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Case Report

A Case Report of the Unusual Presentation of the Left and Then the Right Spontaneous Carotid Artery Dissections Within 3 Weeks and a 7-Year Follow-Up

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Abstract

Aims/Background: Bilateral spontaneous carotid artery dissections are rare, comprising only 10% of spontaneous carotid artery dissection cases. They occur when there is a tear in the innermost lining of the artery called the tunica intima. An intramural hematoma may form which can lead to vessel lumen narrowing or occlusion, causing an acute ischaemic stroke. **Case Presentation**: We present a case of a man in his 50s with bilateral spontaneous internal carotid artery dissection who presented with a sudden onset of headache, neck pain, and right arm weakness. After being treated, the patient presented again after 3 weeks with neck pain on the right side, headache, and transient left arm weakness. He was treated with anticoagulation followed by antiplatelet therapy. **Results**: At 6 months, the computed tomography angiogram (CTA) of carotid and vertebral arteries showed a largely resolved left internal carotid artery dissection (ICAD) and a resolved right ICAD. At the 7 years follow up, the patient continues to do well as he has not had any more transient ischaemic attacks or strokes. **Conclusion**: This case demonstrates that carotid artery dissections should be considered when a patient presents with these symptoms, even if they present bilaterally in a short period, and can be investigated with a CTA or magnetic resonance angiogram, with treatment by antiplatelets or anticoagulation.

Keywords: carotid artery dissection; artery dissection; vertebral; stroke; angiography; case report

1. Introduction

This case report describes a case of bilateral spontaneous carotid artery dissection (CAD). CADs happen annually in 2.6/100,000 people, with about 2–10% of these cases being bilateral CADs, so it is much rarer [1]. It is noted that more than 90% of CAD cases are spontaneous, and of these, 10% are spontaneous bilateral CAD cases [2,3]. In these patients, there is a tear in the innermost coat of the artery, and there is secondary intramural hematoma formation, which can potentially lead to acute ischaemic stroke [2]. Internal carotid artery dissections (ICADs) can cause arterial stenosis, occlusion, or a dissecting pseudoaneurysm [4]. After conducting a literature search, we have found a case where a patient was diagnosed with left ICAD and, three years later, they were found to have a new right ICAD [5]. However, to the best of our knowledge, we present for the first time a case with an unusually short time of three weeks between the spontaneous left CAD and the spontaneous right CAD. Patients with CADs commonly present with headache or neck pain, and can also have clinical manifestations such as cerebral ischemia and pulsatile tinnitus [2,6].

2. Case Report

A man in his 50s first presented to the hospital with a sudden onset of headache, neck pain, and right arm weakness. He usually does not suffer from headaches, and no preceding trauma was reported. His modified Rankin scale (MRS) [7] was 0 (MRS scale 0 to 6). MRS 0 implies independence with no symptoms at all. The MRS increases as dependency and disability increase, with the score of MRS 6 given for death. He had hypertension and takes Ramipril 2.5 mg. The relevant history was his borderline hypertension, as well as the patient reporting a habit of "cracking his neck". His neurological examination revealed 4/5 in his right arm and otherwise normal neurology. This gave him a National Institutes of Health Stroke Scale (NIHSS) [8] score of 1 (NIHSS score 0–42). NIHSS of 1 reflected his mild right arm weakness. NIHSS 0 implies no signs, and NIHSS of 15 or above signifies moderate to severe stroke. His cardiovascular, respiratory, and abdominal system examinations were normal. His right arm weakness resolved after a few hours.

Investigations showed that his routine blood tests were normal. Non-contrast computed tomography (CT) head showed no ischaemia or haemorrhage. Computed tomography angiogram of carotid and vertebral arteries (CTACVA)

