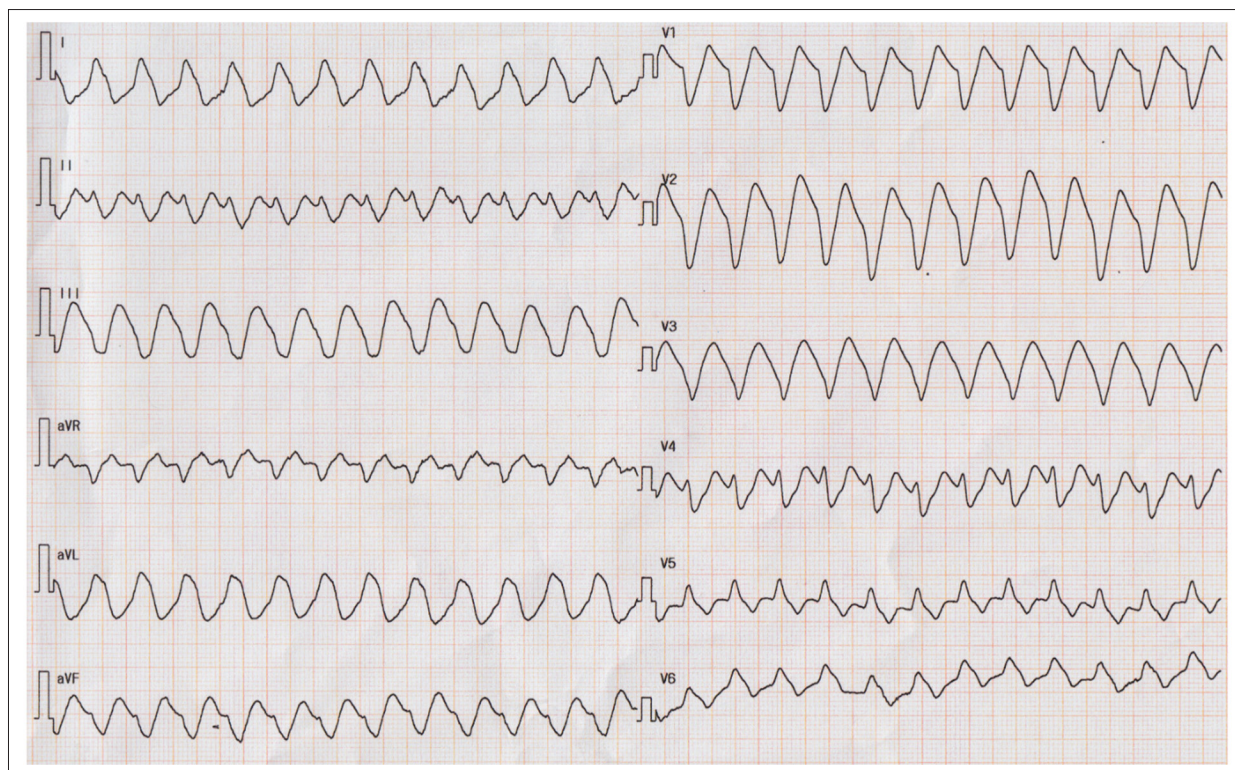


Figure 1. Wide-QRS tachycardia on arrival. Twelve-lead electrocardiogram obtained on arrival demonstrating monomorphic wide-QRS ventricular tachycardia at a rate of 153 beats per minute. Recording speed: 25 mm/s; calibration: 10 mm/mV.

Table 1. Laboratory findings on admission.

