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Case Report
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Treatment Plan for Specific Gastric Varicies: Isolated Gastric Fundal Varicies with Portasystemic Shunt

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Abstract

We present four recent cases of severe gastrointestinal hemorrhage, resulting in profound decrement in hemoglobin. All cases were due to isolated gastric fundal varicies demonstrating a large gastrorenal venous shunt, not amenable to endoscopic sclerotherapy. Each was treated with a combination of TIPS and variceal occlusion with both outflow and inflow occlusion. Follow up for over a year in each case has defined no recurrence of bleeding or variceal development.

Introduction

Gastric varicies represent an extremely difficult disease process in some cases because treatment options have been poor. There are several specific types of gastric varicies. As an extension of esophogeal varices, (GOE1, SARIN) [1] endoscopic sclerotherapy represents an effective treatment path. But isolated cardiac, fundal, varicies (IGV1, SARIN) [1] with a large portsystemic outflow shunt represent a poorly approachable clinical situation by current treatment methods [2]. Not only are such venous vessels often large and tortuous but a direct high flow portasystemic pathway limits treatment options and increases complication potential.

Bleeding from isolated fundal gastric varicies fortunately are a relatively rare process compared to esophogeal varicies (10-36%) [2], however bleeding from such varicies is often massive and therefore more life threatening. This is believed due to lack of treatment options, frequently large vessel size and often (up to 85% of cases) [3,4] demonstrating a large porta-systemic outflow. Endoscopic banding is difficult due to poor support in the gastric fundus and large tortuous vessels. Standard endoscopic sclerotherapy injection has been ineffective because of large vein size and high flow, injection site massive bleeding a disasterous potential complication [2]. Open surgical resection of the varicies has proven unacceptable because of poor general patient status from such a massive bleed limiting patient anesthesia capability and poor anatomic uniformity [2]. Transjugular intrahepatic portasystemic shunt (TIPS) an excellent salvage procedure for esophageal varicial hemorrhage has proven ineffective for isolated gastric varicies with a large portasystemic outflow. Although reasoning behind this observation has not been clarified we believe it likely due to the already low portal pressure in patients with such a large portasystemic outflow. A promising treatment has been multiply reported by Hirota [5], Akahoshi [6] and Katoh [7] among many reports. This option, Balloon-occluded retrograde transvenous obliteration (B-RTO) has been undertaken prophylactically in hundreds of patients in Japan with excellent results. However these patients have, in general been of better Childs classification and have in general not acutely bled. A study in such acutely compromised patients however indicated extremely poor outcomes in a vast percentage. We report four patients, accumulated within one year, with massive bleeding from isolated gastric varicies, treated with a combination of TIPS (despite normal to low corrected portal pressure) and varix obstruction utilizing a combination of outflow occlusion with an Amplatzer II occlusion device and inflow obliteration either with gelfoam soaked in sclerosant sodiumtetradecyl sulfate or an Amplatzer II device if a single large source to the gastric varix is identifiable.

Case Presentation

Case 1

A 45 year old female with no known history of hepatitis, cirrhosis or encephalopathy presented acutely with an initial episode of massive gastrointestinal hemorrhage, hemoglobin decreased to 3 grams. Endoscopy revealed no esophogeal varicies or gastric ulcer. However, isolated gastric fundal varicies with a probable site of prior hemorrhage was defined (Figure 1). No endoscopic treatment was deemed appropriate. Consultation for TIPS was requested. As part of the workup for that

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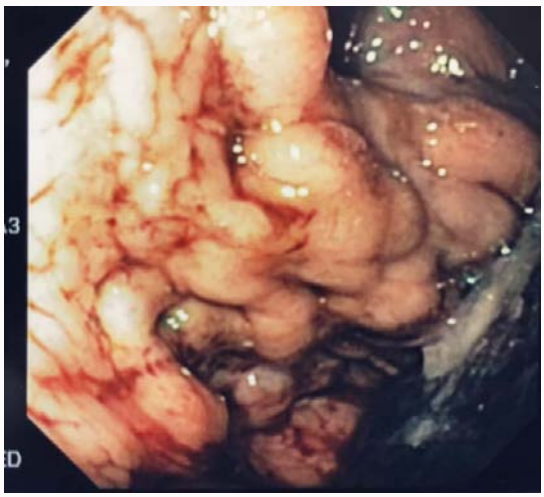

Figure 1: Endoscopic view of isolated gastric varicies, IGV1 (Sarin, 1) which led to profound hemorrhage in this 45 year old female. Hemoglobin fell to 3 gm at admission.

