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CASE REPORT

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A penetrating aortic ulcer rapidly evolving into aortic dissection in a patient presenting with respiratory tract infection to the emergency department: an acute aortic syndrome case report

Victoria Al Karaki[1*] and Aed Saab[1]

Abstract

Background Penetrating aortic ulcers (PAU) are life-threatening conditions which derive from severely advanced atherosclerotic lesions of the aorta. The clinical course is unpredictable; thus clinical vigilance should be maintained. It is very challenging to separate PAU from co-existing AAS as predisposing factors and findings overlap.

Case presentation Case of 58-year-old gentleman, who presented for atypical chest pain in the setting of respiratory tract infection. Computed Tomographic angiography (CTA) of the chest showed a large PAU and intramural hematoma which rapidly progressed into an acute aortic dissection in the emergency department. Close follow up with cardiac point of care ultrasound one hour later detected an intimal flap which was not initially present on CTA. Patient underwent surgical aortic graft replacement and had an uneventful in-hospital stay.

Discussion This case underlines the importance of broadening differential diagnoses in atypical presentations in patients with risk factors. Prompt intervention and careful management are imperative to optimize patient outcomes and prevent complications of aortic lesions. Cardiac point of care ultrasound can help in detecting progression of dynamic atherosclerotic diseases such as acute aortic syndrome.

Keywords Penetrating atherosclerotic ulcer, Aortic dissection, Acute aortic syndrome, Intramural hematoma, Aortic lesion, Emergency medicine

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Introduction

Penetrating aortic ulcers (PAU) are life-threatening conditions which derive from severely advanced atherosclerotic lesions of the aorta [1]. Aortic lesions start at the level of the internal elastic lamina and might progress to multiple complications like pseudo-aneurysms, dissections, intramural hematomas and even aortic thrombus and ruptures [1, 2]. The clinical course is unpredictable; thus clinical vigilance should be maintained.

Acute aortic syndrome (AAS) is an uncommon but fatal condition [3]. It includes three pathologies: aortic ulcer, intramural hematoma and acute aortic dissection (AAD) being the most common (70% of all AAS) and lethal one [4, 5]. PAU forms 2 to 7% of AAS cases especially in patients with hypertension and coronary artery diseases [5]. It is very challenging to separate PAU from co-existing AAS as predisposing factors and findings overlap.

We describe a case of PAU which rapidly progressed into an acute aortic dissection in the emergency department. The ulcer was initially seen on CT angiography of the chest with an intramural hematoma and close follow up with cardiac point of care ultrasound (POCUS) one hour later detected an intimal flap which was not initially present.

Case presentation

History of presentation

A 58-year-old gentleman, non-smoker, with history of hypertension on losartan 50 mg daily, presented for vague atypical central chest pain in the setting of 2 weeks of a respiratory tract infection. Patient reported on presentation a persistent dry cough exacerbated by activity and associated with shortness of breath, dizziness and diaphoresis which improve after rest. Few hours before presentation, the patient had worsening of his shortness of breath and decided to seek care in the emergency department. In the ED, he reported localized retrosternal chest pain of 2 h duration with a severity of 6/10, waxing and waning, pleuritic and ripping in nature. He had finished a course of Levofloxacin 500 mg for a week with mild improvement. Family history is positive for ascending aortic dissection in his mother. Patient denies any recent trauma, substance abuse or previous cardiac history or interventions.

Assessment

the patient's vitals revealed no fever, a blood pressure of 144/85 mmHg, a heart rate of 118 beats per minute, a respiratory rate of 20 per minute and an oxygen saturation of 97% on room air. His physical exam was unremarkable. Patient had normal heart sounds, equal pulses in all 4 extremities, and clear lung exam. He is awake, alert, oriented and cooperative in the ED without any focal deficit. Patient did not show any signs of respiratory distress during his stay and did not require oxygen.

