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Transcatheter embolization of renal–splenic shunt to treat hematemesis

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Abstract

Although the sequelae of chronic liver disease is the most common cause of altered pressure dynamics in the portal and splanchnic circulations, there are other mechanisms resulting in increased venous pressures with subsequent development of splenic and gastric varices; we report a case of a patient without portal hypertension, but with bleeding gastric varices with a presumed splenorenal shunt (SRS) on CT. Venography revealed reversal of flow through the shunt (directed from the renal vein into the splenic vein and out the portal vein), thus an anatomically similar but functionally distinct systemic to mesenteric variant. While anatomically similar to the well-known SRS, the different flow dynamics necessitates a different approach for treatment and important considerations for the use of any liquid embolic.

Keywords

IVC stenosis; varices; splenorenal

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Compliance with Ethical Standards

2. Conflict of Interest

The author declares that he has no conflict of interest.

3. Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Institutional Review Board approval was obtained. This article does not contain any studies with animals performed by any of the authors.

4. Informed consent

Informed consent was obtained from all individual participants included in the study.

5. Consent for publication

Consent for publication was obtained for every individual person's data included in the study.

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INTRODUCTION

The treatment of symptomatic gastric varices via image guided transvenous approaches has made great advances in the past decade [1,2]. The majority of cases result secondary to fibrotic changes in the liver with subsequently increased portal circulation pressures that in turn result in gastric versus esophageal varices with associated risk of hemorrhage [3]. The general principle however, altered venous pressure gradients resulting in development of collateral channels and varices, with increased risk of bleeding, can occur in any pathophysiological context that disrupts the normal venous inflow and outflow channels [2,4,5]. Herein we describe a case in which the resultant pathophysiology (hematemesis from gastric varices) results from a vascular rather than hepatic etiology that in turn alters the flow dynamics and also the appropriate management.

CASE REPORT

A 33 year old female with end stage renal disease secondary to congenital renal hypoplasia status post failed kidney transplant with a right upper extremity arteriovenous fistula, and history of superior vena cava syndrome status post venoplasty, presented with syncope, melena, and hematemesis. Evaluation by esophagogastroduodenoscopy (EGD) revealed medium varices without any active bleeding and no evidence of ulcers. The triphasic CT scan on admission revealed prominent gastric varices and the suggestion of a shunt to the left renal vein (a well known collateral pathway that has been described in the literature [2,6]).

The initial treatment plan was venography and Coil-Assisted Retrograde Transvenous Obliteration (CARTO). Upon venography from the left renal vein there were two salient observations, 1) there was a high-grade stenosis of the inferior vena cava (IVC) and 2) there was retrograde venous flow from the left renal vein to the splenic vein with outflow into the portal vein (Supplemental Video). Subsequent pressure measurements for the IVC-RA gradient confirmed the suspected etiology of the flow reversal resulting in retrograde flow from the IVC to the renal shunt, into the splenic vein and out through the portal vein, secondary to high-grade suprarenal IVC stenosis. Although there was no evidence of hepatic congestion on pre-procedure CT, a diagnostic hepatic venogram was performed to exclude hepatic outflow obstruction (Supplemental Figure 1).

The initial IVC-RA gradient was 26 mmHg (RA 6 mmHg and IVC 32 mmHg). Following venoplasty of the IVC stenosis to 12mm with a high-pressure balloon (Atlas® 12mm x 4cm, BD Bard, Tempe, AZ), the gradient was reduced by more than 50% to 12 mmHg (RA 13 mmHg and IVC 25 mmHg). Attention was then returned to the renal-splenic shunt that had decreased but still exhibited retrograde flow from the left renal vein through varices into anterograde portal vein flow. Coil embolization was performed at the shunt (035, four Azur® 10 × 19 coils Terumo, Somerset, NJ and one Interlock 10 × 20, Boston Scientific, Marlborough, MA). Subsequent venograms showed minimal flow with no collateralization and no opacification of the portal vein from the shunt.

