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Mechanical thrombectomy for deep vein thrombosis with congenital anomaly of the inferior vena cava: A case report

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Abstract

We describe the case of a 12-year-old male presenting with severe lumbar back pain and disabling bilateral swelling and pain of the lower extremities. Investigation revealed extensive bilateral deep vein thrombosis in association with an absent infra renal segment of the inferior vena cava and anomalous venous drainage system. We used catheter-directed pharmacologic thrombolysis and mechanical thrombectomy to treat the deep vein thrombosis. The patient tolerated the procedure well. One year later he remains free of symptoms on oral anticoagulation medication.

Keywords

deep vein thrombosis; anomaly of the inferior vena cava; mechanical thrombectomy; catheter-directed thrombolysis

Introduction

Deep vein thrombosis has multifactorial etiology involving both genetic and acquired factors. Anomaly of the inferior vena cava is a risk factor for deep vein thrombosis, especially in young persons. We used catheter-directed pharmacologic thrombolysis and mechanical thrombectomy in a case with anomaly of the inferior vena cava to treat deep vein thrombosis and restore venous drainage and alleviate symptoms.

Case Report

The patient was 12-year-old previously healthy boy who presented with sudden onset of lumbar back pain and bilateral swelling and pain of the lower extremities after an intensive sporting activity. His symptoms prevented him from ambulation. Neurological and other general systemic examination was normal and his past medical history was unremarkable. He had no recent history of immobility and no family history of thromboembolic disease. The duplex ultrasound scan of the lower extremities showed thrombus in the bilateral femoral veins. Computed tomography scan and venography revealed thrombus extending from the popliteal vein and femoral veins to the iliac veins bilaterally and interruption of the inferior vena cava at the infrarenal portion (just below the junction of the renal vein) with the collateral veins through right second lumbar vein, right ascending lumbar vein, to hemiazygos vein (Figure 1, 2, 3, 4A). Systemic intravenous heparin therapy was initiated without improvement of symptoms. We then

arranged pelvic and lower extremity venography and catheter-directed thrombolysis. Two catheters were placed through the bilateral popliteal veins for catheter-directed thrombolysis with urokinase. After 48 hours of urokinase infusion, venography demonstrated the thrombus was still retained in the bilateral femoral veins and iliac veins. Because the patient had failed to improve on systemic anticoagulation treatment and catheter-directed thrombolysis, we proceeded with mechanical thrombectomy through the bilateral popliteal veins using manual aspiration. When the venous thrombus was hard and large, a three-loop snare was used to fragment it and repeated aspiration thrombectomy was performed. Post treatment venography demonstrated patent venous flow from bilateral popliteal veins to the iliac veins without significant intraluminal filling defects (Figure 4B). The patient's symptoms improved postoperatively. Computed tomography and biomarkers showed the patient was negative for hereditary thrombophilia, autoimmune disorders, malignancy and congenital defects of the heart and abdominal organs. Screening for thrombophilia was performed without any pathological findings. A full thrombophilia screening showed no alternative explanation for the thrombus. It therefore seems that the anomaly of the inferior vena cava itself was the cause of the thrombus formation. Because of the extent of the initial thrombus and the severity of the presenting symptoms, warfarin therapy was initiated. The boy remains free of symptoms on oral anticoagulation after one year.

Discussion

Congenital anomalies of the inferior vena cava have an estimated prevalence of 0.6% in the general population [1]. The incidence of deep vein thrombosis in normal young individuals under 35 years old is approximately 30/100,000 (0.03%) [2].However, in reports of patients between 20 and 40 years old presenting with deep vein thrombosis, the incidence of inferior vena cava anomalies reaches a rate of up to 5%–6.7% [3]. Pathogenesis mechanisms in deep vein thrombosis are basically contained in the Virchow triad: Venous endothelial damage, venous stasis, and hypercoagulable states (genetic or acquired). The abnormal venous system itself is a risk factor for the development of thrombosis. The pathophysiology of deep vein thrombosis associated with anomaly of the inferior vena cava includes inadequate blood return, increased blood pressure in the lower extremity veins, venous stasis, and subsequent deep vein thrombosis.

Several reports have been made of patients with deep vein thrombosis and anomaly of the inferior vena cava with a concomitant hypercoagulable state and hyperhomocysteinemiaor factor V Leiden [4,5]. Anomaly of the inferior vena cava has also been reported as an isolated risk factor for deep vein thrombosis. Chee et al. and Patel et al. reported anomaly of the inferior vena cava among young persons presenting with spontaneous deep vein thrombosis [3,6]. Our patient had no secondary risk factors, no hypercoagulable state and thrombophilia markers were normal. Thus it appears that the anomaly of the inferior vena cava itself was the cause of the thrombus formation.

