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Imaging Teaching Case

Central Venous Occlusion in the Hemodialysis Patient

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Central venous stenosis (CVS) is encountered frequently among hemodialysis patients. Prior ipsilateral central venous catheterization and cardiac rhythm device insertions are common risk factors, but CVS can also occur in the absence of this history. Chronic CVS can cause thrombosis with partial or complete occlusion of the central vein at the site of stenosis. CVS is frequently asymptomatic and identified as an incidental finding during imaging studies. Symptomatic CVS presents most commonly as an upper- or lower-extremity edema ipsilateral to the CVS. Previously unsuspected CVS may become symptomatic after placement of an ipsilateral vascular access. The likelihood of symptomatic CVS may be affected by the central venous catheter (CVC) location; CVC side; duration of CVC dependence; type, location, and blood flow of the ipsilateral access; and extent of collateral veins. Venous angiography is the gold standard for diagnosis. Percutaneous transluminal angioplasty and stent placement can improve the stenosis and alleviate symptoms, but CVS typically recurs frequently, requiring repeated interventions. Refractory symptomatic CVS may require ligation of the ipsilateral vascular access. Because no available treatment option is curative, the goal should be to prevent CVS by minimizing catheters and central vein instrumentation in patients with chronic kidney disease and dialysis patients.

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INDEX WORDS: Central vein stenosis; occlusion; thrombosis; central venous catheters; percutaneous transluminal angioplasty; stents; fistulogram; hemodialysis; end-stage renal disease (ESRD).

INTRODUCTION

Central venous stenosis (CVS) occurs commonly in hemodialysis patients.[1,2] It should be suspected when there is unilateral edema of the extremity ipsilateral to an existing or previous tunneled dialysis catheter. Previously unsuspected CVS may become symptomatic after placement of an ipsilateral vascular access. It is confirmed by angiography demonstrating >50% stenosis of the subclavian, brachiocephalic vein, and superior vena cava in the upper extremity or the iliac vein or inferior vena cava in the lower extremity.[1] Central venous occlusion can result from underlying CVS, and when the occlusion is complete, it may mask the underlying stenosis. We describe a case of a hemodialysis patient with severe symptomatic CVS.

CASE REPORT

Clinical History and Initial Laboratory Data

A 56-year-old man initiated hemodialysis therapy with a right internal jugular vein tunneled dialysis catheter. A left brachiocephalic arteriovenous fistula (AVF) was created, and the 6-week postoperative ultrasound documented an AVF diameter of 9.4 mm with access flow of 1.1 L/min. The catheter was removed 5 months after AVF creation. Over the ensuing year, the AVF diameter increased progressively, accompanied by marked dilation of the external jugular vein. The patient began reporting chronic left-sided neck and facial pain. He did not develop upper-extremity edema or neurologic symptoms. The AVF was cannulated without difficulty and delivered an adequate dialysis dose (Kt/V). Physical examination showed a tortuous and dilated left-arm AVF extending up to the shoulder, as well as a dilated tortuous neck vein (Fig 1). The patient had a remote history of gunshot to his neck without vascular injury. Other than the history of a dialysis catheter contralateral to the AVF, there was no history of another central venous catheter (CVC), cardiac device, or neck surgery.

Imaging Studies

The patient was suspected to have CVS and was referred for imaging studies. A fistulogram demonstrated chronic total long segment occlusion of the lateral left subclavian vein. The AVF drained by the left external jugular vein into the left brachiocephalic vein and then the superior vena cava (Fig 2). Intra-access venous pressure was elevated at 150 mm Hg systolic (concurrent systemic systolic pressure, 200 mm Hg). Recanalizing this chronically occluded long segment venous channel would have required ballooning and stenting the lateral subclavian vein and axillary vein, potentially requiring extension of the stent into the axilla owing to high mechanical stress and bending in this region. A stent in this area would have a poor patency rate, especially when competing against the large external jugular collateral. Given the chronic nature of the central venous occlusion, well-developed collateral circulation, and no upper-extremity edema, endovascular intervention was deferred.

Diagnosis

Severe symptomatic CVS was diagnosed.

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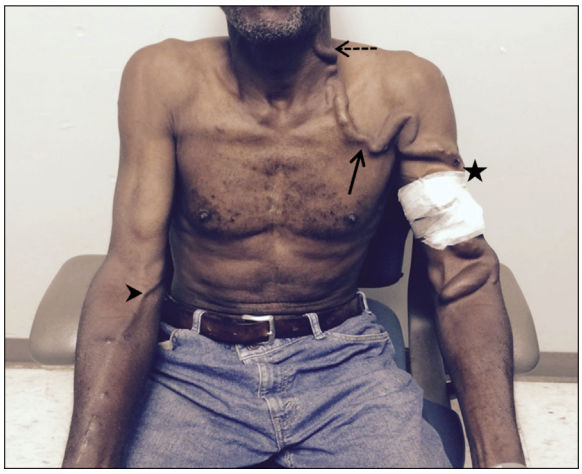

Figure 1. Photograph of the patient with severe central vein stenosis. Note the markedly dilated and tortuous left brachiocephalic arteriovenous fistula (AVF; star), the superficial chest wall collateral extending over the shoulder (arrow), and dilated and tortuous external jugular vein (dotted arrow). There is a new right radiocephalic AVF (arrowhead) that was created and successfully cannulated prior to ligation of the left upper-arm AVF.