She experienced symptomatic relief without recurrent hematemesis. 10 weeks later she was retreated to optimize the gradient (goal < 10 mmHg). The IVC-RA gradient was 14 mmHg (RA 22 mmHg and IVC 36 mmHg) and was reduced to 3 mmHg (RA 20 mmHg and IVC 23 mmHg) following sequential venoplasty to 18mm (Atlas® 18mm x 4cm, BD Bard, Tempe, AZ). At the most recent follow up (11 months post treatment) she remains symptom free with no recurrent episodes of melena, hemoptysis, or hematemesis.

DISCUSSION

Here we present a case in a patient with severe IVC stenosis resulting in flow reversal through a left renal vein shunt to the splenic vein, causing chronically increased pressures and development of symptomatic varices. The significance of the retrograde flow to the portal circulation as a result of the IVC stenosis (as opposed to hepatic cirrhosis and fibrosis in more common manifestations of gastric varices) also has implications for the appropriate treatment. Since the flow was directed from the renal vein to the splenic vein, it is more aptly described as a renal-splenic shunt (in contrast to the more commonly observed splenorenal shunts that typically develop in the context of hepatic cirrhosis).

A hemodynamically significant, benign stricture of the IVC in the absence of liver pathology, venous thrombosis, veno-occlusive disease, congenital variants, or membrane forming etiologies is an unusual presentation [7]. Evaluation of the patient's cross-sectional imaging studies dating back more than a decade revealed, retrospectively, that this stenosis occurred gradually over time. Approximately 5 years prior to her presentation, the suprarenal IVC began exhibiting narrowing in the absence of any clear inciting event (outside of repeated upper extremity dialysis catheter placement), until gradually developing into a high-grade stenosis. By process of elimination of alternative considerations of the differential diagnosis, the etiology is most likely IVC stenosis secondary to recurrent dialysis catheter placement (she also has superior vena cava stenosis) with altered flow dynamics in the suprarenal IVC. The functional measurements (pressure gradient and altered flow on venography) however, clearly indicated the appropriate means to treat. The critical consideration in this patient, regardless of the specific medical etiology, was to reverse the venous outflow of the shunt; this was achieved with venoplasty of the IVC stenosis.

The secondary consideration was occlusion of the renal-splenic shunt, as described in the literature with technical variations on retrograde transvenous obliteration of the shunts [3,6,8], to assist with treatment of hematemesis, given the bleeding varices noted on EGD. Standard methods of shunt occlusion (coil or balloon assisted) involve distal embolization with a liquid embolic [3,6,8]; in this case had the patient been treated by such an approach, the result would have been an unfortunate embolization of the portal system. Given the flow directionality, the liquid embolic would need to be delivered *proximal* to the coil pack. Due to the concurrent treatment of the IVC stenosis, we elected not to use any gelfoam or glue, since gradual re-reversal of blood flow following venoplasty had the potential for non-target embolization.

The improved understanding of different underlying pathophysiologic mechanisms for upper gastrointestinal varices has been accompanied by the development of disease-specific

endovascular treatments [3,6,8,9]. This report of a renal-splenic shunt provides an direct example of an anatomically similar but functionally distinct variant in comparison to the typical gastric-renal-caval (splenorenal) shunts [2,6].