Investigations

On EKG patient was in normal sinus rhythm, he had flattening of T waves in leads II, III, V4, V5 and V6. Laboratory investigations including a full blood count showed no significant abnormalities except mild leukocytosis of 11.700/cu.mm, hemoglobin 13.6 g/dl, chemistries unremarkable, CRP 42.6 mg/L, 2 sets troponin normal (0.010 ng/mL), pro-BNP 115 pg/mL, INR 1.3 and D-Dimer levels 760 ng/mL. Chest XR PA + Lateral was negative for large consolidation, pleural effusion, pneumothorax, wide mediastinum; the cardiac silhouette was unremarkable, it demonstrated bilateral peri-bronchial cuffing

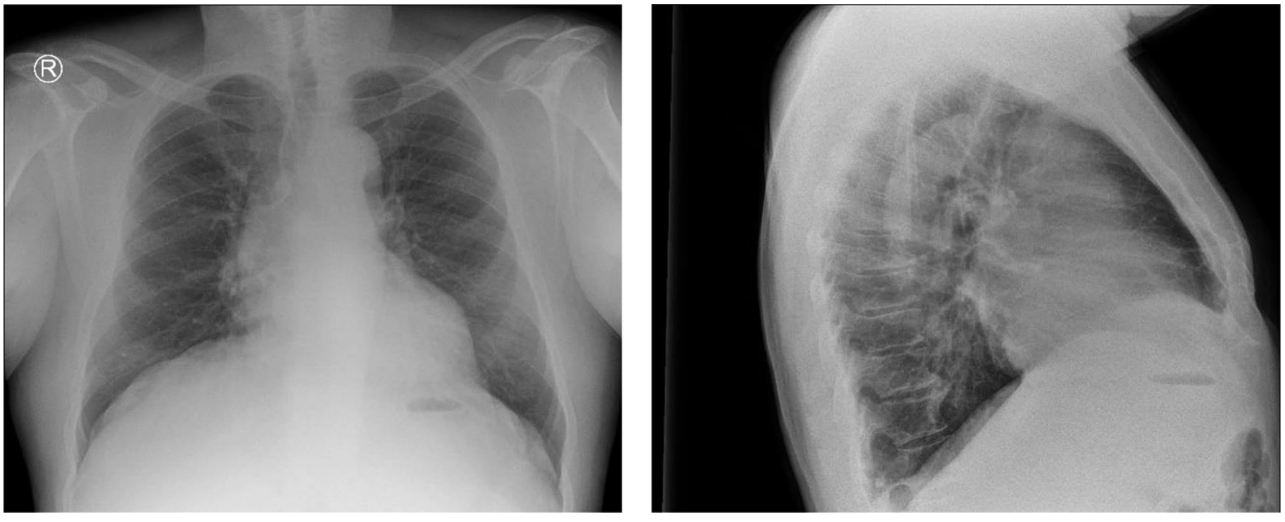

Fig. 1 Chest XR PA + Lateral

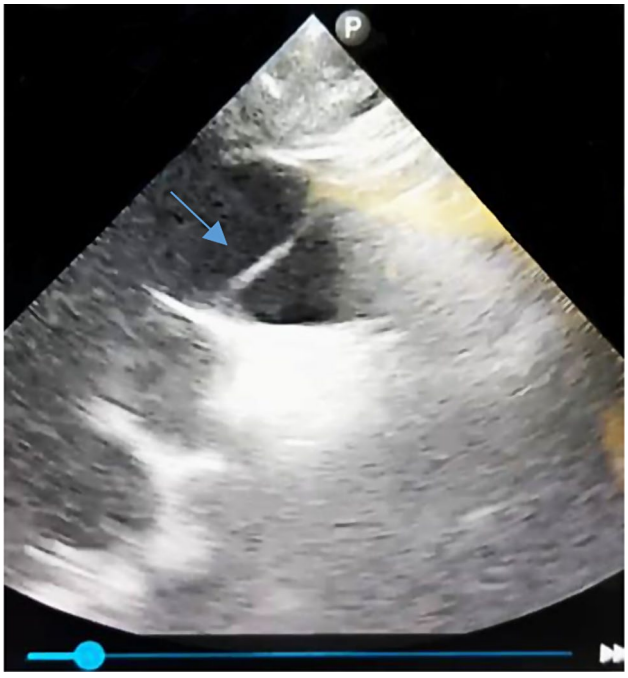

Fig. 2 Suprasternal notch view of cardiac POCUS showing an intimal flap at the level of the aortic arch

suggestive of bronchitis (Fig. 1). Influenza and COVID studies were negative

As part of the initial assessment of patient coming to the ED with chest pain and shortness of breath, a Point of cardiac US was performed at bedside to assess for cardiac status, B-lines suggestive of pulmonary edema, any valve abnormalities showed only a moderate pericardial effusion. Initially no flap was identified on cardiac US substernal notch views, aortic valve is normal and ejection fraction was preserved around 55–60% with good ventricular function.

