

A case of Total Iliocaval Occlusion and Reconstruction

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Louisiana Heart Center

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- Consulting: Medtronic venous.



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Michael Levy



Patient History

- JB initially presented as a 44 yo male with a history of PE x 2 , first at age 17 then age 23. He has been on chronic anticoagulation with full dose enoxaparin prescribed by hematology and suffered from severe postphlebitic syndrome (villalta score 21) despite compliance with 30-40 mm/hg stockings. His initial ultrasound showed diffuse chronic occlusion with recanalization of his CFV's to infrapopliteal veins bilaterally. His Physical exam was striking for well developed varicosities across his abdomen as well as bilateral edema, 5 mm varicose veins in his left calf, bilateral venous status dermatitis and wounds. He had normal pulses, normal echo and a BMI of 26. He was on disability as a result of his chronic disease. His only venous therapy prior to presenting was of sclerotherapy to superficial veins on his right leg. He has no known hypercoagulability per hematology workup but I do still suspect an as yet undefined hypercoagulability.



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Evaluation

- Venous Ultrasound- showed areas of total occlusion and recanalized vein from the common femoral to the infrapopliteal veins bilaterally.

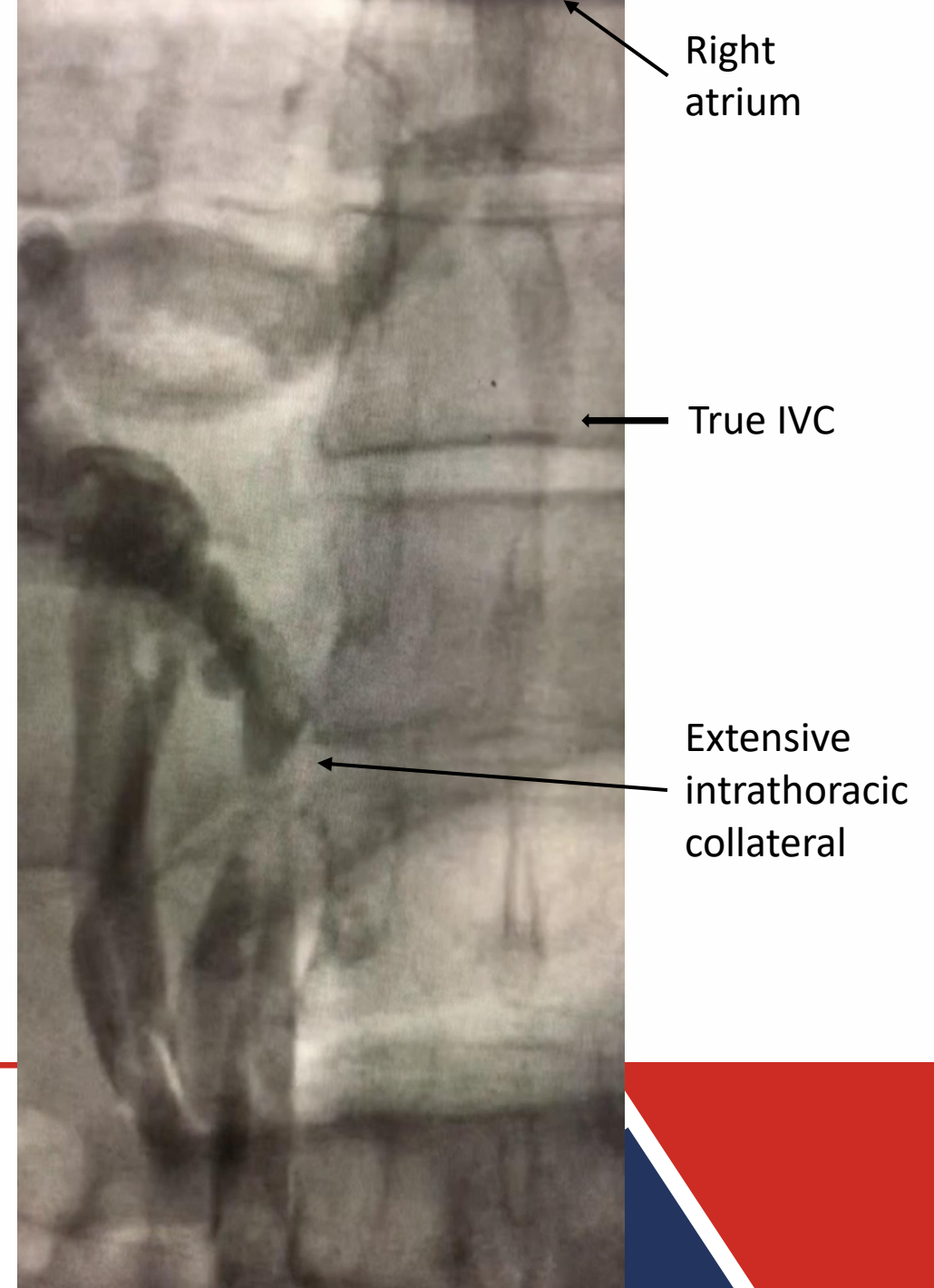


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Initial Venogram

- Initial access was in the Posterior tibial veins bilaterally under ultrasound. Venogram showed total occlusions at the popliteal veins with collaterals and proximal femoral veins into common femoral veins bilaterally. The external iliacs were partially opened and the common iliacs were occluded. Those occlusions continues up the entire IVC to above the hepatic vein. A large serpentine venous collateral had formed to drain the organs.
- Using blunt dissection techniques, we patiently worked the catheters up into the right atrium, ballooning the various segments as we crossed.
- Once in the RA, an IVUS was performed to make sure we were in a safe position to angioplasty. Balloon angioplasty was performed with 4 and 6 mm balloons from the base of the RA to the popliteals bilaterally. Smaller balloons were used after noting the venous size on IVUS in many areas did not exceed 6 mm.
- Of note- The iliac veins ran abnormally high into the main IVC, coming in at the L-3 level.