Clinical Follow-up

As the AVF increased in size, the patient began to have worsening neck and facial pain related to the massively dilated left external jugular vein. Sonography of the AVF was not repeated. He was referred to a surgeon for possible AVF banding. However, the surgeon was concerned that the AVF might clot after banding, thereby necessitating placement of another right internal jugular catheter and potentially leading to superior vena cava syndrome. To avoid this possibility, the surgeon first created a new right radiocephalic AVF. When the new AVF was successfully cannulated, there was no longer a need for the left brachiocephalic AVF, so it was ligated.

DISCUSSION

There is a wide spectrum of clinical presentations for CVS. Many patients are asymptomatic, with CVS identified as an incidental finding during an imaging study.[3] A cross-sectional study documented internal jugular thrombosis in 26% of 143 hemodialysis patients with a current or past history of a dialysis catheter.[4] Similarly, angiography to evaluate flow-limiting vascular access stenosis often identifies a concurrent incidental CVS.[5-7] A previously unsuspected CVS might become symptomatic when an ipsilateral vascular access is created, leading to increased arterial inflow with inadequate venous outflow.[8,9]

There are many ways that a CVS may manifest. Patients most commonly develop unilateral upper- or lower-extremity edema, pain, tenderness, or erythema. Physical examination reveals a dilated tortuous access with aneurysms. Severe CVS may also cause facial edema, engorged neck veins, unilateral breast enlargement, and dilated collateral chest veins depending on the location of the stenosis/occlusion. Rarely, patients have a unilateral pleural effusion. Brachiocephalic vein stenosis or occlusion may lead to reversal of flow in the internal jugular vein, which in extreme cases may cause cerebral venous hypertension or even cerebral infarction or intracranial hemorrhage[10] if the contralateral internal jugular vein is also stenosed or occluded. Sometimes CVS results in prolonged bleeding from the access puncture site after removal of the dialysis needles, difficulty in access cannulation, elevated dialysis

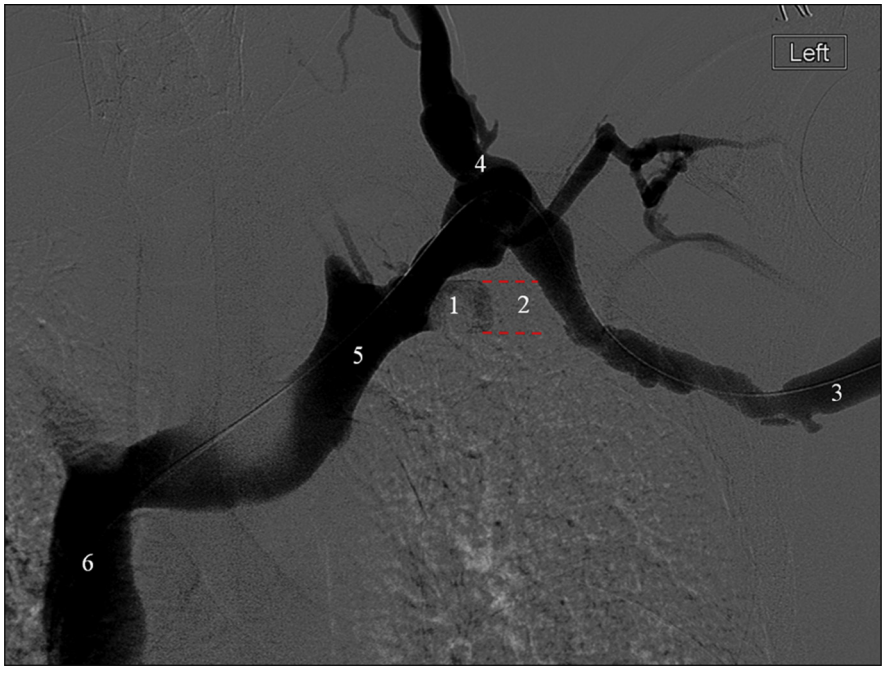

Figure 2. Angiogram of the arteriovenous fistula shows chronic occlusion of the lateral subclavian vein (dotted lines). The arteriovenous graft drains into the external jugular vein, which in turn flows into the left brachiocephalic vein, and then into the superior vena cava. (1) Reflux of contrast into central part of occluded left subclavian vein. (2) Long segment occlusion of left subclavian vein outlined. (3) Superficial chest wall collateral vein. (4) Superficial chest wall collateral draining into left external jugular vein. (5) Left brachiocephalic vein. (6) Superior vena cava.