As the patient was still complaining of pleuritic and ripping chest pain after taking IV acetaminophen for pain, the team decided to proceed with CT angiography of the chest to look for pneumonia or pulmonary embolism as differential diagnoses given that he has elevated D-Dimer levels of 760 ng/mL positive adjusted to his age. The CT angiography of the chest came back positive for a large penetrating aortic atherosclerotic ulcer at the level of the arch with intramural hematoma at the level of the proximal ascending aorta.

Pre and post contrast axial CT images of the chest in the arterial and venous phases with multiplanar reformats showed a large contrast filled intramural hematoma with pseudo-aneurysm formation, showing a wide neck, sequelae of underlying penetrating aortic atherosclerotic ulcer with shearing of the intimal lining, occupying the medial wall of the mid and upper segments of the ascending aorta reaching the aortic arch (Figs. 2 and 3). A single fenestration is identified communicating with the lumen. No definite entry and reentry points of a dissection flap could be identified. There was also associated crescent shaped eccentric hyperattenuation at the level of the ascending aorta laterally seen on noncontrast imaging, measuring approximately 1.5 cm, suggestive of periaortic hematoma (Figs. 4 and 5). There is mild cardiomegaly. There is a moderate pericardial effusion. The aortic root and coronary arteries and branches of the aortic arch are patent. No extension into branch vessels. The visualized abdominal aorta and its major branches are otherwise patent.

One hour after the CT, the patient started to become more tachycardic with a heart rate ranging between 115

