Bleeding isolated gastric varices: a retrospective analysis

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Objective: Isolated gastric varices (IGV) are rare and are believed to be associated with left-sided portal hypertension. We studied patients presenting with bleeding from IGV and compared them with those bleeding from both esophageal and gastric varices. Methods: A retrospective analysis of 14 patients with bleeding from IGV was carried out. Portovenography findings (pattern of collateralization and natural shunts) in these patients were compared with a matched group of 69 patients with both esophageal and gastric varices. Results: Of 14 patients with IGV, 2 had isolated splenic vein thrombosis and 12 had generalized portal hypertension. Portovenograms in 11 of the letter 12 revealed predominantly 'left-sided' collateralization in 8 patients as compared to 17 of 69 (25%) patients with esophageal and gastric varices (p=0.004); natural shunts were seen in 6 of 11 cases and 15 of 69 (22%) patients in the two groups, respectively (p=0.05). Abdominal devascularization operation gave good short- and long-term control of bleeding. Conclusions: Contrary to belief most patients with isolated gastric varices may have generalized portal hypertension rather than splenic vein obstruction as the cause and hence should be treated by a more extensive procedure than just splenectomy. The IGV could be a result of predominant collateralization to the retroperitoneal area (left-sided collateralization and natural shunts) rather than the usual pattern to the azygos system which results in esophageal varices. [Indian J Gastroenterol 1999;18:69-72]

Key words: Portal hypertension, splenic vein thrombosis

The presence of gastric varices in association with esophageal varices is well described. The frequency of this association varies from 6% to 100%; we have previously reported a frequency of 78% at endoscopy. However, isolated gastric varices (IGV) in the absence of esophageal varices are rare and have been described classically to occur in patients with splenic vein obstruction, a condition also termed as 'left-sided' or 'segmental' portal hypertension. These varices are usually located in the gastric fundus.

Occasionally, IGV may also occur in generalized portal hypertension where the block is in the portal vein or liver. The present report is a retrospective analysis of the frequency, clinical features, etiology, diagnostic investigations, portovenography findings and management in patients with IGV presenting to us over an 11-year period.

Methods

Between January 1986 and January 1997, 540 patients with portal hypertension were treated for variceal bleeding in our unit which specializes in portal hypertension surgery and is located in a tertiary care center. Of these, 14 had IGV at emergency upper gastrointestinal endoscopy. Hospital records of these patients were reviewed. None of those patients had undergone sclerotherapy for esophageal varices in the past. All patients had undergone clinical and biochemical evaluation to establish the cause of portal hypertension and to assess the status of liver function and ultrasonography for assessment of portal and splenic veins and hepatic parenchyma.

Eleven of the 14 patients had undergone emergency portovenogram. The portovenograms (9 splenoportovenogram; 2 venous phase of arterioprtovenogram) were reviewed afresh with specific reference to the patency of the splenic vein, portal vein, pattern of collateralization (feeding vessels), and the presence of natural gastrorenal or lienorenal shunts, by a radiologist blinded to the clinical details. The collaterals were labelled as short gastric (coursing from spleen to fundus of stomach), retrogastric (coursing vertically from stomach towards the splenic vein) and left gastric (coursing from splenic /portal vein to lesser curvature of the stomach), as described by us earlier. Prominent short gastric or retrogastric collateralization was labelled as a 'left sided' pattern. A natural gastrorenal or lienorenal shunt was assumed to be present when in addition to directly visualizing the shunt, the renal vein and/or the inferior vena cava was occluded at portovenography. The shunts were graded as described by Watanabe et al. Portovenography findings in this group were compared with those in a group of 69 consecutive patients, well matched for age, sex and etiology of portal hypertension, with esophageal and gastric varices, who were the basis of a previous report. A color doppler study of the portal venous tree was available in one recent patient.

Endoscopic injection sclerotherapy with 3% aqueous phenol was attempted in one initial patient and with cyanoacrylate glue (Histoacyl blue; Braun-Melsingen, Germany) in two patients. Failure of injection therapy was an indication for surgery. All other patients were primarily subjected to surgery. Patients with isolated splenic vein thrombosis were treated by splenectomy alone and those with generalized portal hypertension underwent transabdominal gastric devascularization consisting of ligation of the short gastric, left gastric and retrogastric vessels. The
esophagus was not transected or devascularized and in one patient splenectomy was added. Prominent gastric varices on the serosal aspect of the stomach, if any, were underrun with continuous interlocking silk sutures.