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Initial venogram movie

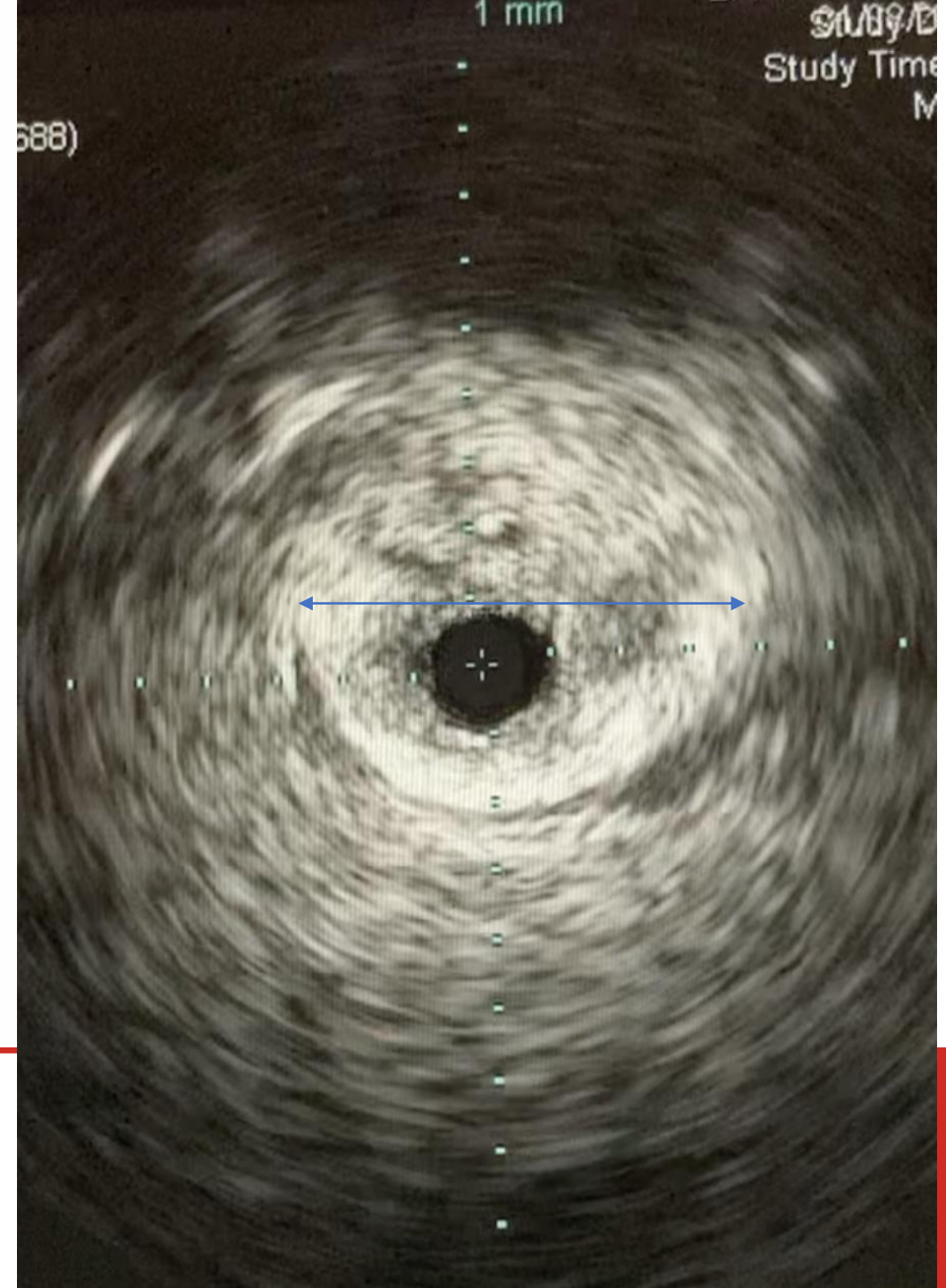


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Initial IVUS

- The Ivis was impressive for the severe involution and small diameter of the IVC. Even a generous measurement was 6 mm in many areas.



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Initial decision making

- The anatomy was very surprising in its extent, size and anatomic variance. We decided to treat this as an investment procedure, creating a return path with angioplasty only.
- Opinions were obtained from other interventionalists who have treated this anatomy and the literature was reviewed.