Parameter	Value	Unit	Reference range
Hematology			
White blood cells	20.51	$\times 10^3/\mu\text{L}$	3.30-8.60
Red blood cells	4.25	$\times 10^6/\mu\text{L}$	4.35-5.55
Hemoglobin	13.4	g/dL	13.7-16.8
Hematocrit	40.4	%	40.7-50.1
Platelets	533.0	$\times 10^3/\mu\text{L}$	158.0-348.0
Biochemistry			
Total protein	8.9	g/dL	6.6-8.1
Albumin	3.7	g/dL	4.1-5.1
Blood urea nitrogen	129.5	mg/dL	8.0-20.0
Creatinine	26.79	mg/dL	0.65-1.07
eGFR	1.7	mL/min/1.73 m ²	60-89
Total bilirubin	0.3	mg/dL	0.4-1.5
AST	24	U/L	13-30
ALT	20	U/L	10-42
γ -GTP	52	U/L	13-64
Alkaline phosphatase	206	U/L	38-113
Lactate dehydrogenase	604	U/L	120-220
Amylase	37	U/L	44-132
Creatine kinase	1707	U/L	59-248
CK-MB	89	ng/mL	0-6
Troponin T	0.052	ng/mL	0-0.016
C-reactive protein	6.66	mg/dL	0.00-0.14
Electrolytes			
Sodium	142	mmol/L	138-145
Potassium	7.6	mmol/L	3.6-4.8
Chloride	103	mmol/L	101-108
Calcium	9.1	mg/dL	8.8-10.1
Phosphorus	10.6	mg/dL	2.8-4.6
Venous blood gas analysis			
pH	7.059		7.35-7.45
pCO ₂	48.8	mm Hg	35-45
pO ₂	< 10.0	mm Hg	80-100
HCO ₃ ⁻	13.8	mmol/L	22-26
Base excess	-16.5	mmol/L	-2 to +2
Lactate	2.1	mmol/L	0.5-1.5