The diagnosis of extrahepatic portal vein obstruction (EHPVO) was based on demonstration of typical portal cavernoma on ultrasonography or portovenogram. The diagnosis of cirrhosis and non-cirrhotic portal fibrosis (NCPF) was made on intraoperative liver biopsy. Patients were followed up at 3-month intervals for one year and every 6 months thereafter. At follow up, endoscopic evaluation was performed to document recurrence of varices. Portovenography was repeated at one year in some patients to study the reformation of collaterals.

Statistical comparison was made using the \( \chi^2 \) test with Yates' correction, wherever applicable. A \( p \) value <0.05 was considered significant.

**Results**

**Incidence**

The overall frequency of IGV in patients presenting with variceal bleeding was 2.6% (14 of 540). Of the 14 cases (age 7-62 y, mean 46; 9 males, 5 females), in two the IGV were secondary to isolated thrombosis of the splenic vein. In the remaining 12 patients, portal hypertension was due to EHPVO in 5, NCPF in 4, and cirrhosis in three. Of the three patients with cirrhosis two were in Child’s B class and one in Child’s C class. The etiology of portal hypertension in the remaining group of 528 patients with esophageal varices with or without gastric varices was EHPVO in 34%, NCPF in 14% and cirrhosis in 52%.

**Clinical features**

Twelve patients presented with hematemesis and two with melena. Ten patients had splenomegaly and three had ascites.

**Endoscopic findings**

IGV were located in the fundus in all 14 patients. Emergency endoscopy revealed active bleeding from the IGV in 8 patients; in the remaining 6 patients, although the IGV were not actively bleeding, these were assumed to be the source of bleed as they were the only lesion in the upper gastrointestinal tract. Two patients had associated portal gastropathy in the fundus and body.

**Portovenogram findings**

The splenic vein was confirmed to be patent in 7 of 11 patients who had portovenography. In 4 patients (2 EHPVO, 1 cirrhosis) there was associated thrombosis of the splenic vein. Color doppler revealed a patent splenic vein in the only patient in whom it was performed. Left-sided collateralization was seen in 8 of 11 patients with IGV; in comparison 17 (25%) of 69 historical controls with both esophageal and gastric varices had this (p=0.004). Natural gastrorenal or lienorenal shunts were seen in 6 (54%) patients with IGV, as compared to 15 (22%) of 69 patients with both esophageal and gastric varices (p=0.05). There was a 3+ shunt (>10 mm) in 2 patients with IGV, 2+ shunt (5 to 10 mm) in 1 patient, and 1+ shunt (<5 mm) in 3 patients. In addition, large intercostal collaterals were seen in 4 patients, all of whom also had predominant left-sided collateralization.

**Treatment**

In one patient, sclerotherapy with 3% aqueous phenol was successful in controlling the initial bleed but the patient rebled in 12 hours and was operated on. Another patient was initially controlled with cyanoacrylate glue injection but also had a rebleed after 48 hours and was operated on. An attempt at glue injection in a third patient had to be abandoned because of torrential hemorrhage. These three, along with five others, were subjected to an emergency devascularization procedure. Acute bleeding could be controlled in all 8 patients. The remaining 4 patients were operated on electively and underwent the same procedure. The 2 patients with isolated splenic vein thrombosis were treated by splenectomy.

**Mortality**

Two patients (both with cirrhosis) died in the immediate postoperative period (<30 days), one each of liver failure and bronchopneumonia.

**Follow up**

Two patients were lost to follow up. The remaining 10 patients are alive for 21 to 98 months (mean 44). One patient bled from recurrent esophageal varices 18 months after surgery; he was treated successfully with sclerotherapy. The other patients have not bled. In 8 patients, there has been no reappearance of varices at endoscopy more than three years after surgery. Repeat splenoportograms in 3 patients with generalized portal hypertension at one year after surgery revealed absence of collaterals around the gastric fundus.

**Discussion**

The 2.6% frequency of IGV seen in our patient population is similar to the figures of 1.6% to 3.5% reported in other studies.\(^6\) In this study isolated splenic vein thrombosis was seen in only 2 of 14 patients with IGV. Thus, a majority of patients did not have left-sided portal hypertension but had obstruction to the portal vein (EHPVO) or in the liver (NCPF, cirrhosis). This finding is contrary to the belief that IGV is a specific sign of isolated splenic vein occlusion.\(^7\) This observation was also made by Levine et al\(^6\) who found that seven of eight patients with IGV had generalized portal hypertension.

