Study of Patient Admitted For Portal Hypertension At Tertiary Care Hospital

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Abstrct: Introduction: The portal venous system is the main vascular system supplying another organ, the liver. Noncirrhotic portal hypertension (NCPH) is a heterogeneous group of vascular diseases that lead to portal hypertension (PH) with normal or mildly elevated hepatic venous pressure gradient and preserved liver synthetic function. Surgery in NCPH aims to bypass the portal resistance site by creating portoportal or, more commonly, portosystemic shunts (PSS); when not feasible, techniques that involve esophagogastric devascularization (direct ligation of varices or esophageal transection), with or without an associated splenectomy may be undertaken. <u>Methods:</u> Prospective interventional study consisted of 30 patients with portal hypertension was carried out at Department of Surgery, New Civil Hospital, Surat during December-15 to January-16. <u>Results:</u> Male female ratio was 7:3 and majority among age group of 31 to 40 years. All 30 patients came with complaint of haemetemesis and abdominal lump in the form of splenomegaly. All patients were having oesophageal varices and features of hypersplenism. No pateint was having ascitis. Portal vein was 170 min. Mean duration of hospital stay was 7 days. <u>Conclusion:</u> After splenectomy with devascularisation surgery, blood counts were increased and patients had relief from symptom of haemetemesis which was assessed 1month, 3 months and 6 months after Operation. [Bina V NJIRM 2017; 8(3):89-92]

Key Words: Devascularisation , Portal Hypertension

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Introduction: The veins of the portal venous system are unique since, unlike other veins, they do not drain directly into the systemic venous circulation or the heart. Instead, the portal venous system is the main vascular system supplying another organ, the liver. Normal total liver blood flow is approximately 1600 ml/minute; 1200 ml (75%) is delivered by the portal vein and 400 ml by the hepatic artery. The liver receives 25-30% of the cardiac output. Blood flow through the liver is higher than in any other organ and it is understandable that changes in hepatic resistance may have marked circulatory consequences. The portal system includes all veins that carry blood from the abdominal part of the intestinal tract, the gall bladder, pancreas and spleen. The portal vein is formed by the union of the splenic vein and the superior and inferior mesenteric veins anterior to the head of the pancreas and extends for a distance of 5 -8 cm to the porta hepatis. There are two main branches while the further intrahepatic distribution is segmental, accompanying the hepatic arterial- and bile duct system. The inferior mesenteric vein usually enters the splenic vein before joining the superior mesenteric vein but may also enter the portal vein at the junction of the splenic and superior mesenteric vein.1-3

Portal hypertension is defined as a sustained increase in pressure in the portal venous system. Portal pressure is the resultant of the volume of blood flowing through the portal venous system and the resistance to that flow. The normal portal pressure is usually expressed as the portal venous pressure gradient, i.e. the absolute pressure in the portal venous system minus the intraabdominal systemic venous pressure, and ranges from 3 to 6 mm Hg⁴. The absolute portal pressure can be elevated by ascites, increments in systemic venous pressure and other phenomena. Since any of these factors increase all intraabdominal venous pressures equally, it is essential to use an internal zero reference, e.g. the unwedged hepatic venous pressure or the inferior vena cava pressure, to correct for artificial effects on the portal venous pressure.

Clinically significant portal hypertension, with formation of oesophageal varices, requires the gradient to increase above 10 mm Hg. Variceal bleeding is rarely observed when the portal pressure gradient is less than 12 mm Hg⁵⁻⁶. In patients with alcoholic cirrhosis, no relationship was found between the degree of portal hypertension and the risk of gastrointestinal bleeding or the size of oesophageal varices⁷. Patients with varices receiving treatment with propranolol become protected against the risk of

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variceal bleeding when the gradient is lowered to 12 mm Hg or less 8 .

Methods: Study Design: Prospective interventional study

Sample Size: With Convenient sampling method, study of 30 patients with portal hypertension was done at Department of Surgery, New Civil Hospital, Surat during December-15 to January-16 after taking approval from HREC(Human Research Ethics Committee).

Inclusion criteria:

- (a) Age 18 -60 years
- (b) Cases of portal hypertension with hypersplenism
- (c) Cases having \geq 2 episodes of hematemesis
- (d) Patients with normal liver function
- (e) Patients not fit for shunt surgery

Investigations:

- 1. Complete blood count
- 2. Liver Function Test with Enzymes
- 3. Renal Function Test
- 4. Chest X-ray
- 5. Ultrasonography of Abdomen
- 6. Portal Venous Doppler
- 7. Endoscopic findings

Data Collection methods: The pretested, semistructured questionnaire was used for data collection.