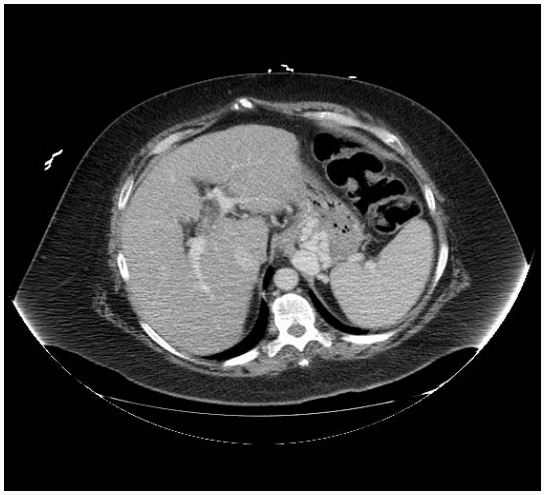

Figure 2A: CT transverse image, intravenous contrast but no oral contrast, defining large isolated gastric variceal set. Inflow seems to come from short gastric veins from the splenic hilum.

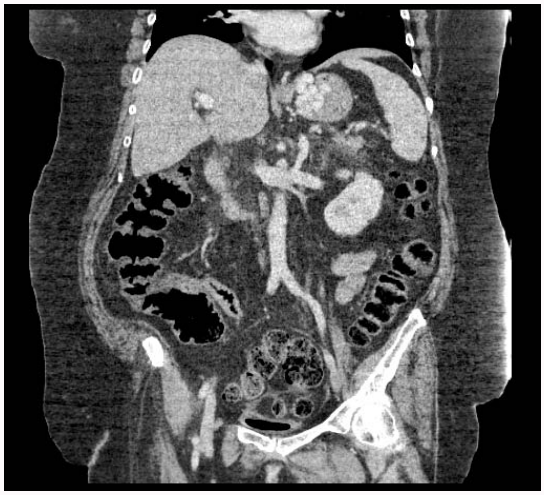

Figure 2B: Coronal CT image, defining the large gastrorenal outflow vein.

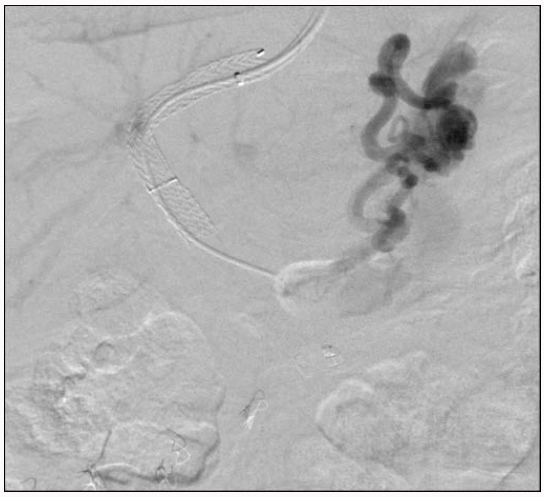

Figure 3: Post TIPS angiographic anatomy defining the large gastro-renal venous shunt. The TIPS led to profound encephalopathy and coma.

consultation we obtained a CT abdomen with arterial and delayed venous phase imaging. We defined the gastric varicies but also a large, 10mm, gastrorenal venous shunt. (Figure 2A and B) This large portasystemic communication certainly resulted in a low corrected portal vein pressure. Instead of facing a situation in which TIPS is known to be advantageous, elevated corrected portal vein pressure with esophageal variceal hemorrhage, we faced a much less favorable pathophysiological situation. Initial consultation assessment was that TIPS would likely not alleviate bleeding potential from high flow gastric varicies and possibly lead to hepatic encephalopathy.