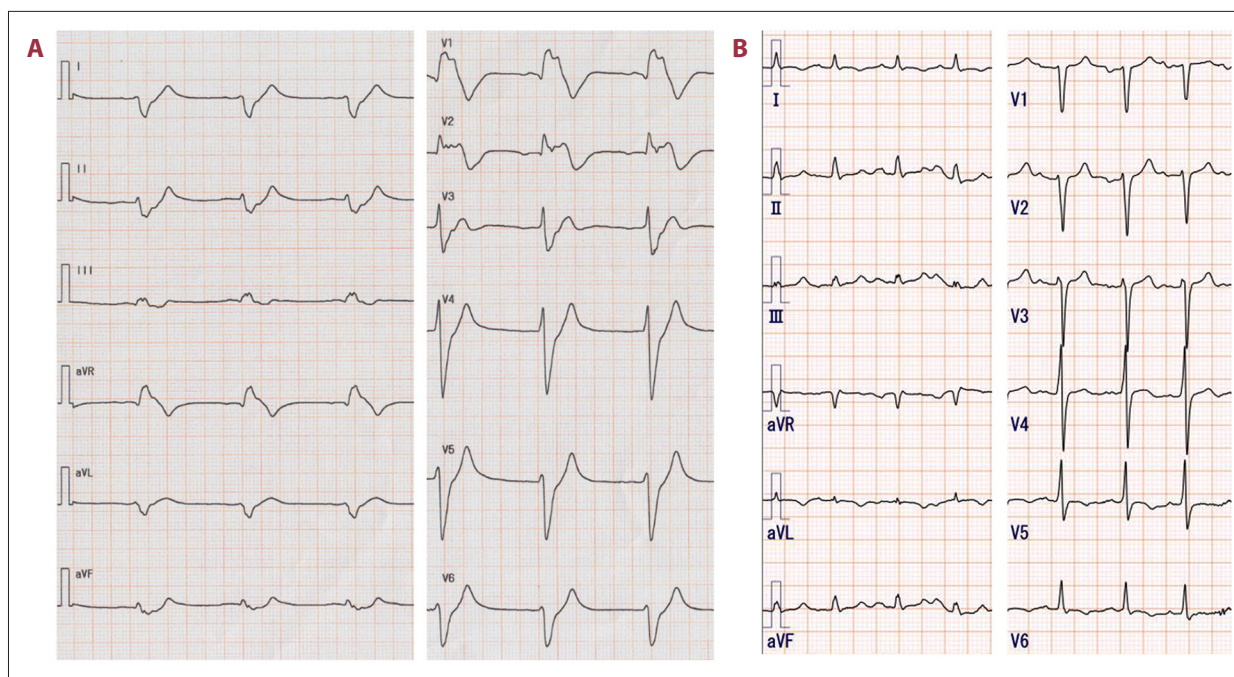


Figure 2. Electrocardiographic changes following cardioversion and treatment of severe hyperkalemia Electrocardiogram obtained after electrical cardioversion showing marked QRS widening, terminal conduction delay, and right precordial ST-segment abnormalities in leads V1-V2 (A). These abnormalities improved rapidly following potassium-directed therapy, including intravenous calcium gluconate administration (B), supporting severe hyperkalemia as the most likely reversible cause of the electrocardiographic abnormalities. The electrocardiographic findings were characterized predominantly by marked conduction delay rather than a definite type 1 Brugada electrocardiographic pattern. Recording speed: 25 mm/s; calibration: 10 mm/mV.

clinical history suggesting drug intoxication. On arrival, he was conscious but in respiratory distress. Electrocardiography revealed sustained wide-QRS tachycardia consistent with monomorphic ventricular tachycardia (Figure 1). His level of consciousness rapidly deteriorated, and electrical cardioversion at 150 J was performed, resulting in transient asystole followed by recurrent VT/VF. Cardiopulmonary resuscitation, repeated defibrillation, epinephrine administration, and intravenous amiodarone were required. Endotracheal intubation and mechanical ventilation were initiated. Laboratory evaluation demonstrated severe metabolic acidosis and marked hyperkalemia, with profound renal dysfunction (Table 1). Following intravenous calcium gluconate and sodium bicarbonate administration, the electrocardiographic abnormalities, including marked QRS widening, terminal conduction delay, and ST-segment abnormalities in leads V1 and V2, resolved promptly (Figure 2). Emergency coronary angiography was deferred because no persistent ischemic electrocardiographic changes or focal left ventricular wall-motion abnormalities were identified on transthoracic echocardiography. Return of spontaneous circulation was achieved after approximately 12 minutes of total cardiac arrest time, with bystander cardiopulmonary resuscitation having been initiated prior to hospital arrival. The patient was admitted to the intensive care unit, treated with

targeted temperature management according to contemporary post-cardiac arrest care recommendations [15], and started on continuous hemodiafiltration. Subsequent evaluation revealed urinary retention due to suspected prostatic obstruction, which was considered to be the cause of the postrenal acute kidney injury. After bladder catheterization and renal replacement therapy, serum potassium levels normalized, and no further ventricular arrhythmias occurred. The patient recovered without neurological sequelae and was transferred for rehabilitation and urological evaluation. No recurrent ventricular arrhythmias were observed during hospitalization, and no antiarrhythmic therapy was prescribed at discharge. The overall clinical course is summarized in Figure 3.

Written informed consent for publication was obtained from the patient. This case report was prepared in accordance with the CARE guidelines.

Discussion

The principal educational value of this case lies in the uncommon combination of severe hyperkalemia, sustained ventricular tachyarrhythmia with hemodynamic collapse, marked transient