Data Management and Analysis: After the completion of data collection, data entry and analysis was done by Excel 3.0.

Results:

Table: 1 Age and sex wise distribution of Study Population.

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Sex	No. (%) n=30
Male	21 (70.0)
Female	9 (30.0)
Age	
<20	6 (20.0)
21-30	7 (23.3)
31-40	10 (33.3)
41-50	5 (16.7)
>50	2 (6.7)

More than two third (70.0%) cases were male and 30.0% were female. The maximum incidence was in

the age group of 31 to 40 years. Minimum incidence was in the age group >50 years. Male patients were affected approximately twice then female patients.

Table: 2 Clinical Profile of Study Population.

	No. (%) n=30
Symptoms	
Haemetemesis	30 (100.0)
Melena	7 (23.3)
Abdominal lump	30 (100.0)
Clinical Findings	
Oesophageal varices	30 (100.0)
Splenomegaly	30 (100.0)
Hypersplenism	30 (100.0)
Complications	
Haemorrhage	NIL (0.0)
Stricture	2 (6.6)
Volvulus	NIL (0.0)
Wound Infection	3 (10.0)

In this study, all 30 patients came with complaint of haemetemesis and abdominal lump in the form of splenomegaly. 7 patients have complaint of melena. All patients were having oesophageal varices and features of hypersplenism. No pateint was having ascitis. Portal vein was >12mm in diameter and there was centripetal blood flow in all patients. Mean operative time duration was 170 min. Mean duration of hospital stay was 7 days. Suture removal was done on 9th post operative day. 3 patients were having infected wound which was healed by secondary healing. Two pateint developed esophageal anastomotic site stricture after one month of OT which treated by regular 3 times balloon dilatation. Patient responded well and having relief of dysphagia later on. One pateint lost to follow up after 1 month. Post operatively 1 mortality was recorded.

Discussion: In patients of portal hypertension most common presenting complaints are haemetemesis and splenomegaly with features of hypersplenism (anemia, leucocytopenia, thrombocytopenia). 30 cases of portal hypertension presenting with features of hypersplenism and complaint of haemetemesis coming at New Civil Hospital SURAT were included in this study.

Pateints were investigated and thorough clinical examination was carried out. Blood investigations in the form of complete blood count, liver function test with liver enzymes, renal function test, serum

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electrolytes were carried out. Chest x-ray was done for preoperative fitness. Ultrasonography of abdomen and portal venous doppler was done to assess changes of portal hypertension, portal cavernoma formation, splenomegaly, condition of liver, ascitis, diameter of portal vein and splenic vein and hepatic blood flow pattern. Upper GI scopy was performed for evaluation of various grades of esophageal varices. Patients were having oesophageal varices and resultant hemetemesis.

Pneumovac and H.influenza vaccines given 1 week prior to elective Operation. In this study, 30 patients with portal hypertension subjected to Elective Devascularization surgery with splenectomy with or without stapled transesophageal resection and anastomosis. Post operatively patients kept on Injection Penidura 1.2 Milion Units every 21 days and Tablet Chloroquine (250 mg) two tablets every weekly for 1 year and Tablet Propranolol 40mg twice a day for 5 years. Patients were follow up at interval of 1month, 3 months and 6 months during 1st year and thereafter upto 5 years. The maximum incidence was in the age group of 31 to 40 years. Minimum incidence was in the age group >50 years. Male patients were affected approximately twice then female patients. In this study, all 30 patients came with complaint of haemetemesis and abdominal lump in the form of splenomegaly. 7 patients have complaint of melena. All patients were having oesophageal varices and features of hypersplenism. No pateint was having ascitis.

Portal vein was >10mm in diameter and there was centripetal blood flow in all patients. Mean operative time duration was 170 min. Mean duration of hospital stay was 7 days. Suture removal was done approximately on 9th post operative day. 3 patients were having infected wound which was healed by secondary healing. Two pateint developed esophageal anastomotic site stricture after one month of OT which treated by regular 3 times balloon dilatation. Patient responded well and having relief of dysphagia later on. One pateint lost to follow up after 1 month. Post operatively 1 mortality was recorded.

Conclusion: In this study most common age group involved was 4th decade male patients. Presenting symptoms were haemetemesis and abdominal lump (splenomegaly). Patients were having esophageal varices and features of hypersplenism (pancytopenia).

After splenectomy with devascularisation surgery, blood counts were increased and patients had relief from symptom of haemetemesis which was assessed 1month, 3 months and 6 months during 1st year and thereafter upto 5years after OT. None of the patients had repeat episode of hemetamesis. There was less incidence of complications in the form of wound infection and anastomotic site stricture which were treated and responded well.

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