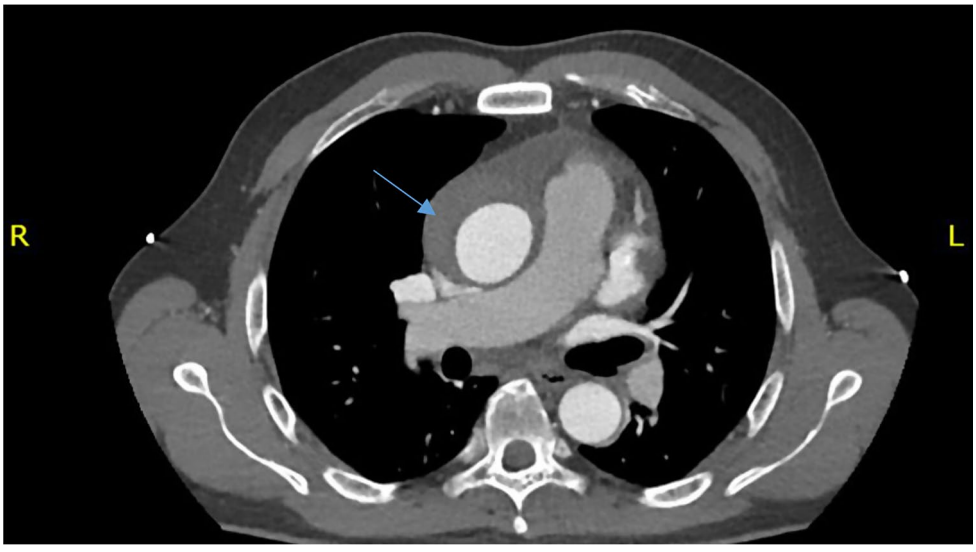

Fig. 3 Axial view CTA chest showing aortic intramural hematoma

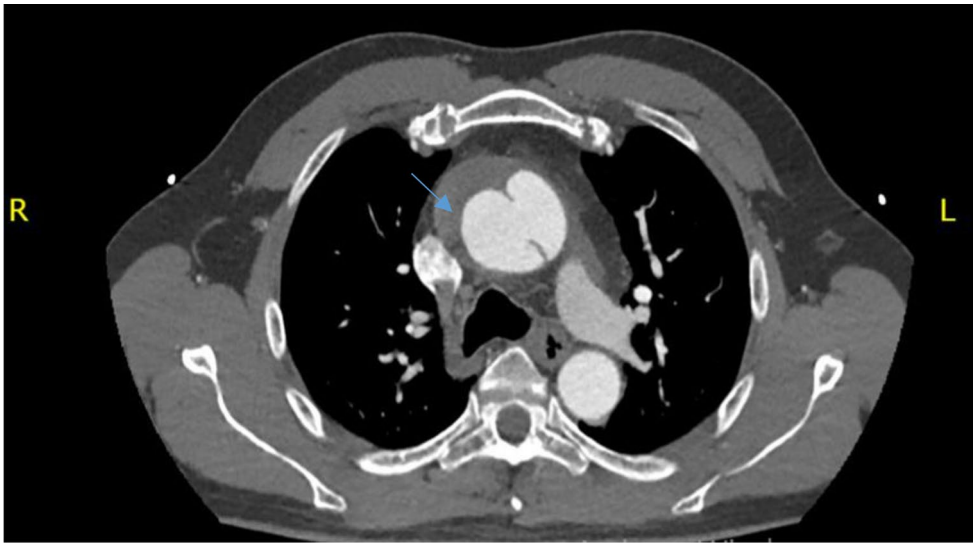

Fig. 4 Axial view CTA chest showing PAU and intramural hematoma

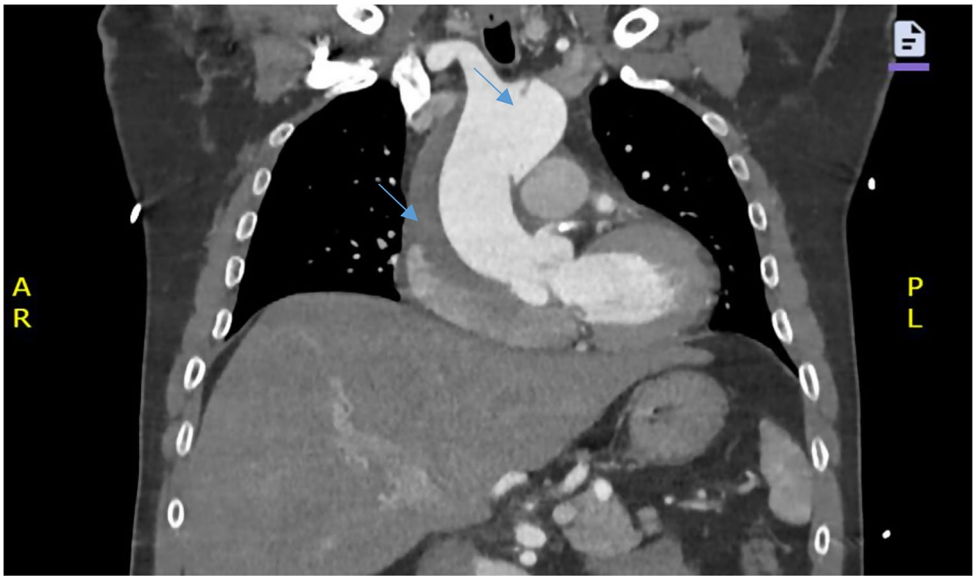

Fig. 5 Coronal view CTA chest showing PAU and intramural hematoma

and 135 beats per minute and his pain was still present, his systolic blood pressure was still on the high side ranging from 145 to 170 mmHg and since he had the finding of penetrating aortic ulcer on CT scan the ED team decided to proceed with a repeat of the bedside cardiac POCUS to assess for progression of his disease in the setting of persistent high blood pressure readings. The repeat cardiac POCUS revealed an expanding intramural hematoma with an intimal flap seen at the level of the arch with trivial aortic insufficiency but with a preserved ejection fraction (Fig. 6).

Management

Patient was given pain medication and labetalol 20 mg three times to target a systolic blood pressure between 100 and 110 mmHg and was taken to the operating room for urgent aortic graft replacement. For bronchitis, patient was given a dose of Ceftriaxone 2 g intravenously and azithromycin 500 mg orally with one dose of nebulizers.

Intraoperatively the patient received 4 PRBC 2 in bypass and 2 after with 2.5 L of Lactate Ringer. Hb remained around 11 g/dl. Pathology revealed an arteriosclerosis with dissecting plaque-like material within the aortic wall; a periaortic acute and chronic inflammation were seen and large blood clots were present.

