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CASE REPORT

Popliteal Venous Aneurysm and Pulmonary Embolism Initially Presenting with Recurrent Pre-syncope: A Case Report

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Introduction: Popliteal venous aneurysm (PVA) can lead to recurrent pulmonary embolism (PE) and therefore necessitates prompt diagnosis and treatment. PVAs are often asymptomatic, and their most common symptoms are associated with thrombosis. The clinical presentation of PVAs varies from asymptomatic to PE induced cardiopulmonary arrest, but there are few reports of cases initially presenting with transient impairment of consciousness.

Report: A 75 year old man was referred with recurrent episodes of pre-syncope. He had normal vital signs and oxygen saturations, and his electrocardiogram was normal. Detailed interview revealed that the patient had suffered from calf pain and swelling before visiting the clinic. Therefore, an evaluation for deep venous thrombosis and PE was conducted. Lower limb ultrasound revealed an enlarged popliteal vein, measuring 20 mm in diameter, with a spontaneous echo contrast. Enhanced computed tomography showed peripheral pulmonary artery embolism. The patient was diagnosed with PE secondary to PVA. An inferior vena cava filter was inserted, followed by tangential aneurysmectomy and lateral venorrhaphy; apixaban 10 mg/day was initiated on post-operative day 1. The filter was removed one week after the surgery, and the patient remained symptom free on completion of treatment and did not complain of any symptoms such as pre-syncope.

Discussion: This patient with PVA presented with the initial symptoms of repeated pre-syncopal episodes that were attributed to recurrent PE caused by thrombi from a PVA. Complete symptom resolution was obtained by inferior vena cava filter placement, PVA surgery, and post-operative anticoagulation. Transient consciousness disorders such as pre-syncope can be the initial symptoms of PVA and PE.

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INTRODUCTION

Popliteal venous aneurysm (PVA) is a rare pathology that can lead to recurrent pulmonary embolism (PE) and therefore necessitates prompt diagnosis and treatment.[1] PVAs are often asymptomatic, and, if present, the most common symptoms include those associated with thrombosis. Some patients present with cardiac arrest secondary to PE.[2,3] Although the presentation varies, there have been very few reports in the literature of PVA and PE initially presenting with transient impairment of consciousness. Herein a case in which recurrent pre-syncope was an initial symptom of PE and PVA.

CASE REPORT

A 75 year old man was referred to the outpatient clinic from a rural emergency department for further evaluation of recurrent pre-syncope. Symptoms included light headedness, sweating, visual disturbances, weakness, and impaired consciousness. He also had trouble standing up and was taken to a rural emergency outpatient clinic where he was managed conservatively by observation and experienced a complete recovery. Routine physical examination revealed no cause for the symptoms which had occurred approximately once a month. Prior to visiting the clinic, dysrhythmia and neurological disease were ruled out based on the Holter electrocardiogram and the head computed tomography (CT) and magnetic resonance imaging scans.

In the outpatient clinic, vital signs were within the normal range; blood oxygenation was normal (oxygen saturation 95% on the room air), and there were no signs of lower limb oedema indicating heart failure. Neurological findings were normal, and there were no signs of pulmonary hypertension, valvular disease, or heart failure on echocardiography.

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Further examination revealed that the patient had been experiencing left calf oedema and pain almost once a month, which was not associated with the pre-syncopal episodes.