Our patient did not show any cardiac and visceral anomalies, even though congenital anomalies of the inferior vena cava commonly occur in association with other cardiac and visceral congenital malformations, including congenital heart disease and kidney dysgenesis [1,6,7].

Several published reports suggest that major physical exertion may be the main precipitating factor of deep vein thrombosis for people with anomaly of the inferior vena cava. Consistent with these observations, our patient also had unusually intense physical exercise before the onset of symptoms.

It is most likely that the collateral vessels developed before deep vein thrombosis were unable to maintain the increased blood flow due to major physical exertion, thereby generating venous stasis and clotting [8].

Deep vein thrombosis in a lower extremity is usually diagnosed with ultrasonography, but detection of inferior vena cava anomalies by ultrasonography is difficult. Contrast enhanced computed tomography scan is the technique of choice to investigate and define the venous system in cases of suspected anomaly of the inferior vena cava [4]. Further evaluation with venography, angio-computed tomography and angio-magnetic resonance imaging are the imaging methods that can produce a diagnosis [9].

Most patients with anomalies of the inferior vena cava who suffer deep vein thrombosis are managed without surgical intervention or thrombolysis by treating them only with anticoagulation drugs. There are limited data on the optimum duration of anticoagulation treatment for these patients. The purpose of anticoagulation therapy in patients with anomaly of the inferior vena cava is symptomatic relief and prevention of post-thrombotic syndrome and recurrent deep vein thrombosis [10]. Most reports recommend a minimum duration of six months, but lifelong anticoagulant therapy may be required, depending on the extent of the deep vein thrombosis, its recurrence, and whether reversible risk factors such as oral contraceptive pills or prolonged immobilization are involved [6].

During mechanical thrombectomy, migration of emboli, which could cause potentially fatal pulmonary embolism, should be a great concern. In these cases, prophylactic inferior vena cava filter should be applied for prevention of life-threatening pulmonary embolism. However, in our patient with anomaly of the inferior vena cava, the drainage veins of the interrupted inferior vena cava were small and tortuous. Therefore, the fragmented thrombi lacked a main conduit through which they could travel to the lungs. Only a rare report had reported pulmonary embolism in patient with anomaly of the inferior vena cava [11]. The incidence of pulmonary embolism in patients with anomalies of the inferior vena cava might be lower than those with a normal inferior vena cava.

This case illustrates that in cases of deep vein thrombosis, especially in young persons, congenital anomaly of the inferior vena cava should be considered as a possible explanation. We obtained excellent results using catheter-directed pharmacologic thrombolysis and mechanical thrombectomy to treat this case of deep vein thrombosis related to anomaly of the inferior vena cava. Mechanical thrombectomy thus appears to be a favorable method of restoring venous patency and for the relief of acute symptoms. Moreover, mechanical thrombectomy seems to be a good therapeutic alternative for patients who do not respond to systemic anticoagulation and catheter-directed pharmacologic thrombolysis, whenever an expert interventional team is available.

Figures

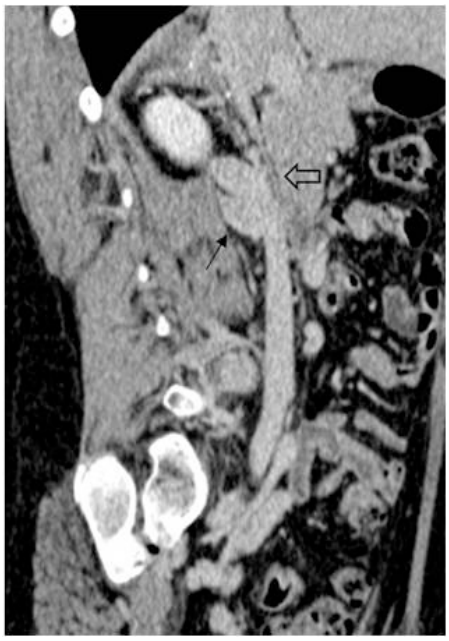

Figure 1: Computed tomography multiplanar reconstruction in the oblique coronal plane shows narrowed caliber of the infrarenal portion of the inferior vena cava (open arrow) and dilated lumbar veins (arrow).