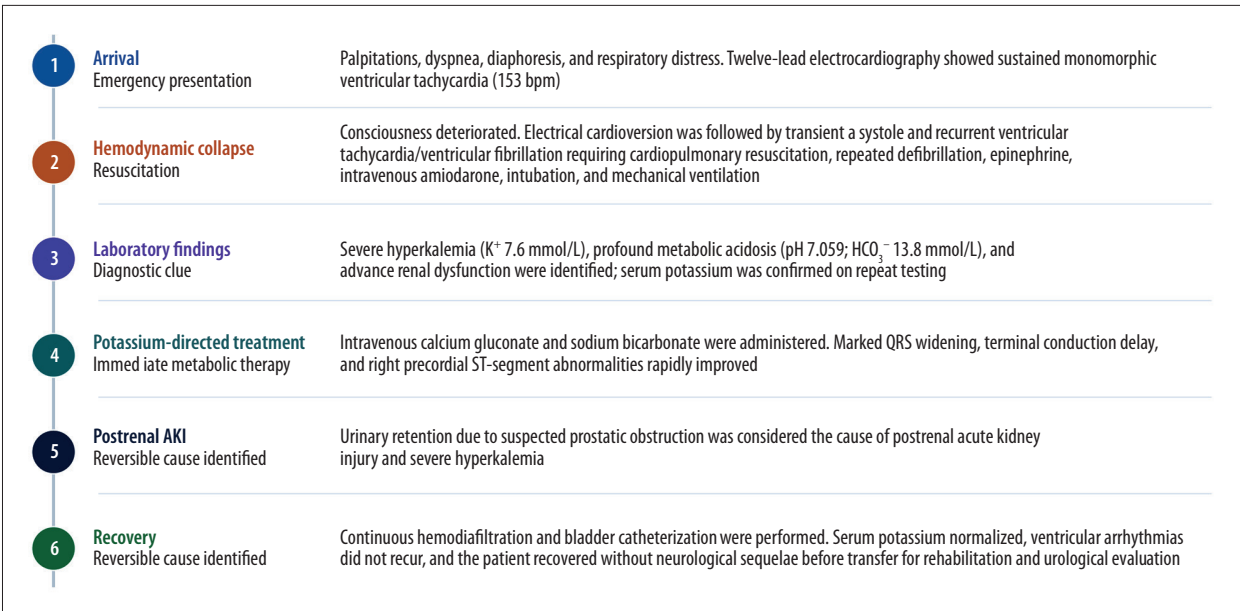


Figure 3. Clinical timeline from emergency presentation to recovery. Clinical timeline summarizing the sequence from emergency presentation with sustained ventricular tachycardia and hemodynamic collapse through laboratory identification of severe hyperkalemia and metabolic acidosis, immediate potassium-directed treatment, recognition of urinary retention causing postrenal acute kidney injury, continuous hemodiafiltration and bladder catheterization, and subsequent recovery without recurrent ventricular arrhythmias. AKI, acute kidney injury; bpm, beats per minute; CPR, cardiopulmonary resuscitation; HCO_3^- , bicarbonate; K^+ , potassium; VT, ventricular tachycardia; VF, ventricular fibrillation.

conduction abnormalities involving the right precordial leads, and reversible postrenal acute kidney injury. Previous reports have described hyperkalemia-associated Brugada phenocopy with a more typical type 1 Brugada electrocardiographic pattern [12,13]. Brugada phenocopy has also been described in diabetic ketoacidosis accompanied by hyperkalemia [14]. In contrast, our patient predominantly exhibited marked QRS widening and terminal conduction delay, and the underlying reversible cause was postrenal acute kidney injury due to urinary retention. There have been few reports describing the simultaneous occurrence of severe hyperkalemia, sustained ventricular tachyarrhythmia causing hemodynamic collapse, transient right precordial conduction abnormalities, and reversible postrenal acute kidney injury. Therefore, the present case provides additional clinical evidence supporting recognition of this uncommon but potentially reversible presentation.

The present findings were most consistent with severe hyperkalemia as the principal reversible trigger. However, concomitant metabolic acidosis, uremia, and post-cardiac arrest physiological changes may also have contributed to the observed conduction abnormalities. Hyperkalemia is a well-recognized cause of life-threatening arrhythmias, typically presenting with bradyarrhythmias, conduction disturbances, and eventual asystole. However, severe hyperkalemia can also provoke malignant ventricular tachyarrhythmias, including ventricular tachycardia and ventricular fibrillation, although this

presentation is less commonly recognized. The observed electrocardiographic changes may reflect sodium channel inactivation caused predominantly by hyperkalemia, potentially augmented by severe metabolic acidosis [2,16]. Such conduction slowing can become heterogeneous, creating regions of functional conduction block and facilitating reentrant ventricular arrhythmias [2].

Hyperkalemia-induced Brugada phenocopy has been previously reported [11,16] and has been proposed to result from accentuation of transmural voltage gradients in the right ventricular outflow tract due to sodium channel inactivation [2,16]. This mechanism is similar to that observed with sodium channel blocker drugs, which are known to unmask Brugada syndrome [8,9]. Unlike previously reported cases demonstrating a definite type 1 Brugada electrocardiographic pattern, the predominant findings in our patient were marked QRS widening and terminal conduction delay. Importantly, this electrocardiographic pattern resolved rapidly after administration of intravenous calcium, strongly suggesting that the electrocardiographic abnormalities were secondary to severe hyperkalemia rather than a primary inherited arrhythmia syndrome.