Further evaluation of deep vein thrombosis (DVT) and pulmonary embolism (PE) was required. Lower limb ultrasound revealed saccular dilatation of the left popliteal vein 20 mm in diameter, which was subsequently diagnosed as left PVA (Fig. 1A,C). There was no thrombus, but spontaneous echo contrast (SEC) was observed. SEC was observed in the PVA alone, and the other veins were normal. Enhanced pulmonary CT revealed peripheral PE (Fig. 2A). A blood test showed D dimer elevation at 1.1 μg/mL. An inferior vena cava (IVC) filter was placed to prevent further PEs from the PVA. A decision was made to proceed with surgery. Thrombolysis was not initiated because the thrombus in the pulmonary artery was so small it did not affect haemodynamics or blood oxygenation. An "S" shaped skin incision was made in the popliteal space to expose the popliteal vein where a saccular PVA was seen (Fig. 3A and B). After clamping the proximal and distal popliteal veins, the PVA was incised. There was no thrombus, and the intima appeared normal on inspection (Fig. 3C). The wall was closed with a 6-0 continuous polypropylene suture with large bites to perform simultaneous venorrhaphy. Two additional venorrhaphies were performed on both lateral walls of the PVA (Fig. 3D). The diameter was decreased to the same size as the proximal and distal popliteal veins (Fig. 1B,D). Apixaban 10 mg/day was initiated one day after surgery.

One week after surgery, left lower limb ultrasound showed no venous thrombus, and the SEC in the popliteal vein had also disappeared (Fig. 1B). Angiography of the IVC revealed no major thrombus trapped by the filter. The filter was subsequently removed, and a small thrombus was observed on the filter fibres (Fig. 2B).

During the six month follow up after treatment, the patient's symptoms, including pre-syncope and left lower limb oedema, had disappeared.

DISCUSSION

PVA is rare, with a prevalence of 0.18%—0.20%,[1] and its aetiology remains unclear. McDevitt *et al.* defined PVA as an

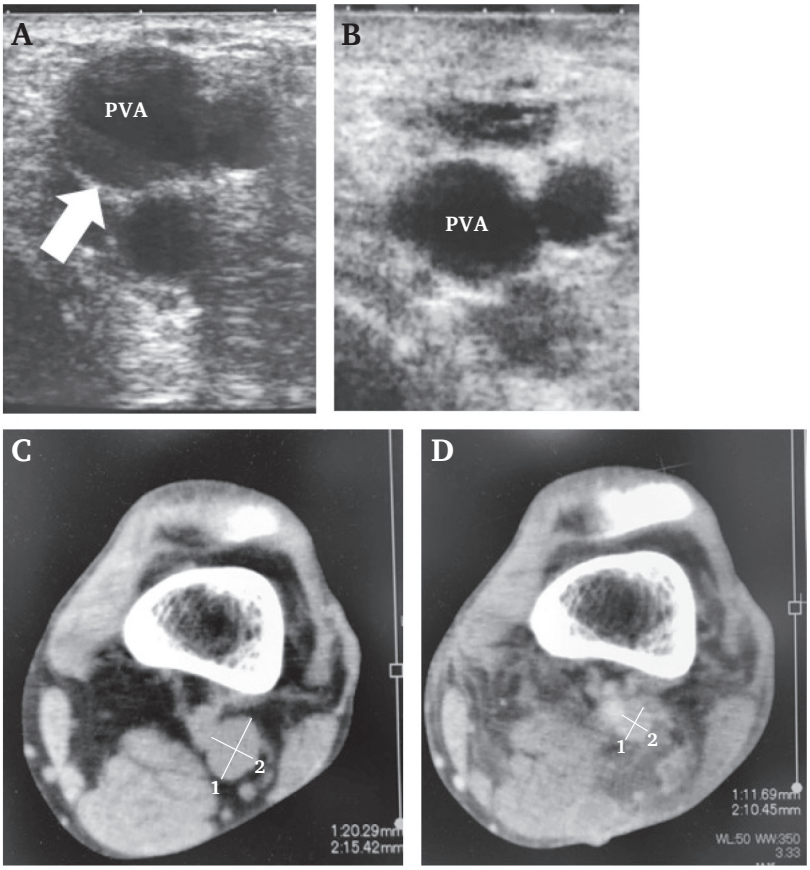

Figure 1. Images of popliteal venous aneurysm. (A) Lower extremity ultrasound in the emergency room. The ultrasound revealed a popliteal venous aneurysm and spontaneous echo contrast (SEC) (arrow mark). There was no spontaneous echo contrast other than in the popliteal venous aneurysm. (B) Ultrasound imaging after treatment. The diameter of the popliteal vein, which had been aneurysmal, had decreased. (C) Plane computed tomography (CT) image before surgery showing a 20 mm diameter saccular popliteal venous aneurysm. SEC was observed on ultrasound, but there was no sign of thrombus on ultrasound imaging. (D) Unenhanced CT after treatment. The diameter was reduced to 11 mm, and SEC was not observed on ultrasound imaging, which suggests improved blood flow in the popliteal vein.

