INVESTIGATIVE APPROACH AND MEDICAL MANAGEMENT OF PORTAL HYPERTENSION

B.R. Thapa
S. Mehta

Portal hypertension (PH) is defined when portal venous pressure is 10-12 mm of Hg (17-20 cm of H_2O). Portasystemic collaterals start developing with portal venous pressures of 10 mm of Hg (17 cm of H_2O) but variceal bleeding occurs while portal pressure exceeds 12 mm of Hg(1,2). Normally, portal vein (PV) pressure is 5-10 mm of Hg. Portal pressure builds up as a result of obstruction to portal venous flow. Depending on the level of obstruction, portal hypertension is divided into three categories:

(*i*) *Prehepatic:* when obstruction occurs in the extrahepatic portion of portal vein and in its tributaries (EHVO). This may result from congenital malformation or thrombosis.

(*ii*) *Intrahepatic:* This is as a consequence of liver disease responsible for the resistance to portal venous flow.

From the Division of Pediatric Gastroenterology, Post Graduate Institute of Medical Education and Research, Chandigarh 160 012.

Reprint requests: Dr. B.R. Thapa, Assistant Professor, Division of Pediatric Gastroenterology, PGIMER, Chandigarh 160 012.

(*iii*) *Suprahepatic:* When there is block in hepatic vein or intrahepatic part of inferior vena cava (IVC). Portal hypertension is also classified as pre-sinusoidal (intrahepatic and extrahepatic PH), parasinusoidal (intrahepatic) and postsinusoidal (intrahepatic and post-hepatic or suprahepatic). Occasionally conditions like tropical splenomegaly, arteriovenous fistula, part of cirrhosis of liver, myeloproliferative disorders, leukemia, Hodgkin disease, splenic hemangiomas, systemic mastocytosis and osteopetrosis may result in forward flow portal hypertension(2).

Break up of 53 cases of portal hypertension managed in our unit is given in *Table I*. Many authors have covered the etiology of portal hypertension in literature(3,5).

The triad of hematemesis and/or melena, splenomegaly and ascites is classical of portal hypertension. Splenomegaly is the single most important clinical finding which is diagnostic of PH when associated with hematemesis(6). Intrahepatic PH is associated with presence of prominent veins on the anterior abdominal wall, caput medusae formation and in case of Budd Chiari syndrome(7), veins can be on the flanks, sides and on the back with flow upwards. In EHVD there may be prominent veins in left flank. There may be associated features of hepato-cellular failure and chronic liver diseases.

Investigative Approach to Portal Hypertension

Investigations are based on:

1. Demonstration of collaterals.
2. Patency or block of portal vein.

TABLE I—*Analysis of 53 Cases of Portal Hypertension*

Extrahepatic portal vein obstruction (EHVO)	23
Intrahepatic portal hypertension	30
—Cirrhosis	28
Postnecrotic cirrhosis	20
Extrahepatic biliary atresia	2
Cystic fibrosis	1
Autoimune CAH* with cirrhosis	1
Idiopathic CAH* with cirrhosis	1
HBsAg	1
Rubella	1
Wilson's disease	1
—Non-cirrhotic portal fibrosis (NCPG)	1

*CAH: Chronic aggressive hepatitis

3. Pressure and blood flow measurements.
4. Concomitant information on liver function and etiology of liver disease.

1. *Demonstration of Collaterals*

(*a*) *Esophago-gastroduodenal Endoscopy:* Upper gastrointestinal (UG) endoscopy is a routine investigation with us whether PH is associated with history of bleeding or not. Varices can be in esophagus, stomach or duodenum. Esophagus is the commonest site and varices are mostly confined to lower end of esophagus. Endoscopic grading of varices is given in *Table II*. Red, tense and varices Grade III to IV indicate impending rupture. Cherry red spot is also considerable an ominous sign of bleeding.

(*b*) *Barium Meal Studies:* Barium meal swallow (thick or thin form) is given and films are taken in erect and supine posture to visualise good mucosal coating. Varices show filling defects and grading is given in *Table III*. They are seen in lower one third of the esophagus and can extend to involve whole of esophagus. In the barium meal study, stomach may show prominent

TABLE II—*Endoscopic Grading of Varices*

Grade I.	On inspiration only	Can be effaced	Straight	Red
II.	Both on inspiration expiration	Can be effaced wavy ±	Straight	Red
III.	Projecting in the luemen less than 50%	Can not be effaced	Straight, wavy ±	Red/blue
IV.	Projecting in thew luemen more than 50%	Tense	Straight, wavy ± Tortuous + Coiled ±	Blue

TABLE III—*Grading of Varices on Barium Meal Swallow Studies*

Grade I : Minimally prominent folds in lower end of esophagus during inspiration under buscopan effect.