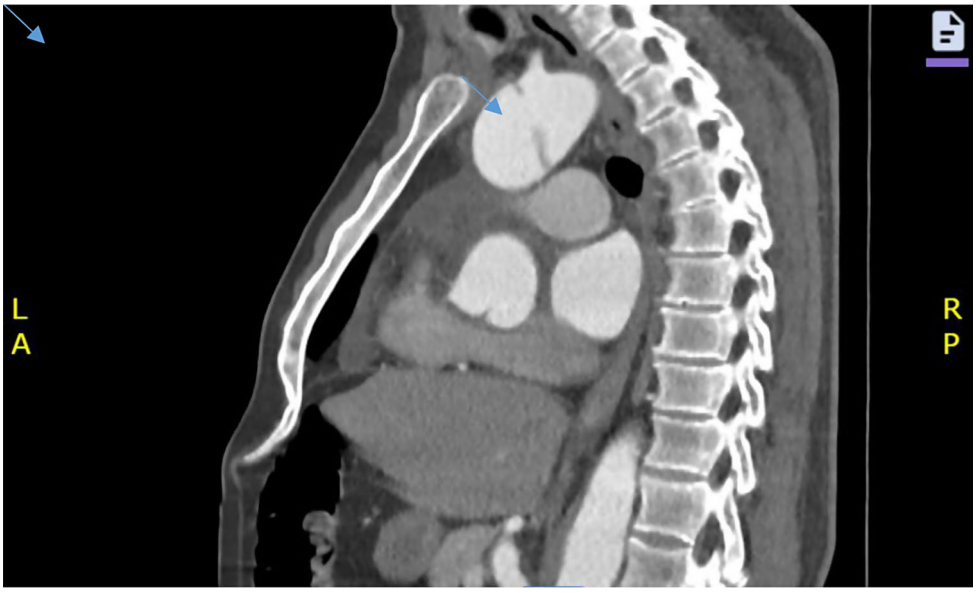

Fig. 6 Sagittal view CTA chest showing PAU

After the OR, patient was transferred to the cardiac surgical unit intubated, clinically and hemodynamically stable with good urine output. Patient was extubated softly without any complication and directly attachment to nasal cannula 12 h postop. He was discharged 4 days later on Bisoprolol 2.5 mg twice daily and losartan 50 mg twice daily. He had an uneventful in-hospital stay. No anticoagulation or antiplatelet agents were prescribed.

Follow-up

2 months later, a lower limb duplex ultrasound showed partial deep vein thrombosis in the left distal popliteal vein with small intramuscular collection in the right calf and possible small hematoma due to muscle tear. Patient was started on Apixaban 5 mg twice daily for 3 months.

Discussion

Deep PAU are very rare pathologies of the aorta, emerging from micro-perforations of its internal intima layer due to advanced age and arteriosclerotic diseases [6]. Intimal fibrosis of the ulcer facilitates leakage of blood into the lumen and formation of intramural hematomas [6]. Intense wall stress from hypertension, intima breaches and ulcerations promotes expansion of the hematoma [6, 7]. Once the medial layer of the vessels is penetrated by atheromatous plaques, extensive aortic pulsatile flow exposes the aorta to shearing forces and ultimately causing aortic dissection commonly in the mid to distal thoracic parts [6, 8]. Given this complex pathophysiology, it is important think of this condition among differential diagnoses in atypical features of chest pain [2]. Thoracic PAU had higher rate of progressing into IMH and dissection than abdominal PAU [9]. IMH was more referred to as the hemorrhage inside the atherosclerotic plaque or the bleeding into the outer layer of the aortic media from rupture of the aortic vasa vasorum [9].