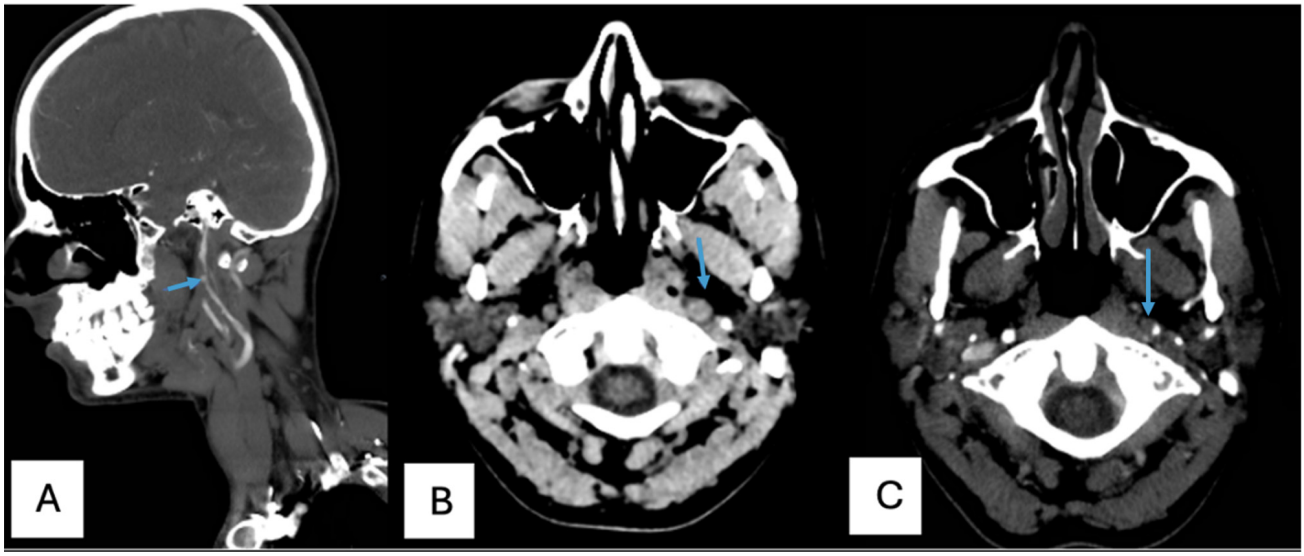

Fig. 1. Computed tomography (CT) angiogram and the CT of the head showing the left internal carotid artery dissection. (A) Sagittal reconstruction of the CT angiogram showing the left internal carotid artery dissection (blue arrow). (B) Unenhanced CT of the head at skull base showing the left internal carotid artery dissection flap (blue arrow). (C) Axial CT angiogram showing the left internal carotid artery dissection with filling defect (blue arrow).

showed left ICAD with a free-floating thrombus (Fig. 1). Other vessels were unremarkable.

He was anticoagulated with Warfarin and discharged a few days after.

However, the patient presented again with neck pain on the right side, headache, and transient left arm weakness 3 weeks after his first presentation. There was no preceding trauma, and he was compliant with the medications prescribed on recent discharge. On examination, he had normal neurological, cardiovascular, respiratory and abdominal examinations. His NIHSS score was 0, implying no neurology.

In his second presentation, non-contrast CT of the head showed no ischaemia or haemorrhage. CTACVA showed right ICAD and a stable left ICAD (Fig. 2). Magnetic resonance imaging (MRI) brain showed no acute infarct or haemorrhage (Fig. 3).

A urine toxicology screen was negative, which was done to rule out Cocaine-induced vasospasm. Tests were done to investigate if there were any underlying conditions that may have predisposed the patient to the bilateral CAD. We tested for the autoimmune conditions antiphospholipid syndrome and vasculitides, but they were negative for Antiphospholipid antibodies, anti-neutrophil cytoplasmic antibody, antinuclear antibodies, extractable nuclear antigen, rheumatoid factor, and erythrocyte sedimentation rate, so these were ruled out.

3. Treatment

This patient was treated with anticoagulation for one year with Warfarin. This was considered ideal because of the bilateral nature of the dissections and the presence of a free-floating thrombus, which posed a thromboembolic risk. After the first year, the patient was given antiplatelet therapy with Clopidogrel 75 mg, ongoing. The initial decision to anticoagulate with Warfarin and subsequent switch to Clopidogrel was influenced by the treating clinician's experience in this field. As we discuss below, the studies showed that there is no difference in outcome in treating dissections with anticoagulation or antiplatelets.

4. Outcome and Follow-Up

The patient was followed up at 6 months with further investigations and serial CTACVAs. Genetic testing was done to identify the collagenous conditions Ehlers-Danlos Syndrome, Marfan's disease, and Collagen vascular disease, these came back as negative. The structural conditions autosomal polycystic kidney disease, which was tested for with Renal Ultrasound, and Fibromuscular dysplasia, tested for by CT angiogram, came as negative. These tests led to the ruling out of these risk factors, and to date, there has been no identified cause for his bilateral CAD except his hypertension.