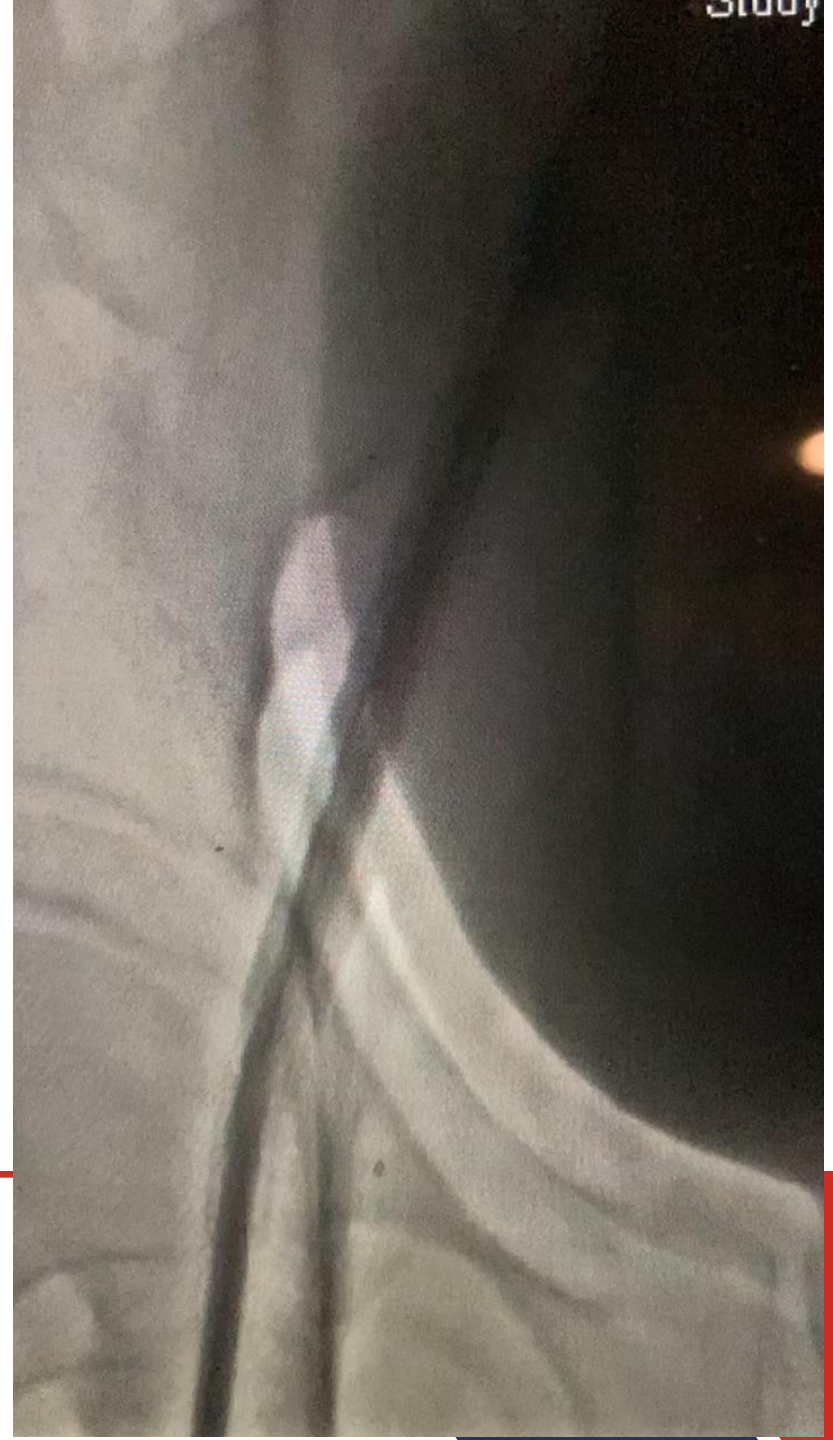


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Second venogram

- Accessing the greater saphenous veins bilaterally we were able to regain wire access through the iliacs and IVC into the RA and parked wires in the subclavian veins. Initial angioplasty with 3, 7 and 10 mm balloons to 12 atmospheres, limited by the pain the patient was experiencing with the larger inflations.
- These cases are rare in the literature, in personal conversations with interventionalists who have treated these cases, some recommended a double barrel stent into the RA. Others used a single Z stent. I did not have access to a Z stent and I felt the sharp ends of a wallstent would be too risky abutting the RA or invading it. So we decided on a 10x25 mm balloon expandable Express-LD ,bare metal stent (BMS) at the base of the RA, followed by a 22x80 wallstent which was deployed slightly in the distal end of the previous BMS. Then double barreled 18x90 wall stents into the iliacs and continuing those into the Common femoral veins bilaterally with 16 mm wallstents.
- The post procedure flow was good and the plan was to let these scar in then try and enlarge them in a future procedure as the lumen was still 6 mm in some areas even after the balloons appears to fully dilate.



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Venogram 3

- In the third procedure about 2 months after the second The right sided stents were widely patent but there was an area of occlusion in the left external iliac vein with extensive collaterals. We crossed and repeated the angioplasty with up to 14 mm balloons.