II : Prominent folds during inspiration as well as expiration leading to scalloping effect.

IV : Filling defects compromising more than 50% of luemen with dilatation of the esophagus.

gastric folds or a mass effect sometimes. Duodenal varices may be seen as lobulated filling defects and duodenal sweep. Portovenography can resolve these collaterals. Barium enema and colonoscopy can show colonic varices. Varices may be in other parts of GI tract and these are called ectopic varices. Angiography picks up these ectopic varices nicely.

2. *Patency or Block of Portal Vein*

(a) Ultrasound: In children, portal hypertension due to intrahepatic or extrahepatic obstruction can be diagnosed by ultrasonography. This is a simple noninvasive technique determining the patency of splenic, portal vein and its tributaries and hepatic veins. The diameter of vessel, extent of block, collaterals and nature of obstruction can be acertained. Thickness of the lesser omentum is never more than the size of aorta but with portal hypertension it is moderately or markedly increased in thickness in 84% of patients(4). Hepatic echotexture can give the nature of liver pathology. In our unit, we routinely do ultrasonography especially when EHVO is clinical diagnosis in younger patients.

(*b*) *Doppler Ultrasound:* Ultrasound guided doppler movements of portal blood flow or hepatic vein blood flow can be seen. Obstruction to the flow and its tributaries can be made out.

(*c*) *CT Scanner:* CT scan can better delineate the PV and enhancement picks up the portasystemic collaterals. In cirrhosis, irregular pattern is appreciated.

(*d*) *Angiography:* Requires good radiological facilities and expert radiologist.

(*i*) *Portal venous system delineation:* It is essential to visualise the portal system for detection of patency, level of obstruction and extent of obstruction and calibre of portal and splenic veins and collaterals. In older children with EHVO with splenomegaly, this can be done to delineate the anatomy of portal vein which is essential before any shunt surgery or transplantation in cirrhosis. A large number of collaterals with distortion of intrahepatic pattern called "tree in winter appearance" may be appreciated.

For indirect angiography (selective celiac angiography); the celiac axis is cannulated through femoral artery. The arterial and venous phases can be seen. The portal venous system is not clearly defined as in splenic venography.

(*ii*) *Digital subtraction angiography:* This is a better technique of contrast resolution with immediate subtraction of images taken before and after injection of intravascular contrast media. Vessels are visualized in venous phase and it is valuable in hepatic lesions.

(*iii*) *Splenic venography of splenoportovenography (SPV):* This is a procedure of choice in EHVO. Contrast is injected into the splenic pulp and is taken up by the portal system rapidly to visualise the splenic and portal vein. Collaterals are also detected. Splenic pulp pressure can be simultaneously measured. SPV is contraindicated in deep jaundice, low PTI and thrombocytopenia. In children there is no difficulty to carry out the procedure. However, in younger children this requires general anesthesia. We do SPV only when shunt surgery is contemplated because of uncontrollable bleeds or recurrent bleeds or in children with very rare blood groups or hypersplenism.

(*iv*) *Other techniques:* These include scinti-photosplenoportography, transhepatic

portography, transhepatic venography, and inferior venacavography.

3. *Portal Venous Pressure and Porta Collateral Blood Flow*

The various techniques which can be utilized for this include wedged hepatic pressure, transhepatic or portal vein pressure, trans-splenic pressure, variceal pressure, estimation of hepatic blood flow, electromagnetic flow meter, estimation of portal systemic collaterals, esophageal blood flow, Doppler ultrasound, and magnetic resonance imaging. These are not routinely done.

Medical Management

The nature of lesion, etiology and level of block determine the investigative approach and therapeutic measures. Portal hypertension leads to hemodynamic changes in body and opening of portasystemic collaterals. Various clinical manifestations of portal hypertension which pose problems are variceal bleed, splenomegaly, ascites and encephalopathy. Treatment of the underlying condition can sometimes alleviate the symptoms of portal hypertension(3-5).