After due consideration, discussion with extensively experienced national colleagues and literature review we considered B-RTO but instead decided to initially assess portal pressure directly as the initial portion of a TIPS procedure. After full and thorough informed consent, clarification of a probable stepwise process the patient agreed to initial TIPS procedure, possible later gastric variceal occlusion. Under general endotracheal anesthesia we initiated transjugular access to the hepatic venous system. Transhepatic access to the portal system was slightly more difficult than usual, possibly due to comparatively low corrected portal pressure. Once in the portal system corrected mean portal pressure was measured 13mm Hg, consistent with wedged hepatic vein pressure. Again the treatment options were considered and we decided to proceed with TIPS creation. Once completed, corrected portal pressure fell to approximately 8mm Hg.

We decided to observe patient outcome; the patient became profoundly encephalopathy, mental status severely impaired, ammonia level 145. We then reevaluated CT and TIPS angiographic anatomy (Figure 3), reconsidering B-RTO. Given lack of experience with B-RTO in the United States, ease of access to the portal system through the TIPS shunt and personal experience with treatment alternatives; we planned an alternative. While confirming reduction of the portasystemic outflow with an Amplatzer ll occluder; we sequentially began to inject gelfoam soaked with sodium tetradecyl sulfate as a sclerosing agent. We injected the sclerosing pledgets into the multiple major primary inflow sources to the gastric varicies. There was no suggestion of pulmonary emboli or fibrosis, encephalopathy altered level of consciousness resolved over 48 hours. Follow up endoscopy and CT at three months showed no evidence of gastric varicies; no gastrointestinal hemorrhage has occurred over two years follow up.

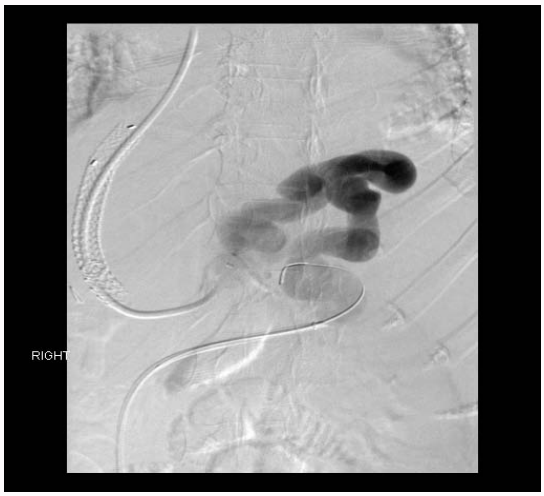

Figure 4: Case three, after TIPS two years prior a large portasystemic shunt remained a huge, high flow gastrorenal shunt. This shunt remained open over that interval, ultimately repeat gastrointestinal hemorrhage occurred, A huge endoscopically confirmed from the isolated gastric varicies.

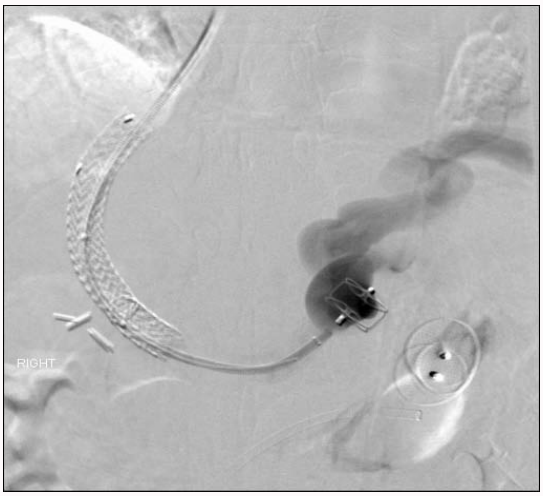

Figure 5: After mapping single inflow from the coronary vein and primary outflow from the gastrorenal shunt of the isolated gastric varicies, we occluded outflow initially with a 22mm Amplatzer II occlusion device. We followed immediately by occluding inflow with a 16mm diameter Amplatzer II occlusion device. The patient demonstrated no further bleeding to 6 months and no gastric varicies at 3 month follow up CT.