CTACVA at 6 months showed largely resolved left ICAD with a residual 5 mm pseudoaneurysm at the distal internal carotid artery just below the skull base, resolved right ICAD and stable vertebral appearances (Fig. 4). CTACVA at 2 years (Fig. 5) and subsequent 2-yearly CTACVAs showed stable appearance of vessels with no new findings.

At the 7-year follow-up, he is continuing to do well without any further transient ischaemic attacks or strokes since his initial admission.

The Care Checklist has been attached as **Supplementary material** associated with this article.

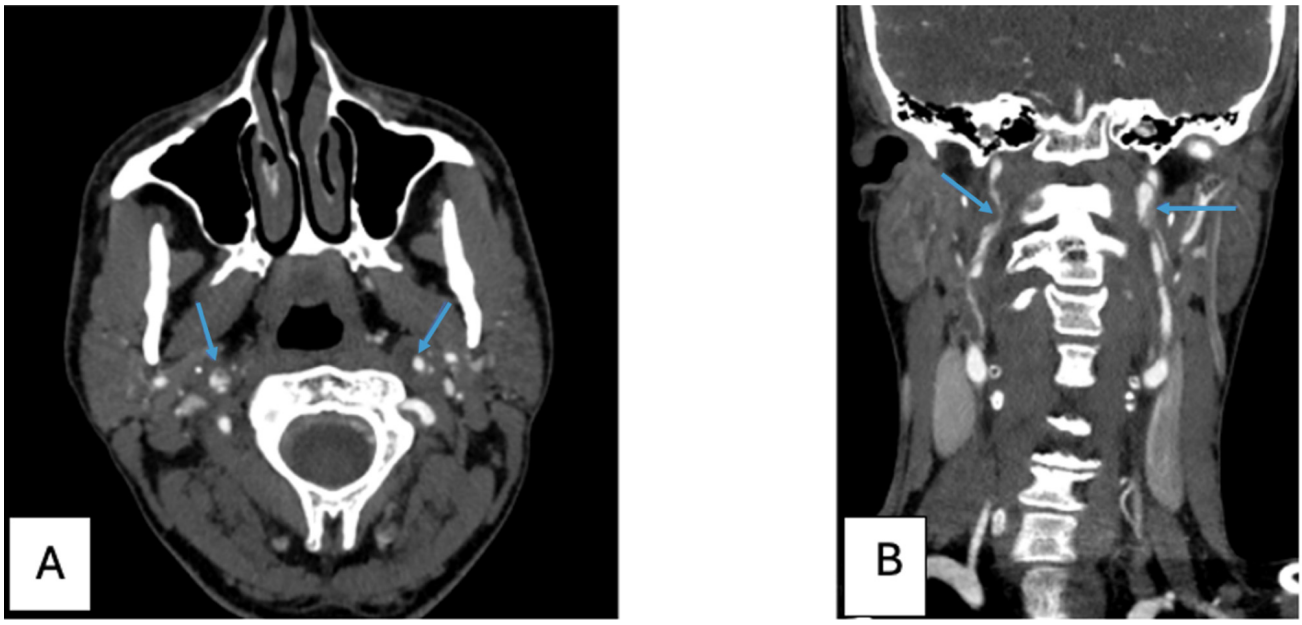

Fig. 2. CT angiogram showing the new right internal carotid artery dissection. (A) Axial CT angiogram showing new right internal carotid artery dissection (right blue arrow) and relatively unchanged left internal carotid artery dissection (left blue arrow). (B) Coronal view of the CT angiogram showing new right internal carotid artery dissection (right blue arrow) and relatively unchanged left internal carotid artery dissection (left blue arrow).