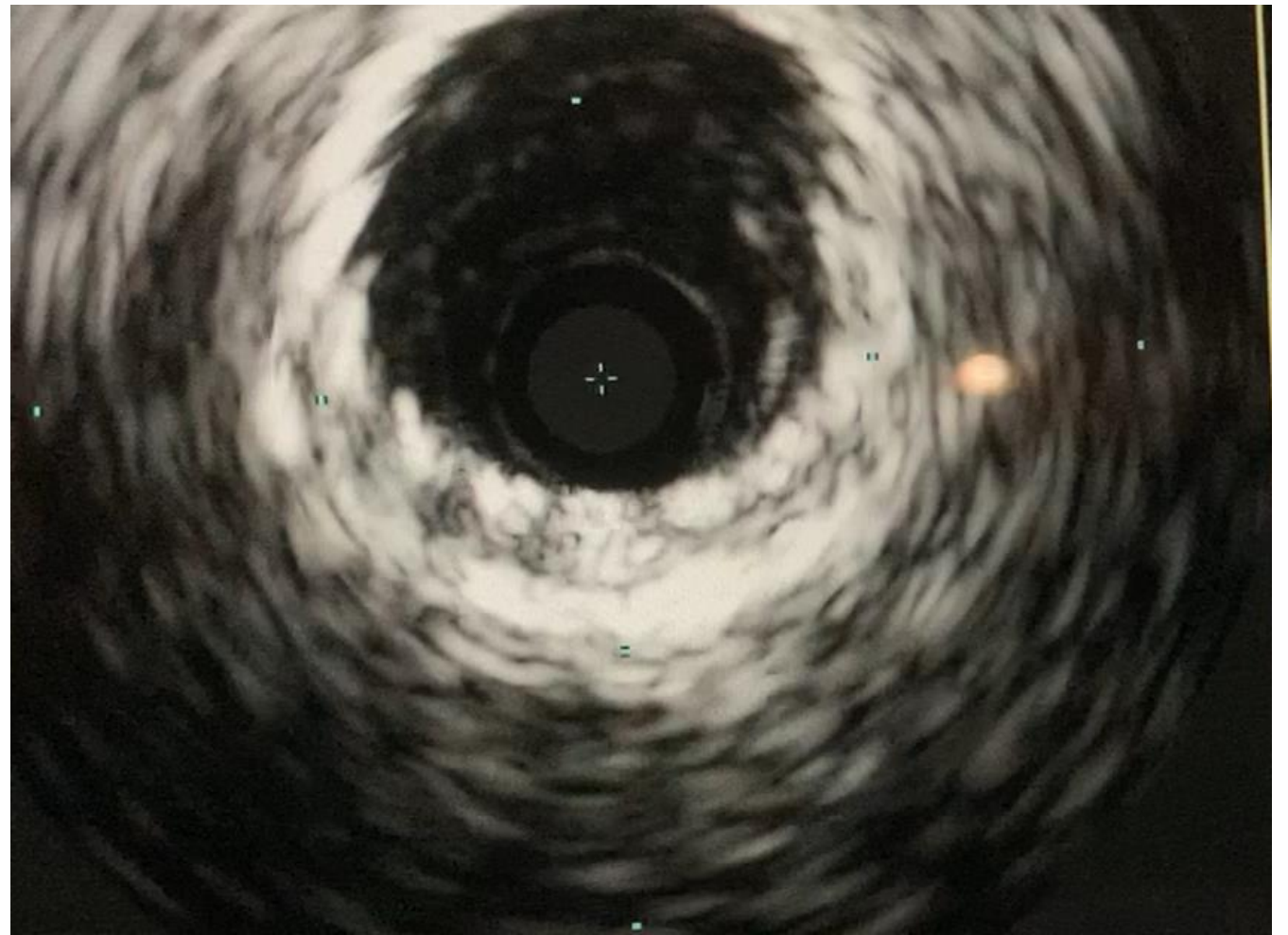
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Venogram 3

Despite aggressive angioplasty even though the balloons would attain nominal size, the stent area was insufficient on IVUS demonstrating compression from external compression.

5 MM
Markers



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Hospital Course and Follow-Up

- Patient was discharged on full dose anticoagulation and did well for about 15 months
- Patient re-presented with symptoms of ambulatory venous hypertension on the left and a new wounds.
- He remained compliant with stockings and anticoagulation.



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Venogram 4

- This time we again accessed the infrapopliteal veins to evaluate and optimize the infrainguinal veins. They had remained patent since the original angioplasty 20 months prior but the left external iliac vein stent had again closed and there was a patent area of narrowing and stent separation at the junction of the common iliac and the IVC. This time we re-stented the iliac with a 16x90 wallstent.



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Hospital course and follow-up

- Pt was again discharged on full anticoagulation of Pradaxa 150 bid, Unfortunately, within weeks his symptoms returned.



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Venogram 5

- Coming from the popliteal vein on the left, Our popliteal and femoral veins remained patent. We found the iliac portion of the stent occluded. This time the occlusion extended into the CFV due to loss of the previous collaterals. It was firm, already feeling chronic. We again, worked across the lesions and performed initial balloon angioplasty using 10/12 mm Athleta balloons due to their resistance to rupturing against wall stent edges. The IVUS showed we still had our chronic problem of externally compressed stent and inadequate lumen in the iliac. So, this time we used 3, 10x37 BMS to support and enlarge the lumen in the areas most affected. This did work to overcome the external forces, then we post dilated to 12 mm. We had to extend another 18 mm wallstent into the CFV several centimeters to ensure inflow. Our final flow was fast with minimal flow into collaterals.



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Hospital course and follow-up

- At three months the stents have remained patent He has substantially less pain and has resumed his normal activity. His Villalta score is still 15 due to the presence of active wound. He still has a small wound on his anterior leg at the level of superficial varicosities. The Next step will be to perform localized ablation of the insufficient GSV feeding these veins. He will remain on lifetime anticoagulation if possible due to the extensive amount of metal that is unlikely to ever be fully covered by endothelium and his still possibly unidentified hypercoagulability.