Management of PH in children remains an enigma inspite of availability of advanced medical and surgical treatment. A practical approach to management of portal hypertension is given in *Figs. 1a and 1b.*

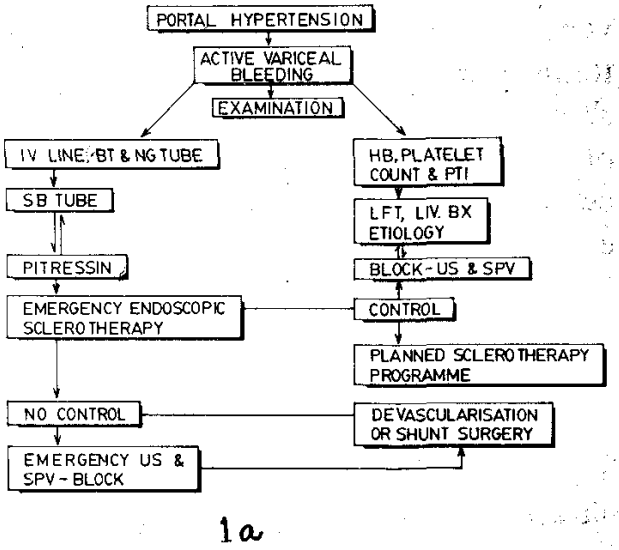

Fig. 1a. Practical approach to a child with portal hypertension

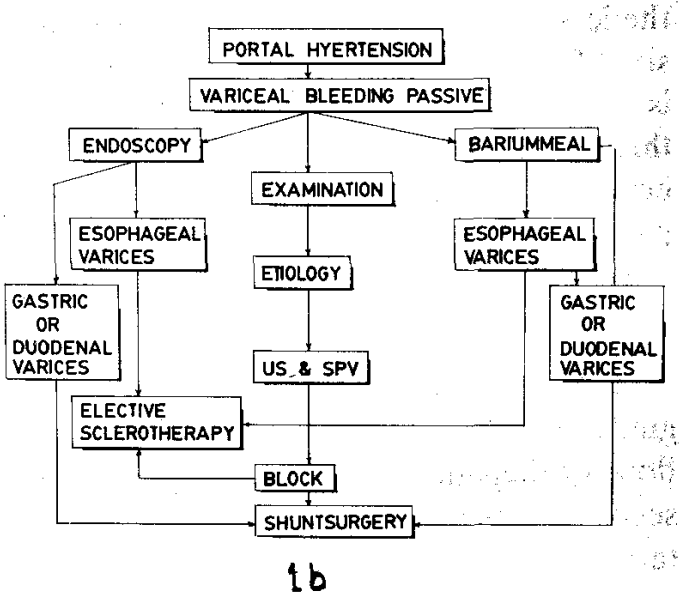

Fig. 1b. Practical approach to a child with partial hypertension.

1. *Variceal Bleed*

This is the most common cause of upper gastrointestinal bleed and is the most dreaded symptom. EHVO is the commonest cause (73.3%) of varical bleeding in children. Varices usually occur in the lower part of esophagus, stomach and duodenum. However, there can be opening of varices in other parts of gastrointestinal tract also (subepithelial and submucosal). Erosions and increased portal pressure cause rupture of varices and hemorrhage. The management of variceal bleed remains a challenge inspite of great knowledge of intensive care. Pharmacological therapy,

hemostatic procedures and sclerotherapy of the varices(10).

A. *Management of Acute Variceal Bleed*

Medical Management of acute variceal bleed is most crucial and determines the immediate outcome. For this, collaboration of Pediatric Gastroenterologist trained in endoscopy and nursing staff trained in intensive care is required. A pediatric surgeon also has to be a part of the team since surgical intervention may be necessary when medical treatment fails.

(*a*) *Supportive measures:* Variceal rupture causes massive hemorrhage and shock supervenes. Sometimes, the bleed may be small and remains undetected and is tolerated well. This can be recurrent and if it remains unattended it could prove fatal. After preliminary examination of the patient, immediately an intravenous line with adequate sized cannula is established. Blood samples are drawn for immediate blood grouping and cross matching and for hemoglobin, platelet count and PTI estimation. Initially normal saline 20 ml/kg over 1 hour is given fast and blood is transfused as soon as it is available. In ideal situation, central vein pressure (CVP) line should be maintained to assess underload or overload. If thrombocytopenia exists then platelet concentrate is required. Injectable vitamin K 5 mg is given daily for 3 days if there is underlying hepatocellular failure.

Nasogastric Ryle's tube is passed into the stomach. This does not enhance the bleed. Gentle suction can be carried ½ hourly and stomach is lavaged with cold saline or water, till returns are clear.