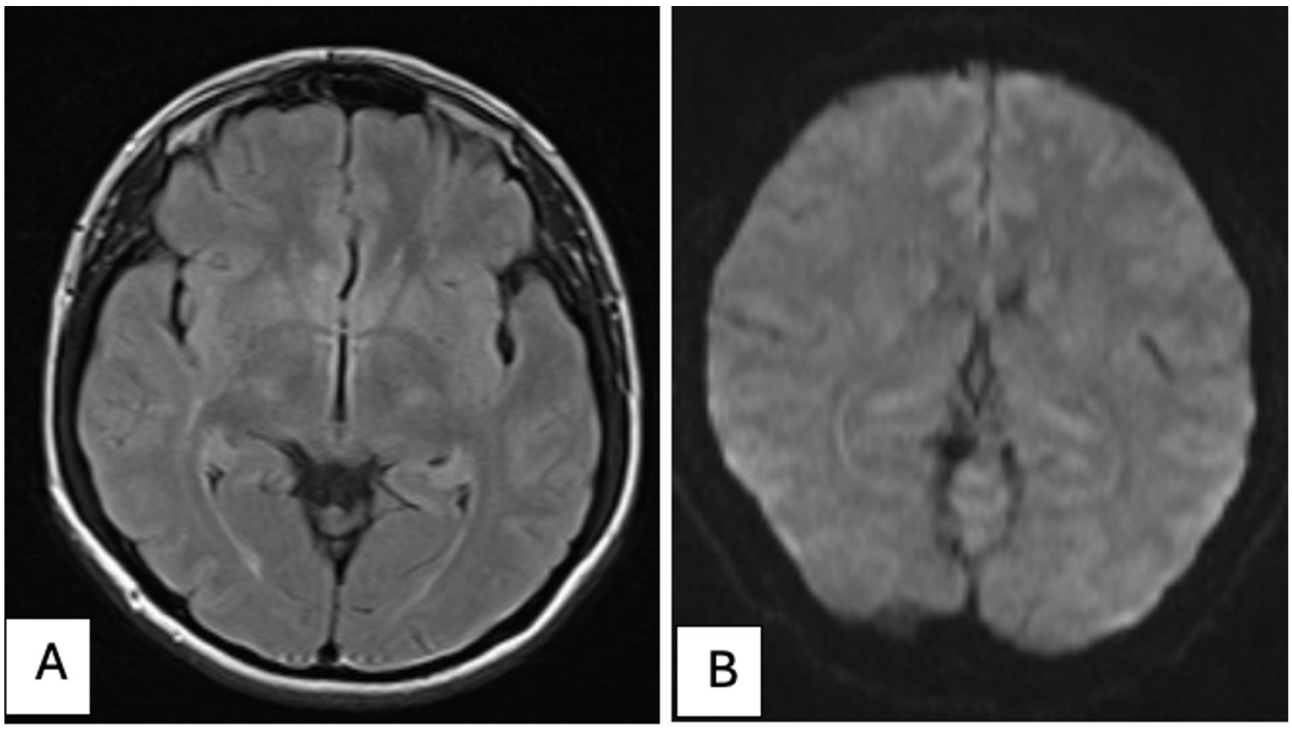

Fig. 3. Magnetic resonance imaging (MRI) brain images. (A) MRI brain T2 image showing a normal brain with no infarct or haemorrhage. (B) MRI brain diffusion-weighted image showing no acute infarct.

5. Discussion

There are three layers of walls in carotid arteries, which are the tunica intima, the tunica media, and the tunica adventitia. Therefore, the mechanism of CAD is when there is a tear in the innermost layer—tunica intima—and there is bleeding into the spaces between the other layers, and that can lead to vessel lumen narrowing or occlusion. This is why ICAD can cause severe acute ischemic stroke [1,2].