CONCLUSION

The description of a renal-splenic shunt resulting from flow reversal due IVC stenosis highlights the value of diagnostic venography at the time of treatment. In this case, it allowed differentiation of renal venous hypertension as opposed to portal venous hypertension. Closure of the shunt with coils alone proved effective, however if a liquid embolic or gelfoam were to be used, delivery would need to be proximal to the coils.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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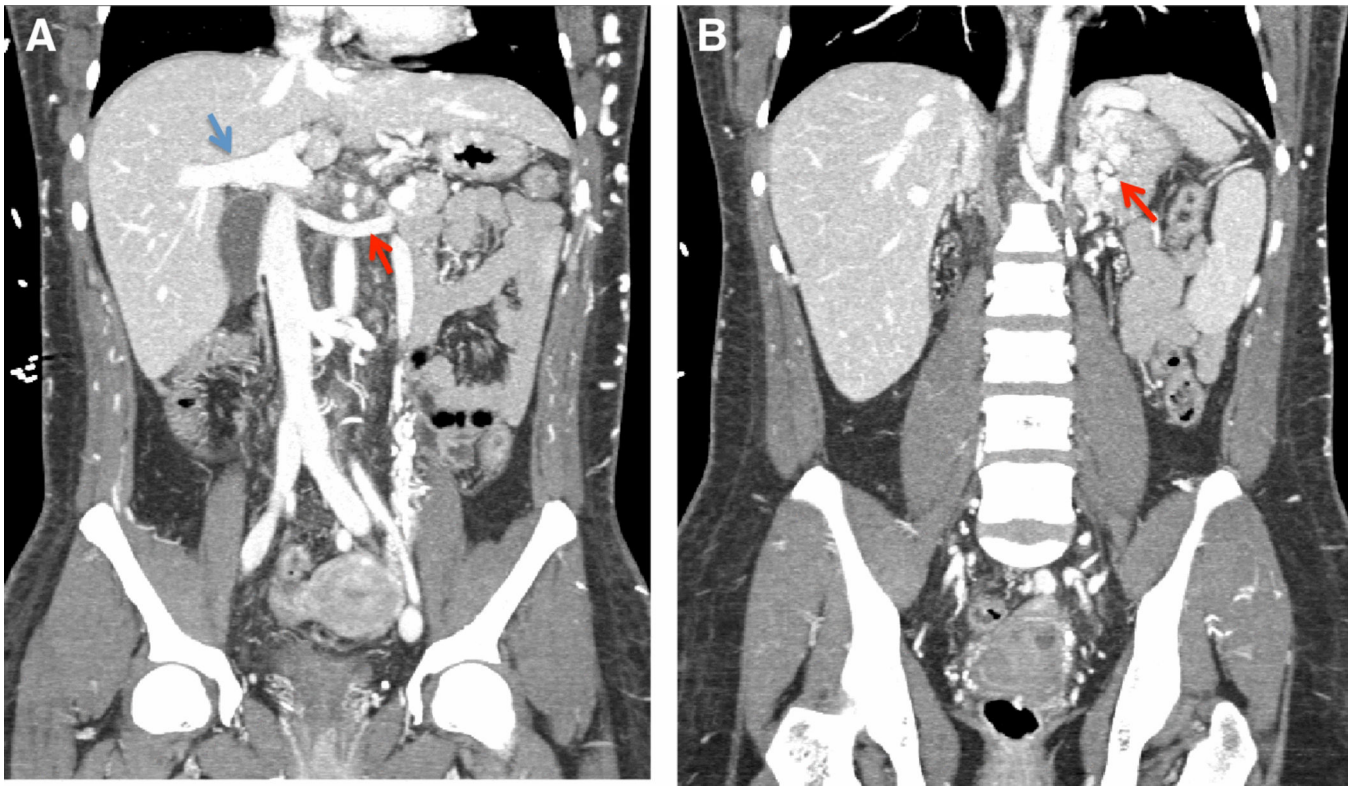

Figure 1:
Coronal pre-procedure images of CT abdomen and pelvis in delayed contrast phase revealing distal IVC stenosis as well as splenorenal shunt with prominent splenic varices as well as subcutaneous varices. A) anterior slice showing the main portal vein (blue arrow) and left renal vein (red arrow). B) More posterior slice highlighting the splenic varices (red arrow) with azygos and portal draining veins.