For chest pain, the first imaging modality is usually chest radiographs; which can demonstrate a pleural effusion or a wide mediastinum [10]. CT angiography of the chest remains the gold standard diagnostic study [11]. In PAU, the image illustrates in general a hypodensity within the aortic wall in comparison to the contrast medium images and in case of dissection or impending rupture, contrast extravasation might be noticed [12]. Transthoracic cardiac echography plays a role in detecting proximal dissections and its complications [5]. This modality can give a diagnosis in 15 min; nevertheless, it is operator dependent and rely on high skillsets [5]. Transesophageal echocardiography might show better images due to close proximity with the aorta, but is an invasive procedure and not readily available in emergency departments [5]. In our case, cardiac POCUS was used to monitor progression of PAU into aortic dissection and this modality helped us expedite care and rush the patient to the operating room. Management of stable PAU remains medical as the initial standard of care unless there is evidence of progression of a dissection, intractable pain, blood outside the aorta and organ malperfusion [13]. If any is present, surgical management becomes imperative and POCUS played a major role in detecting the evidence of PAU progressing into a dissection and expedited the acute care.

Definitive treatment of dissections in the ascending segments of the aorta is surgical [5]. Intervention should be fast to prevent progression of the dissection causing rupture, limb malperfusion or spinal ischemia [5]. Timing

is key as patients who underwent early interventions, had better outcomes and certainly a lower rate of complications [5]. Use of beta-blockers to decrease the systolic blood pressures and reduce the aortic wall stress remains a mainstay [5].

The most common complications seen after aortic dissection repairs are vascular in general. Given the major risk of re-bleeding post-operatively, administration of blood thinners after the surgery is always debatable and depends on surgeon assessment and practice. Nevertheless, providers should always schedule regular follow-ups to assess for vascular complications such as deep vein thrombosis, arterial thrombosis, mesenteric ischemia, aorto-enteric fistula and vascular strokes due to hypoperfusion and hypercoagulable states [5]. Long term outcomes and quality of life of patients depend on detecting these complications early on before they become extensive and cause disabilities. As we see, our patient had a regular scheduled follow-up which helped detecting early on a deep vein thrombosis requiring anticoagulation to prevent further extension into pulmonary embolism.

Another risk factor that we would like to highlight is the genetic predisposition for family history of aortic dissection which was positive for our patient and had increased significantly our index of suspicion and pushed investigations further. A recent case by Patel by et al. in 2024 described a similar case of a patient who was diagnosed with aortic dissection in an undiagnosed genetic syndrome [14]. In literature many disorders have been already studies like Marfan and Ehlers-Danlos syndromes [14]. 37 genes have been identified to predispose to thoracic aneurysms dissections [14, 15]. Another novel gene TGFβR2 splice variant was reported in literature in a case of 31 year-old patient with family history of aortic dissection as well [16]. In addition, a pathogenic variant in the EFEMP2 gene was as well identified in 2024, investigated in an Iranian family and highlighted the dominant pattern of inheriting thoracic aortic aneurysm with high probabilities of dissection and rupture [17]. Based on these data, we shed light on the importance of conducting genetic tests in at-risk families especially first-degree relatives and perform scheduled cardiac monitoring of the aorta with echocardiograms, Computed Tomography and ultrasounds.

Upper and lower respiratory tract infections are common causes of chest pain. However, we were fortunate to push further the investigation in this uncommon worsening presentation of an atypical chest pain and patient obtained a good outcome. Therefore, we highlight the importance of lowering the index of suspicion in patients with family history of aortic dissection and atherosclerosis risk factors such as hypertension, diabetes, hyperlipidemia, smoking and advanced age. At the same time, we emphasize the advantage of cardiac POCUS in following up closely on stable PAU that can turn into unstable aortic dissections at any moment.

Conclusion

Clinicians should be vigilant to rapidly growing complications of penetrating aortic ulcers. This case underlines the importance of broadening differential diagnoses in atypical presentations. Prompt intervention and careful management are imperative to optimize patient outcomes in aortic lesions. Point of care ultrasound can help in detecting progression of dynamic atherosclerotic diseases such as acute aortic syndrome.

Abbreviations

PAU	Penetrating aortic ulcer
AAS	Acute aortic syndrome
AAD	Acute aortic dissection
POCUS	Point of care ultrasound
CTA	Computed tomographic angiography
PRBC	Packed Red Blood Cells

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Nothing to declare.

Author contributions

V.A.K was involved in patient management, designed, and wrote the main manuscript text and prepared the figures. A.S. revised the manuscript. All authors revised the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Consent to participate

Written consent was obtained from the participant to write the article.

Consent for publication

Written informed consent was obtained from the patient for his anonymized information to be published in this article.

Competing interests

The authors declare no competing interests.

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