Caval Agenesis with Hypoplastic Left Kidney in a Trauma Patient on Warfarin for Deep Vein Thrombosis (Poster)

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Caval Agenesis with Hypoplastic Left Kidney in a Trauma Patient on Warfarin for Deep Vein Thrombosis

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INTRODUCTION

In 1891, Wardrop Griffith first reported on a case of inferior caval agenesis. In his case, he reported that the drainage of the lower part of the body was through an enlarged azygos system and the hepatic veins entered the atrium where the IVC typically would.[1] The incidence of congenital interruption of the inferior vena cava is quoted to be 0.3-0.6% of the population.[2] We report a similar case of a middle-age trauma patient with severe head trauma on coumadin therapy for longstanding deep vein thromboses. The patient was found to continue to have lower extremity DVT while in the intensive care unit prompting the need for an IVC filter. He was found to have inferior caval agenesis and a hypoplastic left kidney.

CASE

This is a 50-year-old male with a past medical history of bilateral lower extremity deep vein thromboses on coumadin therapy for 20 years who fell down 14 stairs 2 days prior to presentation. On admission, the patient had an initial Glasgow Coma Score (GCS) of 14 secondary to his confusion. Imaging studies revealed an intraventricular and intraparenchymal hemorrhage. The patient became progressively lethargic from hospital day 1 to 2, and a ventriculostomy was placed for hydrocephalus.

On post trauma day 7 a bilateral lower extremity venous duplex revealed non-occlusive thrombus within the right superficial femoral vein. Since he could not be anticoagulated secondary to his head bleed, the decision was made to insert an inferior vena cava filter via right femoral vein approach. During the procedure, the right femoral vein was accessed easily, but multiple attempts to advance the guidewire under fluoroscopy were unsuccessful. The procedure was aborted and the patient was taken to the interventional radiology suite to attempt a right jugular approach. The interventional radiologists were faced with the same issue. A cavagram was performed and was initially interpreted as the inferior vena cava being absent or chronically occluded just below the hepatic veins.

During the course of the patient's hospitalization he developed acute kidney injury (AKI) with a rise in creatinine from baseline. Urine studies indicated a possible post-renal cause for his AKI and a Doppler ultrasound of the kidneys was performed. The left kidney was unable to be visualized and the right renal artery and vein were patent. A MRI/MRA of the abdomen and pelvis was performed as the patient was unable to receive IV contrast. It revealed a chronically thrombosed versus absent inferior vena cava, a suprarenal segment draining the right kidney and hypoplastic left kidney, along with prominent lumbar and azygos veins. After the evaluation of the cavogram and the MRA, it was concluded by the radiologist that the patient had caval agenesis with a hypoplastic left kidney.

DISCUSSION

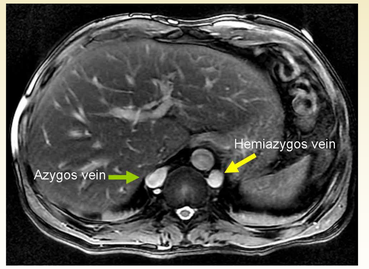
Absence of the inferior vena cava is a rare congenital anomaly, but a recognized cause of ileofemoral deep vein thrombosis. Diagnosis is best made by computed tomography (CT) of the abdomen and pelvis or an MRI of the abdomen. Ultrasound is not sensitive or the diagnosis of IVC anomalies. Although rare, it should be suspected in a DVT diagnosed in a relatively young patient without risk factors.[3]

Schnedl et al. in 2010 reported a case of subcutaneous vein patency in the scope of caval absence. They demonstrated external and internal iliac veins joining to form enlarging ascending abdominal subcutaneous veins allowing blood to return to the heart from the lower extremities by way of the azygous and hemiazygous veins. The same pathway was demonstrated in our patient except that our patient also had a hypoplastic left kidney. The tortuous nature of these collaterals, as well as the number of collaterals, can result in venous stasis and an increased risk for the development of deep vein thrombosis.[4] This lead to the recommendation that patients with deep vein thrombosis in the scope of anomalies of the IVC undergo aggressive treatment with therapeutic anticoagulation and possibly thrombolysis.[2]

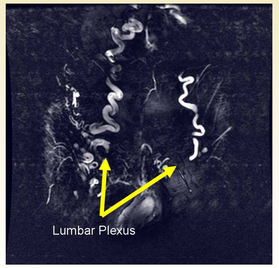
In 2003, Gayer et al.[5] described 11 patients with congenital IVC malformations and right renal aplasia. One patient had complete absence of the IVC where four other patients had a partial absence. They hypothesized that right renal hypoplasia occurs with anomalies of the IVC secondary to right renal drainage through a single renal vein, whereas the left kidney is drained by the renal vein and possibly the gonadal vein and lumbar perforators if needed.

We present a patient diagnosed with DVT 20 years prior to presenting to our institution secondary to trauma. He had been on anticoagulation for 20 years with warfarin, but was never diagnosed with an absent inferior vena cava. The diagnosis was made after attempting to place an IVC filter through a right femoral approach. He was also found to have a hypoplastic left kidney and a normal sized right kidney. This contradicts previous literature demonstrating right renal hypoplasia in similar situation based on the embryologic combinations described by Gayer et al. and Campell and Deuchar. We hypothesize that the left renal vein did not develop and the gonadal vein did not provide sufficient drainage for a normal kidney to develop.

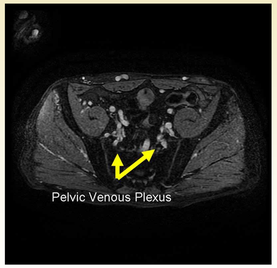
Finally, the discussion was had as to anticoagulation for this patient. He was unable to be anticoagulated after his head injury. This patient is not at risk for a pulmonary embolism, but if the venous collateral were to thrombose, his clinical course would have changed considerably. We kept him off anticoagulation during his hospital stay as his mental head injury did progress; however, the venous drainage of the lower extremities did not thrombose. Eventually the patient recovered from his head injury and was transferred to a skilled nursing facility with his anticoagulation restarted.



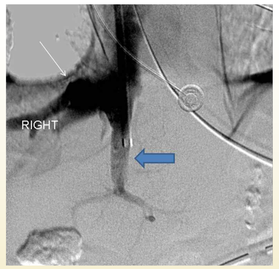
▲ **Fig 1.** MRA of abdomen showing dilated azygous and hemiazygous veins.



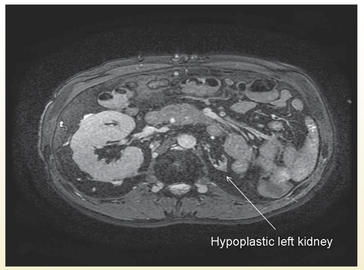
▲ **Fig 2.** MRA of abdomen demonstrating a dilated lumbar plexus aiding blood return from the lower extremities.



▲ **Fig 3.** MRA of abdomen demonstrating a dilated pelvic plexus.



▲ **Fig 4.** Cavagram demonstrating the hepatic veins (white arrow) joining a small suprarenal inferior vena cava (blue arrow) coursing to the heart.



▲ **Fig 5.** MRA of abdomen demonstrating a hypoplastic left kidney.

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