In this study, IGV were seen in all three common causes of generalized portal hypertension seen in India, viz. cirrhosis, NCPF and EHPVO. When the etiology of portal hypertension in this group was compared to the entire group, there was no significant difference.
The mechanism of IGV formation in splenic vein thrombosis is easy to understand. The increased splenic pulp pressure is transmitted to the fundic plexus via the short gastric veins which in turn drain into the portal vein via the coronary vein, thus bypassing the block in the splenic vein. The dilated fundic veins form the isolated gastric varices. The likely mechanism of formation of IGV in generalized portal hypertension with a patent splenic vein has also been postulated.9 Watanabe et al9 found certain typical features in patients with IGV. These included predominantly ‘left’ type of collaterals, marked gastroesophageal shunts, low portal venous pressures and a type ‘c’ flow in the superior mesenteric vein, i.e. all blood from the superior mesenteric vein flows into the splenic and left gastric veins with no opacification of the portal vein. Although we have not performed pressure studies our finding on portovenogram of dominant short gastric or retrogastric collaterals in 8 of 11 patients is suggestive of a predominant ‘left-sided’ collateralization to the retroperitoneum.

When this pattern was compared to historical controls with esophageal and gastric varices, the difference was significant. In addition, the presence of natural gastroesophageal shunts in a significantly higher number of patients as well as intercostal collaterals in 4 patients also suggest similar collateralization to the left side, away from the esophagus. These findings could suggest that IGV formation represents a predominantly ‘left-sided’ collateralization as opposed to the normal ‘right-sided’ shunting into the ayzygos vein which results in the development of esophageal varices. The reason why some patients collateralize to the left side is an open matter of conjecture. Four of the 12 patients in our study had splenic vein thrombosis associated with a block in the portal vein or liver. This is rare with EHPVO, a condition in which such an extension of the thrombotic process from the portal vein to the splenic and other splanchnic veins is known.10 The contribution of the associated splenic vein thrombosis to the formation of IGV is not clear to us at present.

The incidence of bleeding from gastric varices in general is much lower than from esophageal varices. However, in case of IGV, since they form the main collateral pathway they are more likely to bleed than gastric varices associated with esophageal varices.6 Although sclerotherapy has been used to control bleeding from gastric varices the results have not been satisfactory, especially when dealing with fundal varices.11 This has been our experience too. Intravarical glue injection has been advocated as a useful method of controlling bleeding from fundal varices.12 Although our experience with it is limited, it seems more suited for small localized bunches of varices rather than a large mass of varices as was the case in our two patients in whom glue injection failed to control bleeding.

We have been performing transabdominal gastric devascularization with satisfactory results for bleeding gastric varices in patients in whom the esophageal varices have been obliterated by sclerotherapy.13 A similar procedure has been used in patients with IGV. We believe that splenectomy is not necessary as part of devascularization unless there is associated symptomatic hypersplenism. Further, retaining the spleen enables a lienorenal shunt to be performed if required later. Theoretically, there is a possibility of re-formation of collaterals after a devascularization procedure. However, in the small number of patients in whom we have performed a postoperative venogram this has not occurred.

It is possible that the low postoperative mortality seen in this report is partly because of the fact that a majority of our patients had normal liver functions. However, the good long-term control rate indicates that devascularization is effective in preventing rebleeding and reformation of varices. Alternative surgical procedures used to tackle gastric varices include underrunning of the varices through a gastrotomy,14 proximal gastrectomy and decompression by a shunt.15 Although a shunt procedure is an option, it is time consuming and thus unfavorable for the emergency situation. Moreover, it requires patent veins, which is often a problem in patients with EHPVO.10

Transjugular intrahepatic portosystemic shunt is now used in the West to control variceal bleeding as a bridge to transplantation. It cannot be recommended for a situation like ours where non-cirrhotic causes of portal hypertension are common and the portal vein is blocked (EHPVO). In addition, this procedure is expensive and not easily available in our country.

To conclude, a significant number of patients with isolated gastric varices have generalized portal hypertension as the underlying cause. It is important to identify these patients as they cannot be treated with splenectomy alone and require a more extensive procedure. Gastric devascularization seems to give good control although confirmation of our experience with larger numbers and longer follow-up is necessary. In the various classifications proposed for gastric varices16 IGV have usually been classified as a separate category. In a previous report we had subdivided gastric varices depending on their location in the stomach.3 We now propose that isolated fundal (gastri) varices be subdivided into subtype ‘a’ associated with splenic vein thrombosis and subtype ‘b’ associated with generalized portal hypertension. This division is important since the therapeutic approach to the two subtypes is different. Subtype ‘a’ can be treated with splenectomy alone whereas subtype ‘b’ will require devascularization or a shunt.

References

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