isolated dilation of twice the normal popliteal vein diameter.[4] According to Maldonado-Fernandez *et al.*, a venous dilation becomes an aneurysm when the diameter is at least three times that of the normal vein (> 20 mm).[3] These are the most commonly used definitions of PVA. Moreover, a PVA with a diameter > 20 mm has been associated with DVT.[5] A PVA greater than 20 mm should be treated promptly even if there are no thrombotic events or if thrombus is not visualised on ultrasound. This is because severe events, such as massive PE, could be the initial presentation[2,3] without prior symptoms. Moreover, ultrasound cannot always detect a mobile thrombus similar to the one presented here.

There are few reports of a PVA initially presenting with transient consciousness disorder in the literature. PVAs are usually discovered by ultrasound during investigation for heaviness, tiredness, swelling, or mechanical pain of the knee region and by duplex ultrasound when DVT is suspected or when assessing for DVT following the diagnosis of PE.[3] Sessa *et al.* reported that the percentage was divided equally between chance findings (49%) and presentation as thrombo-embolic disease (51%).[2] Despite the clinical presentations and severity of PVA ranging from asymptomatic to cardiac arrest secondary to PE,[2,3] there are few reports of a PVA initially presenting with transient consciousness disorder in the literature.

A massive PE can cause cardiac arrest that results in loss of consciousness, but the patient presented with normal haemodynamic and blood oxygenation status. The consciousness disorder in this patient was transient and the symptoms fully resolved on observation. Prior to visiting the clinic, his pre-syncope had been considered idiopathic. Transient loss of consciousness has been recognised previously as an important presentation of PE.[6,7] Although the rate of PE in transient consciousness disorder cases is subject to debate, PE should be considered as a cause of transient consciousness disorder.[7] One of the mechanisms accounting for this is the transient effect on the vagus nerve.[8]

In the presented case, the PVA was detected by ultrasound of the left lower limb during pre-syncope investigations, and PE was confirmed by enhanced pulmonary CT. After PVA treatment, all symptoms had disappeared. In accordance with these findings, it was assumed that the patient experienced recurrent thrombosis in the left PVA, causing left calf oedema, and that the thrombus travelled to the pulmonary arteries, causing pre-syncope. The differential diagnosis included neurological diseases, orthostatic hypotension, and cardiovascular diseases,[6] all of which were ruled out.

PVA treatments include anticoagulation and/or surgery.[2,3,5] Anticoagulation alone is inadequate because the PE recurrence rate can be as high as 43%.[9] Surgery is recommended where possible,[9] otherwise, lifelong anticoagulation is required.[5] Tangential aneurysmectomy with lateral venorrhaphy is widely preferred for PVA surgery, but direct anastomosis, interposing, and grafting are also effective choices.[2,3]

Anticoagulation therapy using apixaban was initiated one day following surgery and was continued until the six month follow up. The need for and efficacy of anticoagulation after surgery in this disease remains unclear. A factor Xa inhibitor was selected because they are reported to be as effective as conventional warfarin therapy, but with fewer adverse effects, in the treatment of DVT.[10]

It is assumed that an IVC filter is necessary for all patients who undergo surgery for PVA because the absence of thrombus in the PVA does not rule out the possibility of new onset PE caused by newly formed thrombi in the PVA during surgery. In addition, there was concern about the possibility of dislodging minor thrombi, which are not detected by ultrasound imaging, during surgical manipulation. In fact, minor thrombi were observed in the filter following its removal.

