

Case Report

ILIO CAVAL BYPASS FOR HYPOPLASTIC INFRARENAL INFERIOR VENA CAVA

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ABSTRACT

Congenital anomalies of the IVC are rare. They are seen most often in young males. They usually are dormant for a long time. They usually present as deep venous thrombosis or Obstruction with symptoms of Chronic Venous Insufficiency. Patients with chronic occlusion of the IVC may be asymptomatic or present with mild edema and calf pain. They may also present with venous claudication pain or venous ulcers and severe swelling. Results of open surgical treatment for deep venous occlusions have been less than satisfactory for many years. Only in the past two decades have improvements in diagnosis, patient selection, and surgical technique and the availability of better graft materials resulted in more successful implantation of venous bypasses in patients. Endovascular techniques with balloons and stents have also been described with variable results. We report on the first patient with Hypoplastic IVC and Circumaortic left renal veins that had excellent outcome after Ilio-Caval bypass.

Keywords: IVC- Inferior Vena Cava, Hypoplastic, AVF-Arterio Venous Fistula, DVT-Deep Vein Thrombosis

CASES

50 year old male patient presented with left leg ulcer and swelling. His ulcer was present near the medial malleolus. The ulcer was painful and present for 6 months. He gave history of open varicose vein surgery 5 years back after which he developed Deep Vein Thrombosis of the left leg 2 years ago. He was on Oral Anticoagulation subsequently.



Figure 1: Hypoplastic Infrarenal IVC with Normal Iliac veins

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The ulcers were not responding to compression therapy or stockings. A Contrast enhanced CT Venogram of the IVC (Figure 1) showed a Hypoplastic IVC with Normal Iliac veins bilaterally. Further the CT venogram also revealed a Circum Aortic Left renal vein (Figures 2 &3).