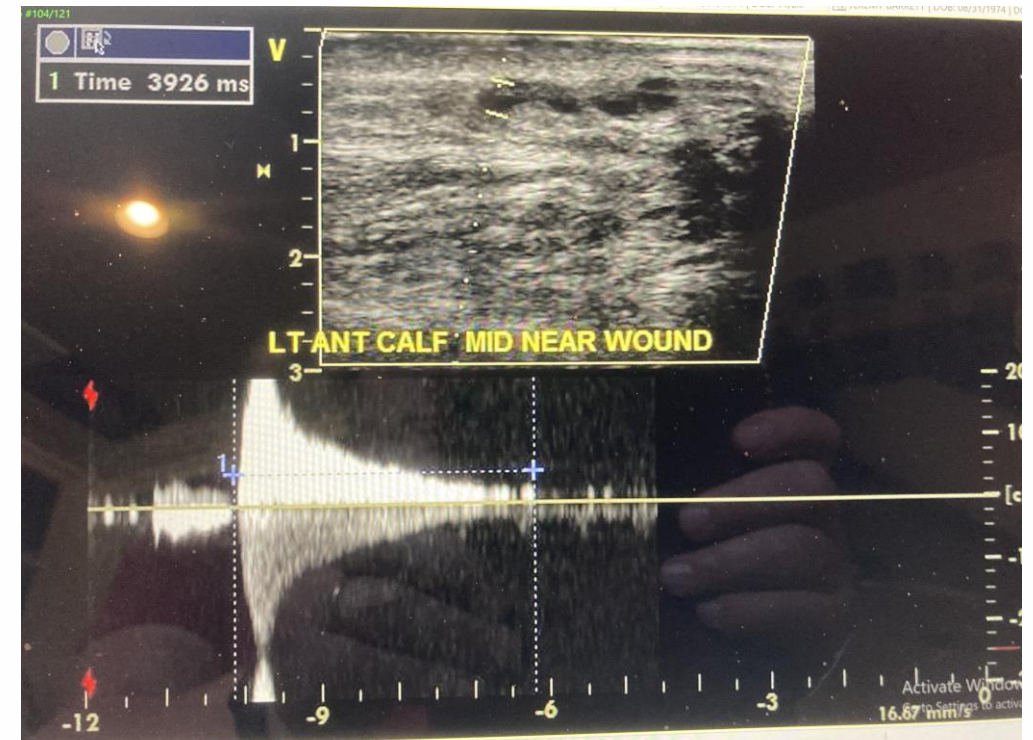
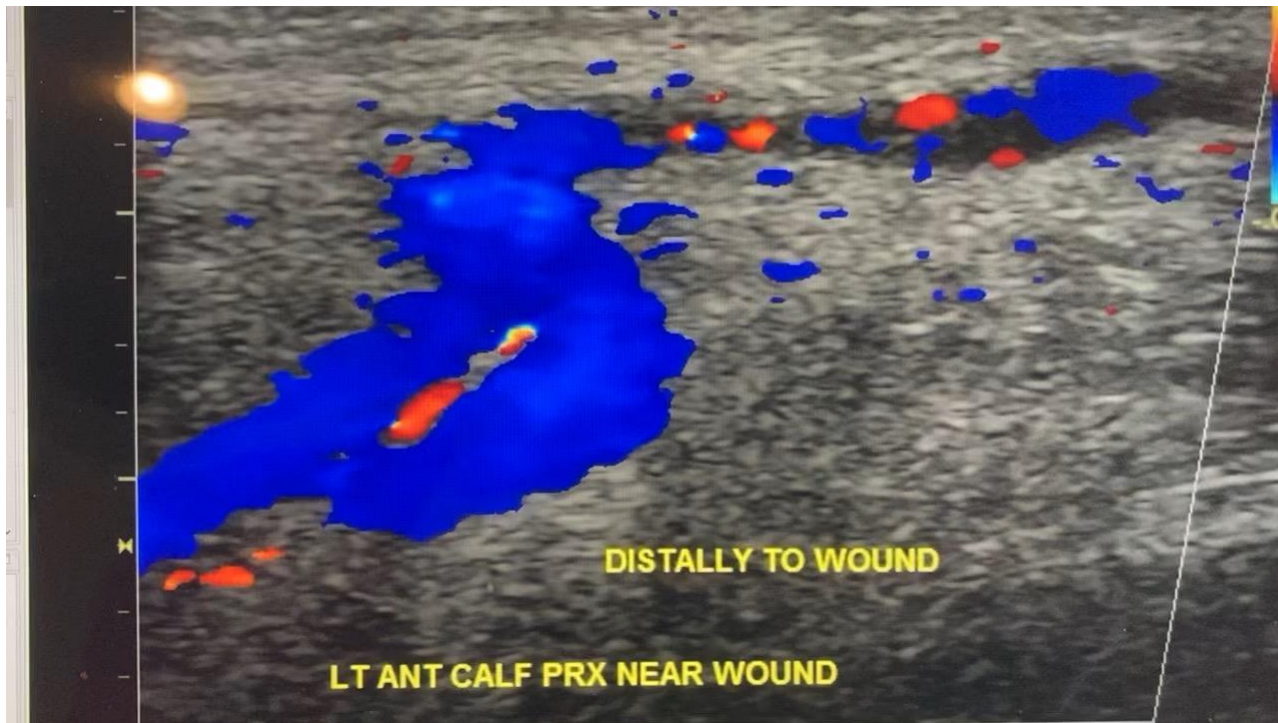


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Greater Saphenous Vein Intervention Complication

- As mentioned previously the patient had a chronic vein wound on the left leg fed by insufficient GSV as well as perforators.