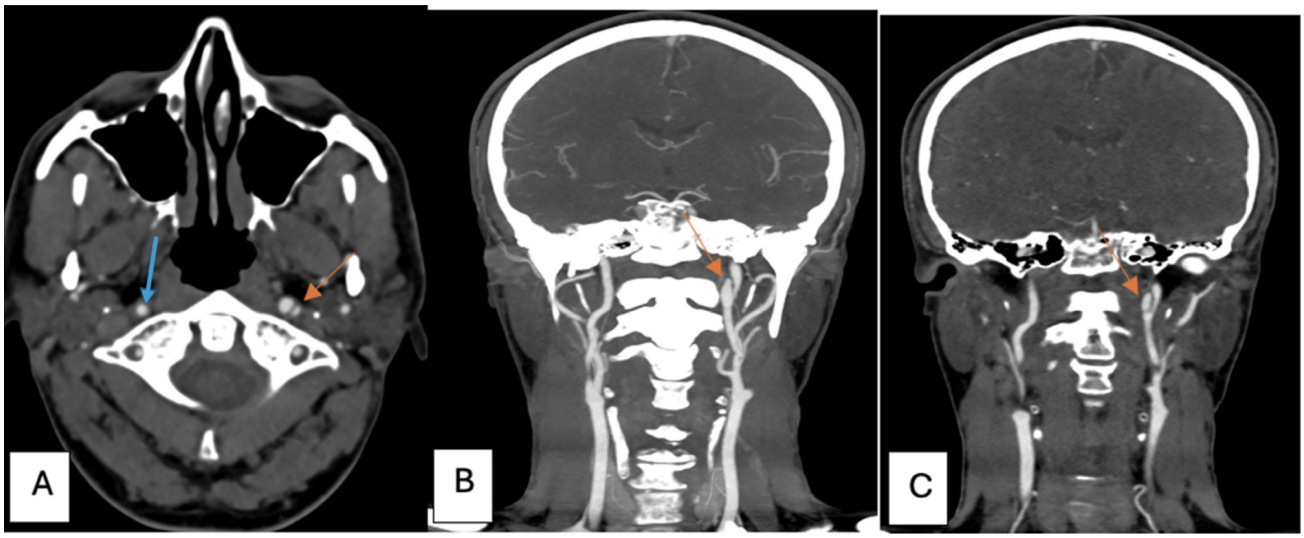

Fig. 4. CT angiogram at 6 months showing the resolution of the right internal carotid artery (ICA) and the pseudoaneurysm of the left ICA. (A) Axial CT angiogram showing the resolution of the right ICA (blue arrow) and the 5 mm pseudoaneurysm of the left ICA (orange arrow). (B,C) Coronal CT angiograms showing the 5 mm pseudoaneurysm of the left ICA (orange arrows).

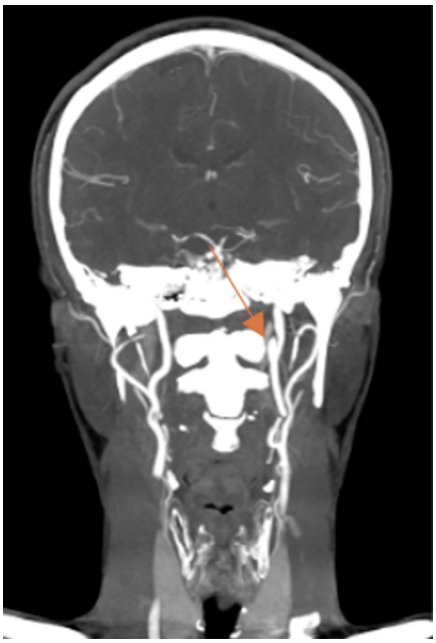

Fig. 5. Coronal CT angiogram at 2 years showing the stable appearances of the right and left ICAs. Persistent stable 5 mm pseudoaneurysm of the left ICA (orange arrow).

Chiropractic manipulation, coughing, sneezing, and vomiting can contribute to the development of CAD. The patient in our case had a habit of 'cracking his neck', the association of this to simultaneous bilateral ICAD is seen in a case report where a man in his 30s developed bilateral ICAD following chiropractic manipulation [9]. In a paper based on a MEDLINE literature search about ICADs published between 1966 and 2000, they inferred that a concerning causation for ICAD was chiropractic manipulation [10], and this could be due to the twisting causing trauma to the artery wall. Our patient had borderline hypertension, and there have been studies that suggest that cardiovascular risk factors, including hypertension, can increase the chance of spontaneous CAD occurring [11]. Additionally, patients with spontaneous CAD often have arterial tortuosity, with a higher amount of tortuosity being found in patients who had recurrent dissections, so this is a factor that could be investigated in these types of cases [11–14]. Other underlying conditions, such as fibromuscular dysplasia [15], Fabry disease [16] and autosomal dominant inheritance Ehlers-Danlos [17], are involved in the occurrence of CAD.

In another case of spontaneous bilateral ICAD in a man in his 40s who had a similar presentation to our patient, but additionally had an ischemic stroke due to his ICAD, endovascular stenting in the almost totally occluded left side ICA gave a good neurological recovery [18]. There have been cases of bilateral ICAD that were treated with bilateral carotid artery stenting with good outcomes [4,19].

These are examples of how endovascular treatment and antithrombotic treatment have been applied successfully in conjunction with each other, which could be an indication for future treatment pathways, providing more research is carried out on the effectiveness of carotid artery stenting.