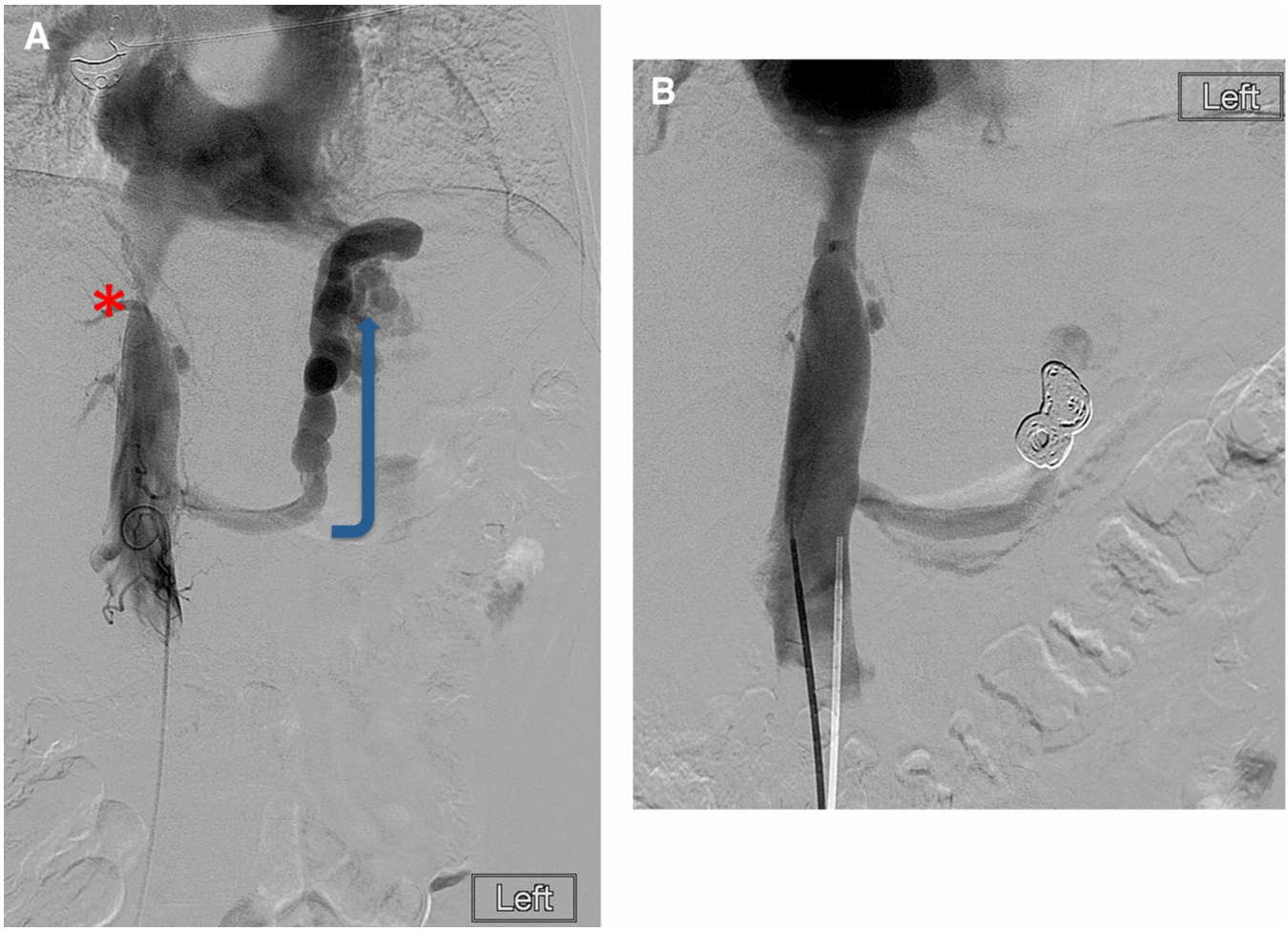

Figure 2:

A) Initial diagnostic venogram with high-grade IVC stenosis and renal-splenic shunt (red asterisk). Blood flow was from the renal vein shunt towards the splenic vein and associated varices (blue block arrow indicates the direction of blood flow of the renal vein to splenic vein). B) Successful post embolization and venoplasty venogram.