Case 2

Within six months of Case 1 an exactly similar patient presented acutely with profound gastrointestinal hemorrhage, hemoglobin reduced to 5 grams and endoscopic demonstration of isolated fundal gastric varicies. With our experience from Case 1 we proceeded to CT defining arterial and venous phase, defining the large gastric varicies and a large gastrorenal portasystemic shunt. We proceeded in exactly the same sequence; TIPS creation day 1 during which we found a low corrected portal vein pressure, likely due to the large portasystemic collateral. Multiple inflow branch occlusions were accomplished while outflow was diminishing after Amplatzer II plug placement successful on day 2. The 24 hour interval was useful to assess inflow anatomy and plan sequential outflow and subsequent inflow branch occlusion. Again, a three month endoscopic and CT follow up demonstrated no evidence of gastric varicies. No recurrent gastrointestinal hemorrhage at one year.

Case 3

Our third case developed in a slightly different sequence. Initial bleeding two years prior was related to large isolated gastric fundal varicies. This was before we fully understood the importance large varix diameter and torrential flow in such gastrointestinal hemorrhage as different from esophageal varicies. At initial presentation a TIPS was performed although measured corrected portal venous pressure was low. The large gastric varix with gastrorenal shunt remained despite the TIPS. Bleeding seemed controlled, the patient did not develop significant hepatic encephalopathy. She was discharged and stable for two years. Then a repeat episode of severe gastrointestinal hemorrhage developed. The CT defined the large gastric varicies with gastrorenal shunt outflow. The patient was returned to the interventional laboratory. From a transjugular approach we investigated the TIPS, it remained patent. Pressure in the portal system was not elevated. A large, inflow coronary vein to the large isolated gastric fundal varicies was accompanied by a very small short gastric vein inflow. A huge gastrorenal outflow was confirmed (Figure 4). We then dilated the TIPS shunt to confirm stability. We proceeded to both primary inflow and outflow occlusion of the gastric varicies with Amplatzer II occlusion devices (Figure 5). We did not chase the small short gastric inflow believing it not significant if outflow totally occluded. Follow-up at 3 months demonstrated minimal residual gastric varicies and no further bleeding.

Case 4

51 year old female admitted emergently for upper gastrointestinal hemorrhage. Admission two years previous for the same diagnosis, endoscopy defined esophageal varicies; they were successfully banded. The patient was lost to follow up but reported abstinent from alcohol. She recently began drinking alcohol, leading to the current episode. The patient has Type II diabetes and hypertension, but is otherwise reported in good health. Endoscopy at this admission defined a large gastric variceal set with a single esophageal varix defined. It was therefore unclear if the current pattern represented EOG1 esophagogastric varicies or IGV1 isolated gastric varicies. Portal and hepatic vein ultrasound defined hepatopetal portal vein blood flow. There was no thrombus in the portal vein and good hepatic vein flow. The INR was 1.2; Total bilirubin 0.8; creatinine 1.0; ammonia level 51. This gave a Meld Score of 4, indicating 90 day mortality 3%. We were asked to evaluate the patient for TIPS to control gastric variceal hemorrhage. A CT abdomen was obtained with intravenous contrast only. This demonstrated a dense gastric variceal set, seemingly supplied only from a prominent coronary vein, no obvious short gastric supply, no obvious esophageal varicies and a large, approximately 9mm diameter draining gastrorenal vein. Given this information we suspected IGV1 isolated gastric varicies. We proceeded to TIPS after fully informed consent obtained. We decided if corrected portal pressure was significantly elevated TIPS alone may be sufficient treatment. A normal corrected portal pressure would not resolve the gastrorenal shunt, high shunt flow would be maintained with a high likelihood of further variceal distention and probable massive gastric variceal hemorrhage. In addition the increasing flow in the varicies bypassing the liver would lead to progressive encephalopathy. Standard TIPS procedure was straightforward, first pass entry to the portal venous system; directly measured corrected portal venous pressure 13mmHg, minimally elevated. The large coronary vein supply to the isolated gastric varicies and drainage through the gastrorenal shunt. There was a single additional small supplying vein branch to the gastric varicies arising