Although hyperkalemia is classically associated with bradyarrhythmias, ventricular tachycardia can occur in severe cases due to marked conduction slowing and heterogeneous refractoriness, which promote reentry [2,6]. Severe metabolic acidosis,

which was also present in this patient, can further exacerbate sodium channel inactivation and conduction slowing, thereby increasing arrhythmogenicity [2]. Hyperkalemia can also produce a variety of systemic manifestations affecting neuromuscular and cardiovascular function [17].

Another important clinical issue in this case was the differential diagnosis of irregular or wide-QRS tachycardia in the emergency setting. Severe hyperkalemia can mimic ventricular tachycardia, sodium channel blocker toxicity, Brugada syndrome, or acute myocardial ischemia [1,6,11,16]. Recognition of hyperkalemia is critical because the treatment strategy differs fundamentally from that for primary ventricular tachyarrhythmias, and early potassium-directed therapy remains the cornerstone of emergency management [1]. The rapid improvement after intravenous calcium strongly supports membrane stabilization as an important therapeutic mechanism, although a causal relationship cannot be established from a single case [1].

In this patient, the underlying cause of hyperkalemia was postrenal acute kidney injury due to urinary retention. Postrenal acute kidney injury is a reversible but frequently underdiagnosed cause of severe hyperkalemia, particularly in elderly men with prostatic disease. Early recognition and relief of urinary obstruction are essential for definitive management [3].

Although pharmacological provocation testing with a sodium channel blocker such as ajmaline can be useful when Brugada syndrome remains clinically suspected [9], it was not performed in the present case because the electrocardiographic abnormalities resolved completely after correction of hyperkalemia, and no spontaneous type 1 Brugada pattern was observed during hospitalization. Such testing could be considered during follow-up if future clinical suspicion for Brugada syndrome were to arise. Finally, the indication for implantable cardioverter-defibrillator (ICD) therapy was carefully considered. Current ESC and AHA/ACC/HRS guidelines recommend ICD implantation for secondary prevention in patients with ventricular fibrillation or hemodynamically unstable ventricular tachycardia in the absence of a reversible cause [18,19]. Because the ventricular arrhythmia in the present patient was most consistent with a correctable metabolic trigger and did not recur after normalization of serum potassium, ICD implantation was not pursued. No recurrent ventricular arrhythmias were documented during the index hospitalization, further supporting this management strategy.

This report has several limitations. As a single case report, a causal relationship between severe hyperkalemia and the observed electrocardiographic abnormalities cannot be established

definitively. Furthermore, serial potassium measurements obtained before treatment initiation and previous electrocardiograms were not available. Pharmacological provocation testing and genetic testing for Brugada syndrome were not performed because the electrocardiographic abnormalities resolved completely after correction of the electrolyte disturbance and no further clinical suspicion remained. Finally, because the patient was transferred to a hospital closer to his residence shortly after completion of acute treatment, only limited follow-up data were available, precluding a comprehensive assessment of long-term clinical outcomes.

Conclusions

This case suggests that severe hyperkalemia caused by reversible postrenal acute kidney injury can present with both malignant ventricular tachyarrhythmia and transient electrocardiographic abnormalities involving the right precordial leads that can mimic inherited arrhythmia syndromes. Although hyperkalemia is more commonly associated with bradyarrhythmias and conduction disturbances, clinicians should recognize that life-threatening ventricular tachyarrhythmias can also occur. Early identification and treatment of hyperkalemia are critical because prompt correction can completely reverse both the electrocardiographic abnormalities and the arrhythmic substrate.

Department and Institution Where Work Was Done

Department of Cardiovascular Medicine, Nippon Medical School Hospital, Tokyo, Japan.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Disclosure

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) for English-language editing and improvement of readability. The authors reviewed and edited all AI-assisted content and take full responsibility for the final manuscript.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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