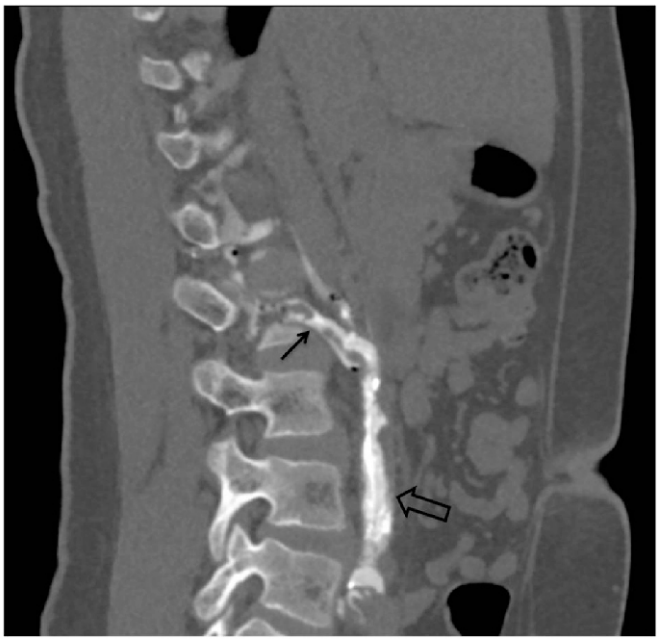

Figure 2: Computed Tomography (CT) angiography shows catheter tip within the inferior vena cava (open arrow), near the interrupted site, and dilated lumbar veins(arrow).

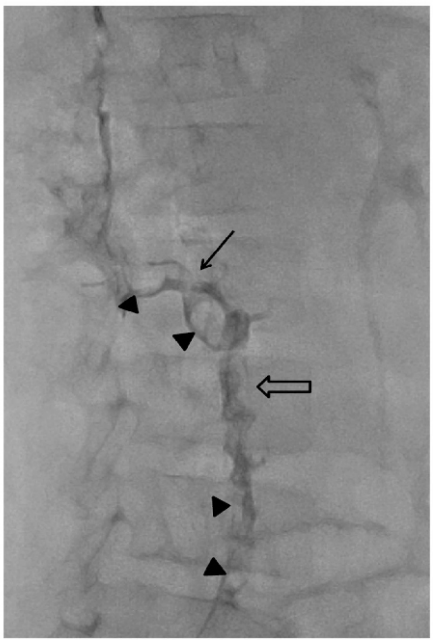

Figure 3: Venography revealed a) thrombus (arrow head) in the inferior vena cava(open arrow) and right second lumbar vein (arrow), and b) interruption of the inferior vena cava at the infrarenal portion with collateral veins through right second lumbar vein, right ascending lumbar vein, to hemiazygos vein.

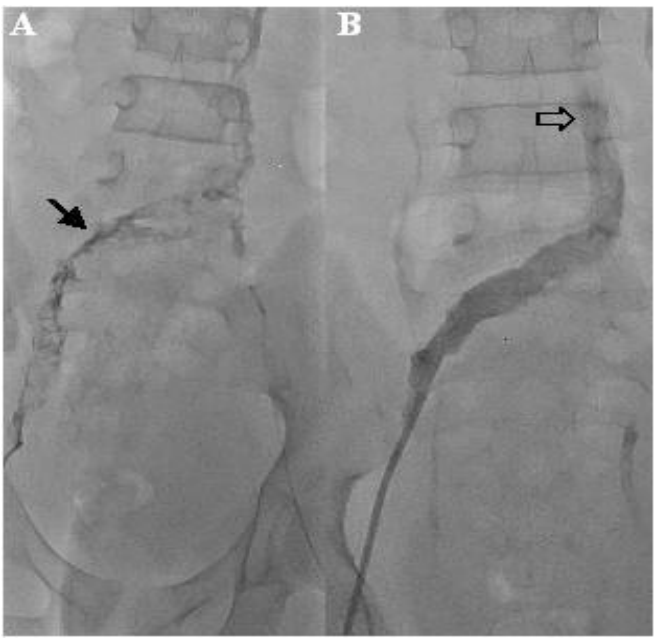

Figure 4: (A) Initial venography (approached via the popliteal vein) in the prone position shows extensive thrombus in the left common iliac vein (arrow). (B) A venogram after aspiration thrombectomy demonstrates restored flow up to the inferior vena cava (open arrow).

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