from the renal vein. We completed the TIPS procedure, dilating the transhepatic course to 8mm, placed a Viator 10mm diameter covered stent and post dilated to 8mm. Reevaluation of gastric variceal flow demonstrated persistent flow toward the varicies, even though post TIPS corrected portal pressure only 5mmHg mean. Venous access to the left renal vein from the left common femoral vein was then accomplished. Occlusion of the gastric variceal inflow coronary vein and gastrorenal vein outflow near simultaneously was accomplished without difficulty. Amplatzer II plugs, 14 mm into the inflow coronary vein and 16 mm into the outflow gastrorenal vein were placed through Boston Scientific curved 7F renal sheaths (Figure 5). We then reevaluated corrected portal vein pressure, 6mmHg mean. Follow up angiogram demonstrated essentially no flow to the gastric varicies. The patient did well, discharged 2 days later without further bleeding. Abdominal CT at 1month defined no evidence of the gastric varicies; there was no bleeding in the interval.

Discussion

We have now identified 4 cases of isolated gastric varicies, IGV1 (Sarin, 1) at one institution in 18 months. We believe this may indicate a genetic bias in our population or more likely under diagnosis of this condition in the general population. Such possible under diagnosis may be related to endoscopic difficulty defining the gastric fundus, the lack of effective treatment of this condition and the massive resultant bleeding which can be rapidly fatal [2]. Given the torrential blood flow through the portasystemic shunt even small ulcerations in these varicies can lead to death before reaching treatment. Endoscopic treatment of gastric varicies is very challenging and therefore limited due to gastric fundal size, complexity and interconnection of varicies which means that placing a hole into such a complex, high flow vascular system could produce worse hemorrhage and exsanguinations [2,8,9] even using the latest tissue occlusive material such as 2-octyl cyanoacrylate. The anatomy of IGV1 has been clarified and a seemingly completely successful treatment plan, in the short term, defined. By occluding both inflow and outflow of the gastric variceal network we believe we have removed the etiology of gastrointestinal hemorrhage in a more complete but similar manner to B-RTO only sclerotherapy [10]. The TIPS is essential for accurate definition of portal pressure and variceal anatomy, especially possible multiple inflow sources. A non elevated corrected portal vein pressure indicates that TIPS, a decompressive procedure, alone will be unlikely to resolve flow through a large, tortuous portasystemic communication [10]. However, the liver disease underlying this physiology, hepatitis or cirrhosis produces the increased resistance to normal portal venous blood flow. This results in various collateral portasystemic flow patterns. Defining why esophageal (EOG1), esophagogastric (EOG2) or isolated gastric varicies (IGV1), all of which can result in life threatening hemorrhage, develop has not yet been solved. Indeed, understanding the why of development of Ascities,, likely due to preferential stagnant flow through the mesenteric veins has yet to be clarified. But understanding this wider difference may be an easier step than determining why different variceal collateral patterns develop. Suggested treatment plans for isolated gastric varicies include TIPS alone. But as cases 1 and 3 have shown TIPS alone could result in terminal encephalopathy (Case 1) or continued gastric variceal flow and eventual rebleeding (Case 3). The presumed goal of the Japanese groups treatment plan, Balloon occlusion retrograde transvenous injection (B-RTO) of tissues adhesive is an attempt to completely seal the complex, interconnected isolated gastric variceal network. Without controlling inflow the retrograde penetration of cyanoacrylate will be unpredictable. Additionally the volume and tortuosity of the isolated gastric variceal network, adhesive injection pressure and time will result in further variability of retrograde penetration into the network. Insufficient volume of occlusion could lead to development of alternative outflow pathways and continued hemorrhage risk. Too vigerous injection could lead to fatal portal venous occlusion as reported in one of Morimasas cases. We believe the stepwise treatment plan we have presented represents a more complete understanding of variceal anatomy and physiology; a graduated treatment plan to avoid under treatment or overtreatment. This understanding and the inflow and outflow pathway occlusion with the Amplatzer II device represents a more controlled, reproducible, safer treatment option. The range of pathologic processes possible within the presentation of isolated gastric varicies is neither a one size fits all treatment plan nor a treat it and forget it option. Obviously the underlying liver dysfunction due to portal triad scaring must be monitored and evaluated for progression, TIPS patency, development of alternative portal flow pathways and alternate sequels.