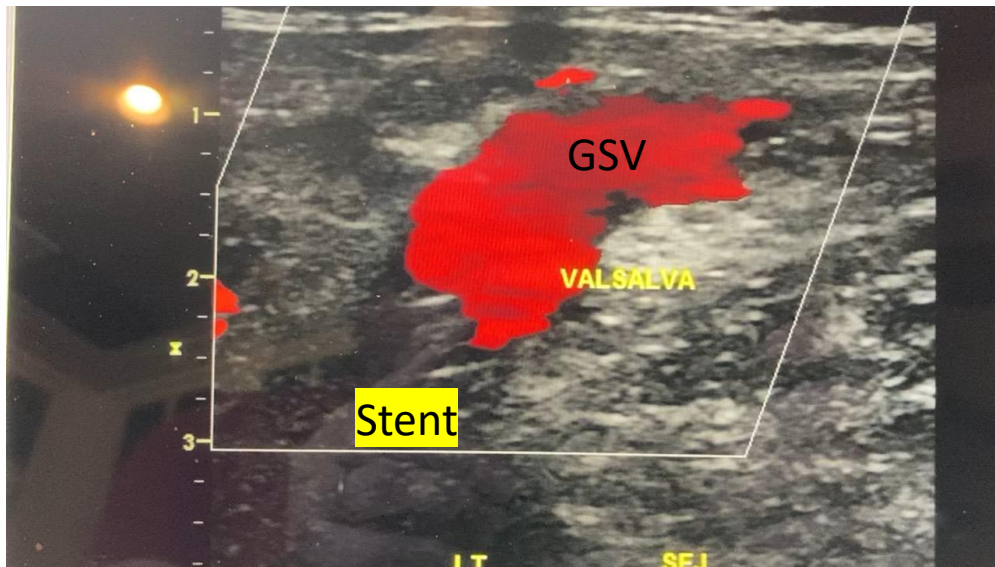


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Greater Saphenous Vein intervention Complication

- The GSV fed a fair amount of flow into the side of the stent at the Left Common Femoral Vein. We did not want to diminish the flow significantly, concerned diminishing flow may close the stents. The plan was to Venaseal the left lower GSV to the point where a large perforator fed the GSV just above the knee allowing the thigh portion to remain patent and free flowing into the stent. We avoided sclerotherapy out of DVT concern and phlebectomy to avoid creating additional wounds.



Procedure Date: 11/10/2023 Physician: [Signature] Technician: [Signature]

OBJECTIVE:

Limb Selected for Treatment: Right Left

Access Site: Groin Knee Ankle

Type of Anesthesia: Local Spinal General

Anesthesia: 5 mL Delivered

Vein Access Procedure: Percutaneous Skin Incision

Vascular Access: 21 Gauge Needle 4 FR. Sheath

VEIN SEGMENT TREATMENT:

Segment	Length (cm)
<input type="checkbox"/> Great Saphenous Vein, Above Knee	_____
<input type="checkbox"/> Great Saphenous Vein, Below Knee	_____
<input type="checkbox"/> Entire Saphenous Vein	_____
<input checked="" type="checkbox"/> Other: GSV to dist thigh	40

LASER DETAILS: (Vari-Lase Kit # 7157)

Total Laser Energy _____ Joules

Power _____ Watts Continuous Wave

Total Procedure Time _____ Seconds

Total _____ Joules / cm

COMPLICATION (S):

No Yes If Yes, Specify...