Antacids or cimetidine or ranitidine have no role in prevention of rebleed. However, cirrhosis is the cause of PH and child is having hepatocellular failure with encephalopathy, ranitidine may help in taking care of stress ulceration or existing erosions or peptic ulcer. The patient should be kept in 45° position to avoid gastroesophageal reflux. Avoid retching, coughing, vomiting and straining. Monitor pulse rate, blood pressure and CVP every 10 minutes initially. If the child has stabilized and there is no more bleed, monitor 1 hourly for 24 hours. After stabilization, upper GI endoscopy is done to visualise the nature and site of the lesion. Sometimes there may be bleeding from gastric or duodenal ulcers especially in cirrhotics(8). If there are esophageal varices then emergency scelrotherapy is done(9). Management of gastric and duodenal varices differs. Sclerotherapy of gastric varices has been attempted with variable results.

When there are no facilities to endoscope in first 24 hours, keep the Ryle's tube for 24 hours after control of the bleeding. Gradually introduce oral feeds. Packed cell volume of patient should not exceed 30% (10 g hemoglobin) because over expansion can precipitate rebleed.

Ingestion of aspirin or aspirin containing products is avoided. Antibiotics should be administered on suspicion of infection. Management of associated hypothermia, hypoglycemia disseminated intravascular coagulation and encephalopathy should be carried simultaneously.

(*b*) *Hemostatic Procedures*

(*i*) *Gastro-esophageal balloon tamponade:* Balloon tamponade is used when hemorrhage continues inspite of supportive measures, in case of failure of vasopressin administered to control hemorrhage or complications precluding its use. Pediatric Sengstaken-Blackmore (SB) tube is used. Proper size of SB tube and nursing care

determines its efficacy. A triple lumen Pediatric SB tube which has got gastric and esophageal balloons, is usually utilized–the balloons are checked before use. The third lumen communicates with stomach for aspiration of intragastric contents. In case of four-lumen SB tube, 4th lumen opens in esophagus above the esophageal balloon to prevent pooling of secretions in the airway. In case of trilumen SB tube, extra tube is passed through the mouth. Gastric balloon is first inflated with air from 25-100 cc depending upon the size and gently taken out till some resistance is felt at gastro-esophageal junction preferably under fluoroscopic control. Esophageal balloon is inflated slowly by use of air tight pressure manometer system in range from 30-40 mm of Hg (in adults use 40-60 mm of Hg) to avoid further mucosal injury. Intragastric part of tube drains gastric secretions by gravity. In case of extrahepatic portal vein obstruction (EHVO) this is effective in about 85% cases to control bleeding(2,9,10). There is a possibility of rebleed.

Complications can be life threatening because of malpositioning of tube, impaction, obstruction of airway, slipping of tube and ulceration. Care should be taken to avoid abrupt changes in balloon pressure. Aspiration in airway is prevented by constant suction of esophageal secretions. Inspite of proper position of the SB tubes the persistence of bleeding suggests ineffectiveness to compress esophageal varices and associated gastric or duodenal varices. Linton-Nachlas tube may be helpful when gastric varices are bleeding in adults. Nearly 500-600 cc air is inflated in the gastric balloon. There is no need of traction. Fix the tube on the head of the patient.

Compliance and cooperation of the child is a problem. For this diazepam or fortwin can be used to sedate the child. Deflated tube can be kept for 12-24 hours to take care of rebleed, if any. Retching, vomiting, and coughing should be avoided. Duodenal variceal bleed is difficult to control and requires surgical treatment.

(*ii*) *Pharmacological:* Drugs have also been used to control the bleeding but the results have been variable(11-17). The various drugs which have been employed are listed in *Table IV*. The broad categories are vasoconstrictors such as vasopressin, vasodilators such as nitro-glycerin and miscellaneous drugs such as pentagastrin and domperidine. Each of these drugs have proved of limited help in the control of bleeds. In addition they have several side effects. Pitressin can be used in a titrated drip form intravenously(11).

TABLE IV–*Drugs Used in Portal Hypertension to Control Variceal Bleeding*

A. Vasoconstrictors (Splanchnic vasoconstrictors)
   * Vasopressin
   * Triglycyl-vasopressin (Glypressin)
   * Somatostatin
   * Propranolol and other Beta-blockers.