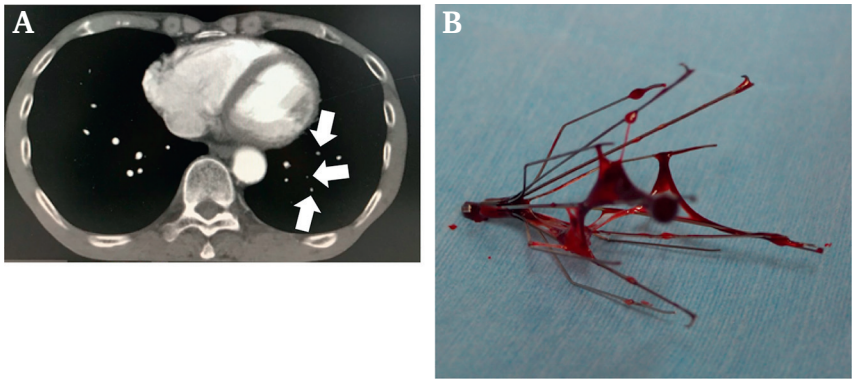

Figure 2. Pre- and post-operative images of pulmonary embolism. (A) Enhanced computed tomography (CT) scan in which thrombus was detected in peripheral pulmonary arteries (arrows). (B) Inferior vena cava (IVC) filter removed from the patient after treatment. Even though enhanced CT and IVC venography were performed, which ruled out major thrombus, minor thrombus was observed. This meant that the small thrombus was formed in and had travelled from the popliteal venous aneurysm.

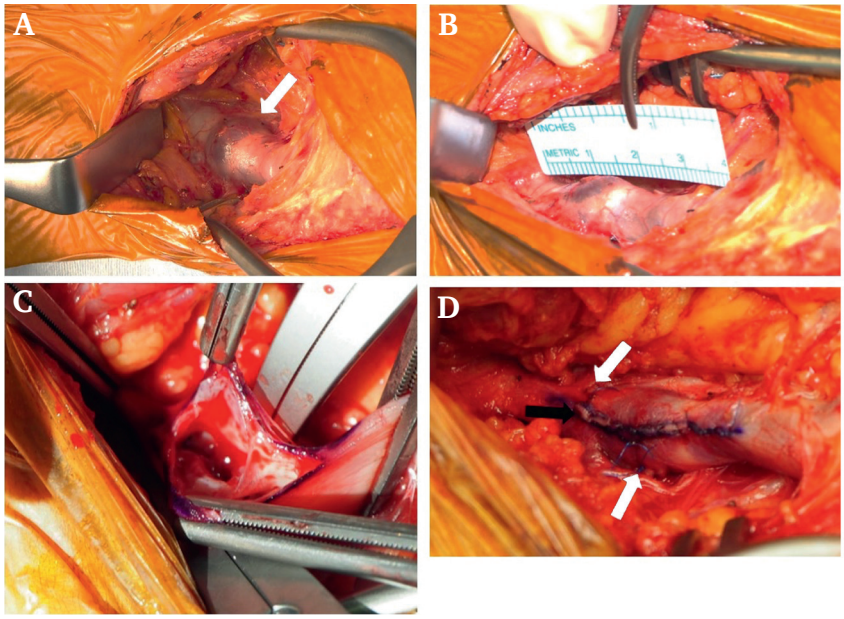

Figure 3. Images of operative findings. (A,B) Intra-operative images before aneurysmorrhaphy. A 20 mm saccular popliteal venous aneurysm was observed which was almost the same as in the pre-operative evaluations (arrow mark). (C) The aneurysm was incised after clamping. There was no evidence of thrombus, and the intima appeared normal on inspection, which justified aneurysmorrhaphy rather than grafting or interposing. (D) Imaging after completion of aneurysmorrhaphy (arrow mark: sutured line). The saccular aneurysm disappeared, and the diameter became the same as the proximal and distal veins.

CONCLUSIONS

A case of PVA initially presenting with recurrent presyncope is described. The patient was accurately diagnosed and was treated promptly and effectively by IVC filter placement, followed by tangential aneurysmectomy with lateral venorrhaphy, and post-operative anticoagulation.

CONFLICT OF INTEREST

None.

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