Vessel Perforation Hemorrhage Hematoma

Vasospasm Phlebitis Paresthesia

Medtronic VenaSeal™ Closure System

Barcode: (01)00763000240778(17)230531(1)063574

REF: VG-404 [LOT] 43574 2023-05-31

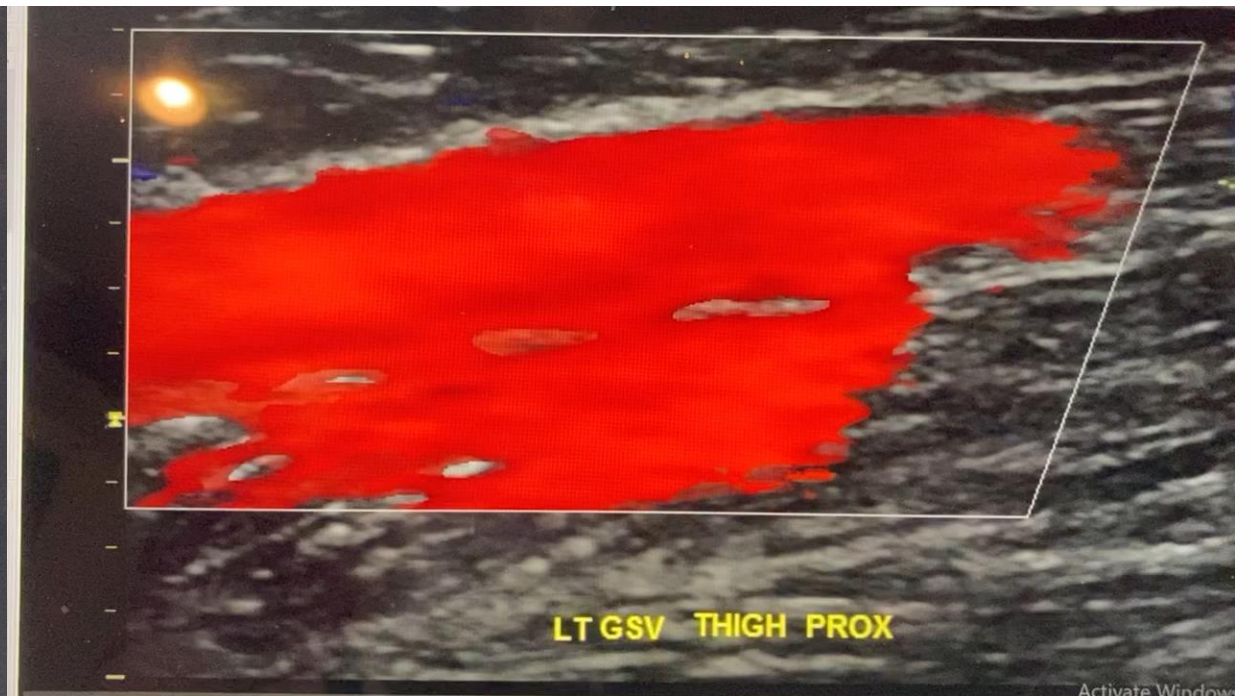
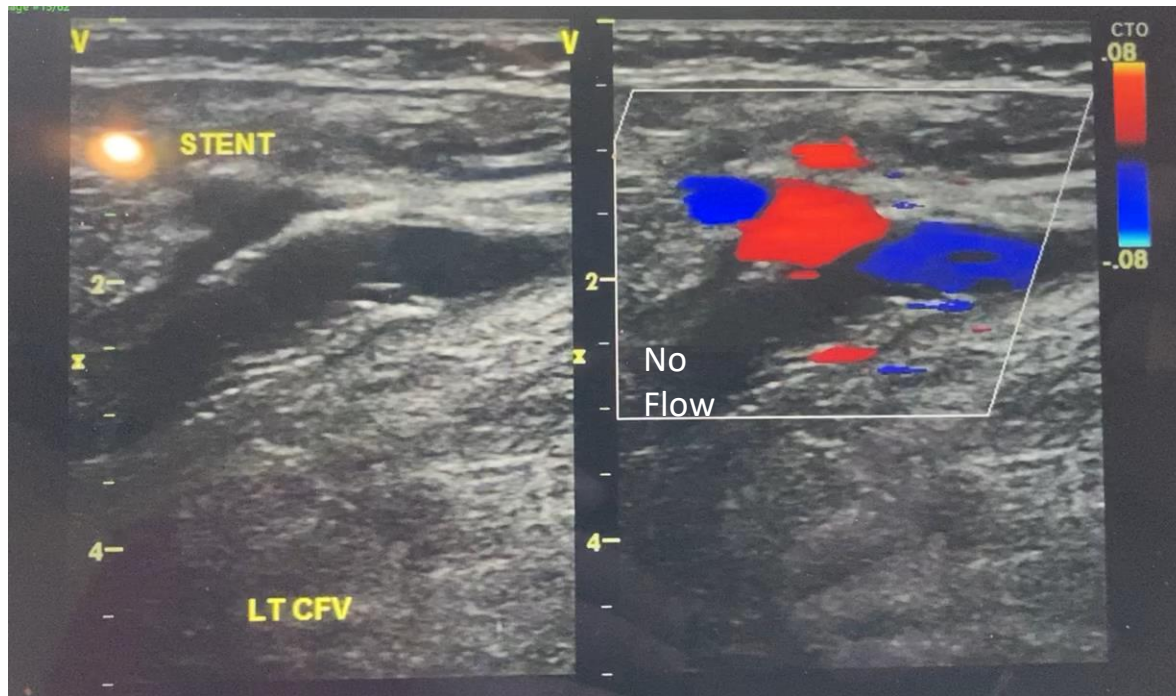


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Greater Saphenous Vein intervention Complication

- The plan appeared to go well but on the day 3 venous US, the stent was again occluded despite the thigh GSV remaining patent with good flow.



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Venogram 6

- Coming from a right brachial vein approach, 8 fr x 90 sheath, we performed a Power Possis Angiojet with 10 mg of TPA and angioplasty of the left sided stents, again using 12 mm balloons to try and enlarge the stents as much as possible. The occlusion was confined to the stent and the previously opened femoral veins had remained widely patent.



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Venogram 6

The Top of the double barrel portion of the Stents was noted to have significant thrombus. It was treated with Power Possis then double ballooning with good outcome.



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Follow up

- At this point it has been two months since the last procedure and his left leg wounds remain healed and bilaterally his stents are still patent. He does complain on some pain with bending the left hip which I am concerned may be negative stent interaction. The stent remains straight as the vein tries to bend creating recurrent micro trauma and scarring past the time usual restenosis would be expected to occur. We plan to do serial ultrasounds every 2 months in order to try and be in front of any restenosis.



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Question

- What are the two most described non-iatrogenic, non-cancer related causes of suprarenal IVC occlusion?
 - a. Budd-Chiari Syndrome
 - b. IVC trauma
 - c. Combined hypercoagulabilities
 - d. Congenital IVC hypoplasia



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Answer

- Budd-Chiari Syndrome^{2,3} and Congenital IVC hypoplasia¹.
- It is impossible to know for sure but considering this patients lack of hypercoagulabilities, no history of hepatic disease and high takeoff of his iliac veins. I believe he suffered from an anomalous IVC formation persistent left IVC and possibly hypoplastic suprarenal IVC.