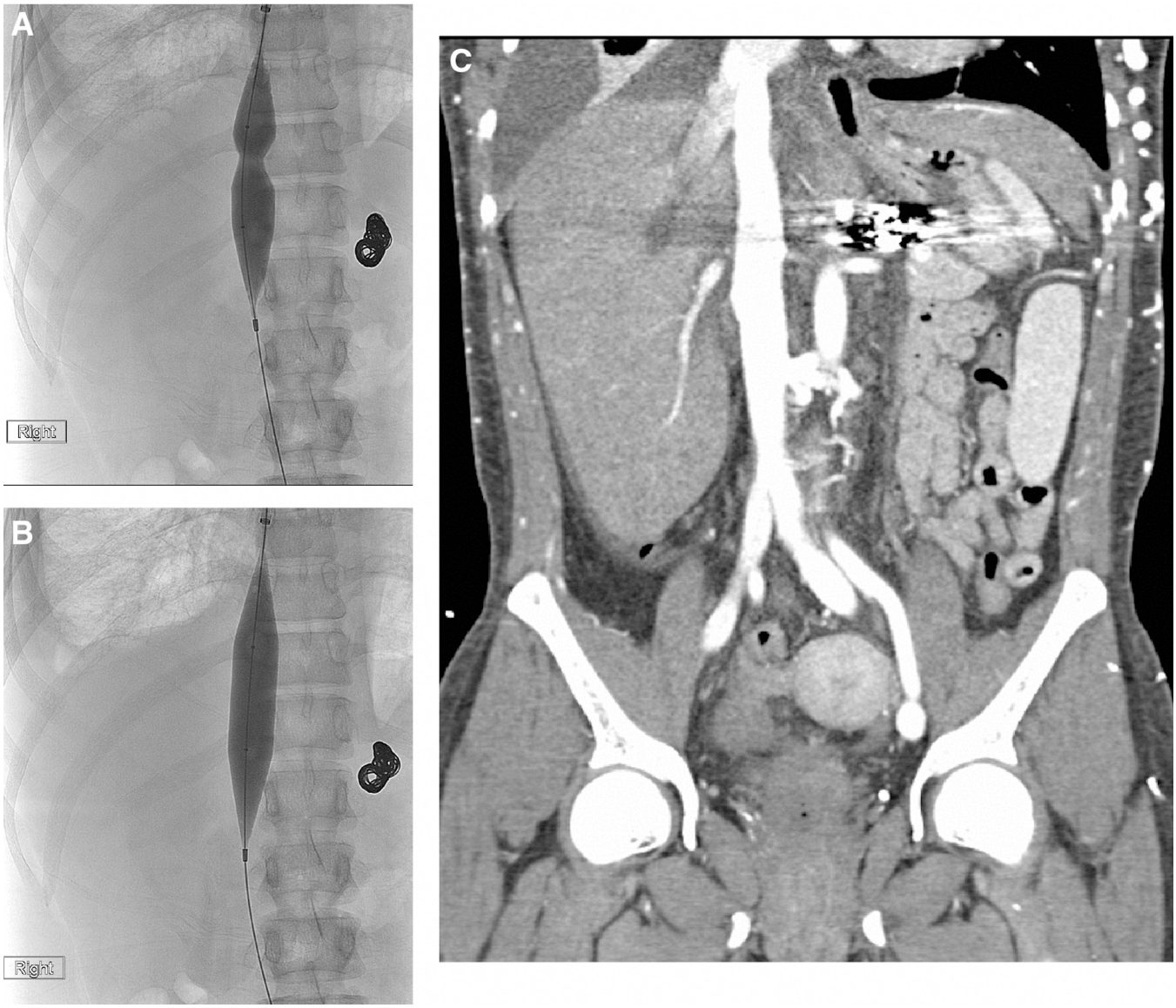

Figure 3:

A) Repeat venoplasty to 18 mm revealing a residual waist. B) The stenosis was eliminated with high-pressure balloon venoplasty to 18mm. C) Post-venoplasty CT venograms in coronal planes at 10 weeks without residual stenosis.