Conclusion

We have presented a treatment plan refinement for isolated gastric varicies (IGV1, Sarin). It combines the primary option prevalent in the United States, TIPS only, with the primary treatment plan recommended in Japan, Balloon occlusion retrograde transvenous injection of tissue adhesive (B-RTO). We have added physiologic assessment, defining corrected portal venous pressure as a treatment decision point. We also suggest inflow anatomic mapping of the complex interconnected gastric variceal network to determine the best means of inflow occlusion. We have used the Amplatzer II occlusive plug to seal large primary inflow and outflow veins because of stability of placement in the high flow, tortuous gastrorenal shunt. We have added sclerosant soaked gelfoam sponge injection into smaller multiple inflow sources to gastric variceis after outflow occlusion with the Amplatzer II. This outflow vessel is often difficult to ascend because of severe tortuosity precluding catheter stability adjacent to the left renal vein. The ease of use of the Amplatzer II in high flow positions as well as the ability to remove the device after deployment before release add to procedure safety and successful isolated gastric variceal occlusion.

References

1. Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, Classification and Natural History of Gastric Varicies: A Long-Term Follow-up Study in 568 Portal Hypertension Patients. Hepatology, 2005; 6: 1343-1349.

2. Hashizume M, Akahoshi T, Tomikawa M. Management of Gastric Varicies. Journal of Gastroenterology and Hepatology. 2011; 26: 102-108.

3. Ryan BM, Stockbrugger RW, Ryan JM. a Pathophsiologic, Gastroenterologic and Radiologic Approach to the Management of Gastric Varicies. Gastroenterology, 2004; 126: 1175-1189.

4. Matsumoto A, Kitamoto M, Imamura M. Three-Dimensional Portography Using Multislice Helical CT is Clinically Useful for Management of Gastric FundicVaricies. American Journal of Radiology, 2001; 176: 899-905.

5. Hirota S, Matsumoto S, Tomita M, Sako M, Kono M. Retrograde Transvenous Obliteration of Gastric Varicies. Radiology, 1999; 211: 349-356.

6. Akahoshi T, Hashizume M, Tomikawa M, Kawanaka H, Yamaguchi S, Konishi K, Kinjo N, et al. Long-Term Results of Balloon- Occluded

Retrograde Transvenous Oblireration for Gastric Variceal Bleeding and Risky Gastric Varicies: a 10 Year Experience. Journal of Gastroenterology and Hepatology, 2008; 23: 1702-1709.

7. Katoh K, Sone M, Hirose A, Inoue Y, Fujino Y, Onodera M. Balloon-Occluded Retrograde Transvenous Obliteration for Gastric Varicies: The Relationship Between the Clinical Outcome and Gastrorenal Shunt Occlusion. BioMed Central Medical Imaging, 2010; 10: 2.

8. Wu BU, Carr-Locke DL. The Problem with Gastric Varicies. Medscape General Medicine, 2006; 8: 72.

9. Goff JS. Treatment of Gastric Varicies in 2005: Is There a Role for Endoscopy? Visible Human Journal of Endoscopy, 2005; 4: 1-4.

10. Ninoi T, Nakamura K, Kaminou T, Nishida N, Sakai Y, Kitayama T, et al. TIPS Versus Transcatheter Sclerotherapy for Gastric Varicies. American Journal of Roentgenology, 2004; 183: 369-376.