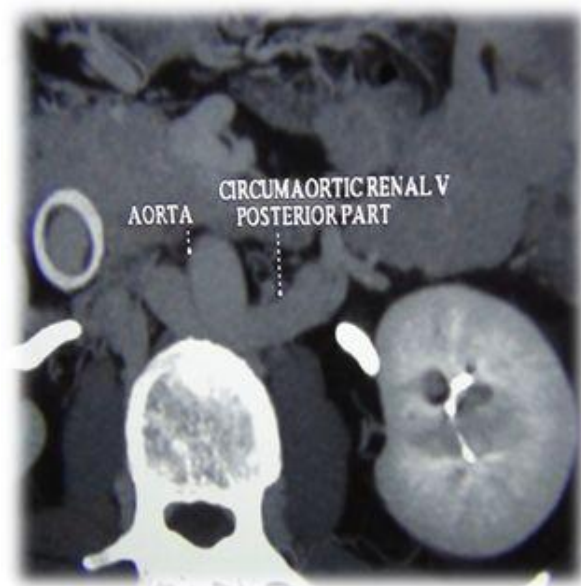


Figure 2: Circumaortic Left renal Vein- Posterior Part



Figure 3: CircumAortic Left renal Vein- Anterior part

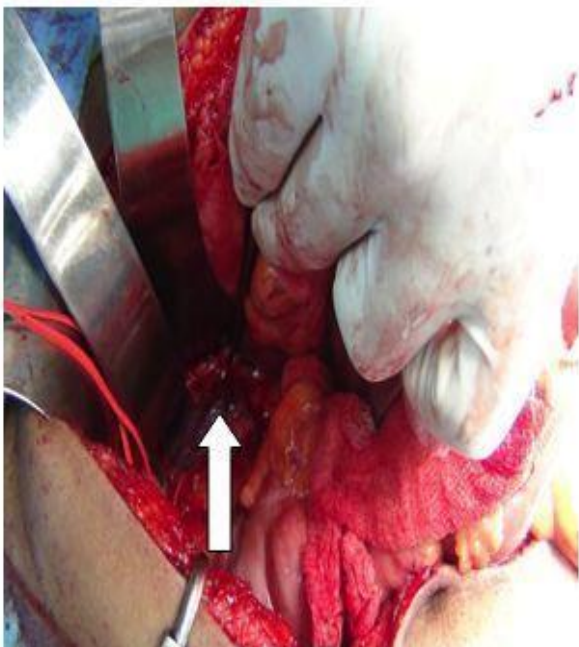


Figure 4: IVC with Blebs on the surface: White arrow



Figure 5: Ringed PTFE graft from RT Iliac Vein to Suprarenal IVC



Figure 6: Patent Ringed graft: black arrow

He was taken up for a Right Common Iliac Vein to Suprarenal IVC Bypass, using a 16mm Ringed PTFE graft. A Midline Laparotomy and Retroperitoneal exposure was used to expose the IVC and Bilateral Common Iliac veins. IVC was exposed suprarenally and bilateral Renal Veins were exposed. There were Blebs (Figure 4) found on the surface of the Infrarenal IVC which led us to believe that the IVC hypoplasia was probably congenital in origin. There were no Thrombi in the Lumen of the IVC. A 16mm Ringed PTFE graft (Figure 5) was used to perform a bypass from the Right Common Iliac Vein to the Suprarenal IVC tunneled above the renal veins. An Arterio-venous Fistula was not performed as there was good forward flow from the iliac veins and profuse back bleed from the suprarenal IVC. Post operatively patient was maintained on oral anticoagulation and compression stockings with 30 mmHg pressure. His leg ulcers healed in 3 months. On follow up after a year the PTFE Graft is still patent (Figure 6), leg edema has significantly reduced and pain is absent and he is compliant with medication and compression stockings.

DISCUSSION

The embryology of the IVC is complex. It involves the formation, regression and fusion of three pairs of longitudinal veins. The Infrarenal IVC is formed from the confluence of the postcardinal veins and right supracardinal veins. The left renal vein is formed from the fusion of left subcardinal plexus and left supracardinal vein, and the posterior component of the left renal vein regresses after the 8th week.

Aplasia or Hypoplasia of Suprarenal IVC results in Azygos continuation into the right atrium. These patients may also have other cardiac defects (0.6%-2%) or may be the solitary congenital defect (0.3%) (Bass *et al.*, 1999). Anomalies of the infrarenal IVC is extremely rare. No associated congenital anomalies have been reported in this group. This suggests that aplasia or hypoplasia of the infrarenal IVC may be an acquired defect, or secondary to thrombosis (Tharumenthiran *et al.*, 2001). However our patient had hypoplastic infrarenal vena cava associated with circumaortic left renal vein. This led us to attribute it to a congenital cause. He also had blebs on the surface of the vena cava which confirmed our beliefs.

The most common anomaly of the IVC is Aplasia and Hypoplasia, where venous blood from the legs is drained via the azygos vein into the Superior vena Cava (Ueda *et al.*, 1983). The Azygos vein and Lumbar veins will compensate for the IVC and carry the lower limb and pelvic venous outflow. Total and Partial agenesis of the IVC seem to be affected with a high rate of thrombosis. For a thrombotic episode to occur, a further risk factor should occur, namely venous stasis, immobilization or thrombophilia (Sagban *et al.*, 2010).

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Patients with IVC anomalies may be totally asymptomatic or may present with DVT. Some remarkable features being, that the patients were younger, with no precipitating cause. They often had bilateral DVT or Iliac vein thrombosis and frequently recurrent thrombosis (Baesko *et al.*, (2007). Some cases are seen incidentally in CT/MRI scans or during radiological workups (Vijayvergiya *et al.*, 2005).

Duplex scanning is usually the first imaging modality in evaluating patients with leg ulcers and varicose veins, but IVC anomalies will be missed. CT and MR Venography are the best methods to visualize for IVC disease. Extensive collateral flow is observed in our case and azygos and hemiazygos systems are prominent. Collaterals along the abdominal wall and paravertebral venous plexus are also seen. CT and MRI can also detect other associated congenital anomalies.

Our patient's pain, swelling and ulceration were restricted to the left leg. This could be explained as he previously developed deep venous thrombosis of the left leg veins which could have resulted in valvular insufficiency.

Initial treatment is conservative, including compression stockings, leg elevation and anticoagulation drugs. However in symptomatic IVC obstruction with venous ulcerations, severe swelling and pain, surgical or endovenous intervention is recommended.

Sagban *et al.*, (2010) have reported on 15 patients for whom they have performed bypass for occluded IVC. They report a 1 year patency of 66%, with improvement of symptoms in all patients. Baesko *et al.*, (2007) have reported on 44 bypasses for 42 occluded ilio caval systems. Only 2 of their patients had congenital IVC occlusion and the remaining were for post thrombotic sequelae. They reported a two year primary patency of 54% and clinical improvement in 64%. They also suggested that creating an AVF may have a negative effect on graft patency. This they suggested may be due to increased venous hypertension. However Sagban *et al.*, (2010) suggest that AVF creation improves the graft patency and they ligate the AVF after 3 months. We did not perform an AVF for our patient. All authors agree that anticoagulation is imperative after interventions; however the duration of anticoagulation is debatable. This patient was on anticoagulation and compression stocking.

Seshadri *et al.*, (2006) reported on endovascular stenting for 97 out of 120 patients with Iliocaval obstructions. Only 7 of these patients had isolated infrarenal IVC occlusions. Three of these patients became symptomatic due to stenosis of the collateral veins and 4 presented with iliac vein thrombosis. Stenting could not be done for collateral stenosis.

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