1. McDevitt JL, Srinivasa RN, Hage AN, et al. Total endovenous recanalization and stent reconstruction for naive non-inferior vena cava filter-associated chronic ilio caval occlusive disease: Placement of 352 venous stents in 69 debilitated patients. *Vascular Medicine*. 2019;24(4):349-358. doi:10.1177/1358863X19834354

2. Okuda K. Inferior vena cava thrombosis at its hepatic portion (obliterative hepatocavopathy). *Semin Liver Dis*. 2002 Feb;22(1):15-26. doi: 10.1055/s-2002-23203. PMID: 11928076.

3. Horton, J.D., San Miguel, F.L. and Ortiz, J.A. (2008), Budd-Chiari syndrome: illustrated review of current management. *Liver International*, 28: 455-466. <https://doi.org/10.1111/j.1478-3231.2008.01684.x>

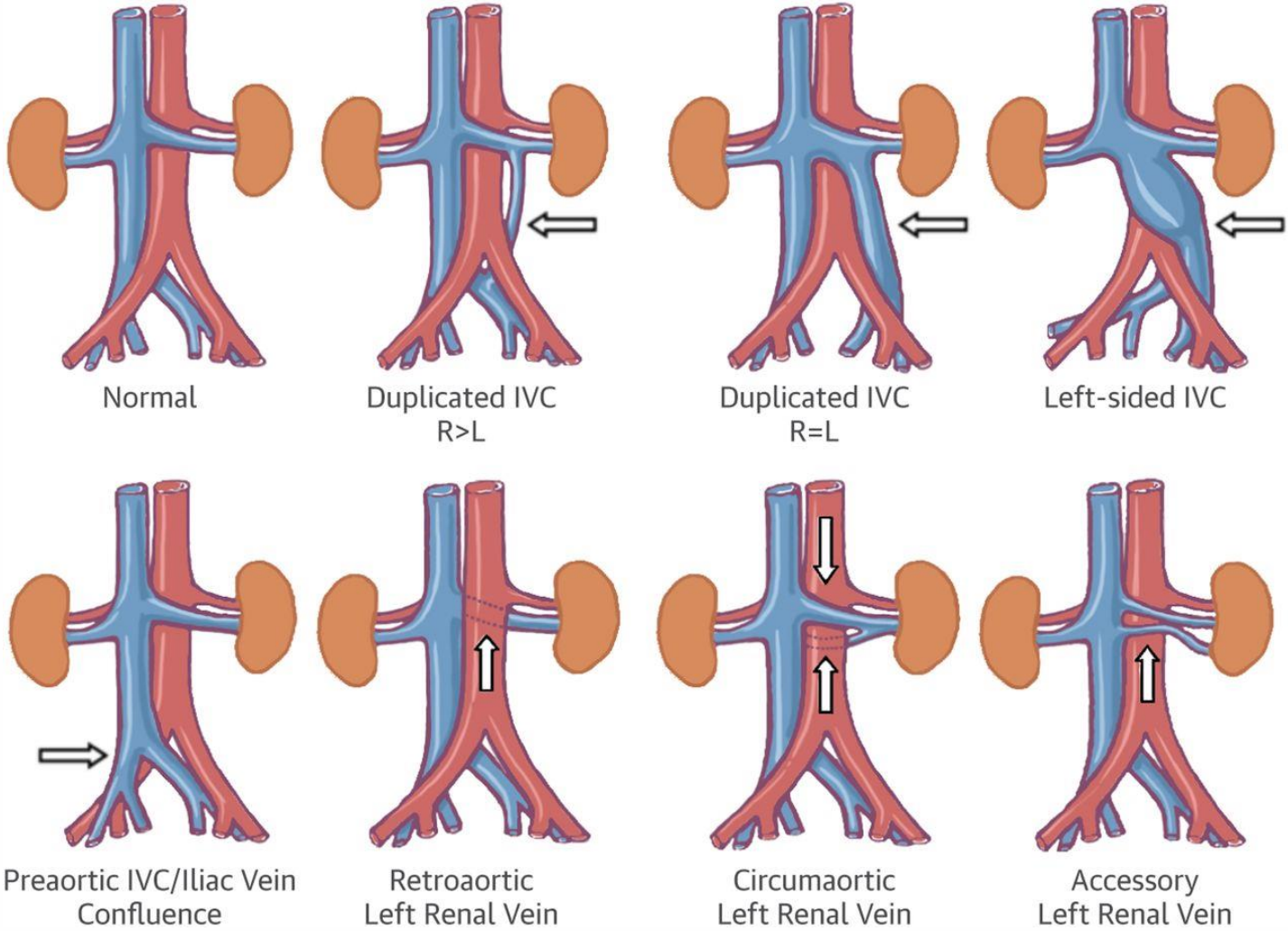


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Learning Points

Here are some of the more common IVC variants



Learning points

- The main challenge in this case other than length was the small size of the veins we were treating. In truly chronic DVT endothelial cells cease to function in the veins and this creates involution and fibrosis¹.
- From a procedural perspective our main issue was inability to attain adequate lumen size on the left. Possibly due to hypoplasia of that segment in addition to the chronic involution. Finally, we achieved a reasonable lumen size by adding more rigid stents to the standard wallstents.
- We still have concern due to the length of the stented region on the left that he may continue to develop restenosis at the common femoral vein. We unfortunately do not have a bendable stent adequate for that purpose in a vein size, 12mm+.



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1. Raju, Seshadri & Neglén, Peter. (2009). Percutaneous recanalization of total occlusions of the iliac vein. Journal of vascular surgery. 50. 360-8. 10.1016/j.jvs.2009.01.061