Eagle syndrome is when the patient has an elongated styloid process, and this can compress the cervical internal carotid artery and can cause arterial dissection. There was another case where the patient had bilateral ICAD due to Eagle syndrome in a patient with vascular Ehlers-Danlos Syndrome (EDS), and the treatment path was difficult to determine because cases where the dissection is caused by Eagle syndrome are treated surgically by resection of the elongated styloid process or carotid artery stenting. However, in patients with EDS, surgery is not preferred due to the vascular vulnerability. This patient was treated with aspirin [17].

In our experience, we found four key outcomes of ICAD: fully healing, narrowing, occlusion and pseudoaneurysm; such outcomes dictated our short- and long-term management approach.

When a patient has ICAD, they are most commonly treated with an antithrombotic treatment—consisting of either anticoagulation, single antiplatelet treatment, or dual antiplatelet [2,20–22]—or they are treated with endovascular treatment such as carotid artery stent implantation [23,24]. The Cervical Artery Dissection in Stroke Study Trial stated that antiplatelet and anticoagulation treatment are equally effective in the prevention of strokes [22]. A systematic review and meta-analysis showed anticoagulation was superior to antiplatelet therapy in reducing ischemic stroke but carried a higher major bleeding risk [25]. In patients with CAD, endovascular treatment may be utilised if the patient has recurrent ischemic strokes, transient ischemic attacks or if they have progressive luminal narrowing [21]. It is suggested that in ICAD patients whose neurological symptoms have not improved with antithrombotic therapy, they may respond to endovascular stent implantation, however, the long-term durability of this treatment requires more research [26]. Due to the lack of long-term follow-up study data of carotid artery stenting, these are used in treatment with caution, and instead results of the Cervical Artery Dissection in Stroke Study recommend that for ICAD patients, 3–6 months of antiplatelet or anticoagulation therapy can be utilised as they produce similar results in their reduction of major bleeding, mortality, and stroke recurrence [22]. European stroke organisation guidelines [27] suggested given the overwhelming evidence of a very low rate of recurrent ischemic events in post-acute ICAD patients under medical treatment and the lack of evidence for an impact of residual stenosis or dissecting aneurysms on the rate of these events, all but one expert suggest against routine use of endovascular/surgical treatment in these patients: based on current limited evidence, endovascular/surgical treatment of post-acute ICAD may be carefully considered in exceptional situations, such as recurrent ischemic events despite optimal antithrombotic therapy or expanding dissecting aneurysms causing compression, after assessment by a multidisciplinary team [27].

6. Conclusion

In patients presenting with new onset of headaches and neck pain with or without neurological symptoms, carotid or vertebral artery dissections should be considered as a differential diagnosis. When spontaneous CADs occur, it is crucial to consider the underlying cause and investigate appropriately, as they could be related to a range of issues such as hypertension, chiropractic manipulation, and connective tissue disorders such as Ehlers-Danlos Syndrome. The gold standard investigations for detecting dissections are CT angiogram or MR angiogram of the carotid and vertebral arteries, and these patients can either be treated with antiplatelets or anticoagulation.

Learning Points

- Spontaneous bilateral carotid artery dissections can occur consecutively, with one side affected first, followed by the opposite side within a few weeks, which was a unique feature of this case, therefore, clinicians should note this as a possibility when faced with similar presentations.
- Long-term follow-up of bilateral carotid artery dissections was also a unique feature of this case, which can be used as a reference to clinicians.
- There are many aetiologies of spontaneous carotid artery dissections which were discussed, and specific investigations are required to rule out each cause.
- Chiropractic manipulation can cause damage to the artery walls and is, therefore, important to consider in cases of carotid artery dissections.
- Carotid artery dissections should be treated with either anticoagulation or antiplatelets, in the first instance, with no differences in outcome between the two.

Availability of Data and Materials

All the data of this study are included in this article.

Author Contributions

AS and VR designed the work. AS identified the case and was the principal clinician for the patient. AS collected the data for the case. AS annotated the images. AS analysed

and interpreted the data. VR wrote the first draft of the case report. Both authors made principal editorial changes to the manuscript. Both authors participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Written informed consent was obtained from the patient for publication. This research was completed in accordance with the Declaration of Helsinki.

Acknowledgement

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/BJHM50186.

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