B. Vasodilators:
   * Nitroglycerin
   * Isosorbide dinitrate
   * Prazosin
   * Sodium nitroprusside
   * Ketanserin

C. Miscellaneous drugs:
   * Esophageal constrictors
   * Pentagastrin
   * Domperidine
   * Metachlorpramide
   * Collagen dissolution

(*iii*) *Emergency injection sclerotherapy:* The concept of emergency injection sclerotherapy is to stop variceal bleeding by thrombosis of bleeding varix secondary to intravariceal injection of sclerosant or thickening of submucosa by injecting sclerosant in submucosa of the varix(18-24).

For this rigid endoscopes and fibroptic endoscopes can be utilized. Since rigid endoscopes require general anesthesia, flexible fibroptic endoscopes are preferred. These are reports of emergency sclerotherapy in children. GIF-P2 fibroptic endoscope and flexible sclerosing needle has been successfully used by us in children. We try to do endoscopy as early as possible when the patient is established in 24 hours of the bleed. Other indications for emergency sclerotherapy include drug or balloon tamponade failure. We have done emergency sclerotherapy in 6 children without any significant complication. Only one child had mild bleeding(24). Chances of bleeding increase when there is underlying liver cell failure or thrombocytopenia. Pulmonary and esophageal complications have been reported in 4-16% cases. After the initial control of bleeding a planned prospective approach is followed for subsequent injections.

(*iv*) *Emergency percutaneous (transhepatic transumbilical or transjugular) obliterations:* Emergency percutaneous transhepatic obliteration of varices requires transhepatic catheterisation of an intrahepatic branch of portal vein. Transumbilical and transjugular vein catheterisation of portal vein can also be carried out(6). Fibrin gelfoam pellets soaked in sodium tetradecyl sulfate are delivered into coronary gastroesophageal drainage system under fluoroscopy. In case of EHVO its role is dubious. This procedure is associated with iatrogenic complications. These have been reported in 30% cases. Major complications are intraperitoneal bleed and subcapsular hepatic hematoma. Deaths have also been reported. There can be technical failure and in children, it is more difficult. This procedure is not of much use when safe ways to control bleeding are available as discussed earlier.

B. *Prevention of Recurrent Bleeding*

Most of the times esophageal variceal bleeds are recurrent. However, sometimes after an initial bleed there may not be subsequent bleeds, this is due to opening of new collaterals. Children with PH require constant follow up and they should be aware of the disease. Blood transfusion facilities should be nearby. We follow patients of PH in the Pediatric Gastroenterology Clinic for clinical monitoring and repeat endoscopy or endoscopic sclerotherapy on outdoor basis.

(*a*) *Endoscopic Sclerotherapy*

This is now a well established mode of therapy of bleeding esophageal varices in children(18-24). Unfortunately these facilities are not available in all medical colleges in the country.

We use the same endoscope as mentioned earlier. For this procedure, in infants and children sedation with diazepam and fortwin (Pentazocine) is enough. One ampoule of buscopan (0.5 ml/kg) is given intravenously to relax GE junction.

After defining esophageal varices in lower part of esophagus, sclerosant is injected. Injections could be intravariceal, paravariceal or combined. Intravariceal injection is appreciated when there is no bleb formation and after taking out the needle there is bleeding which can be stopped by

pushing the endoscope in stomach to compress the varix. The phenomena occurs when initial injections are given and varices vary from Grade III to IV. When varices are getting sclerosed, then injections are paravariceal. Paravariceal injection is paravariceal in the submucosa or mucosa but partially it can be intravariceal also. In one sitting we inject 3-4 varices and inject 5-10 ml of sclerosant. Injections are repeated after every 2-3 weeks till avariceal state is achieved. Injections can be given at weekly interval also. After this, patients are followed every 2 months for repeat endoscopy to see the formation of new varices in the esophagus, stomach and duodenum(24,25). Presently, 30 patients are on our sclerotherapy programme. Endoscopic sclerosis was achieved in 27 (90%) cases(24). Sclerosants used include absolute alcohol, ethanolamine oleate, sodium morrhuate, sodium tetradecyl and ethoxyskerol. Various complications noted are retrosternal discomfort, esophageal ulceration perforation, stricture formation, development of ectopic varices(24) and rarely pulmonary thrombosis and extensive thrombosis extending is splenoportal axis. Strictures are easily manageable by repeated dilatation. Four patients after sclerosis of esophageal varices developed fundal varices and required shunt surgery. Sclerotherapy is very useful as surgery can be postponed till appropriate age and size of splenic vein is available. The children are given a chance to grow and tolerate surgery. Complications of sclerotherapy can be tackled medically. Varices develop in other sites like stomach and duodenum which invariably demand shunting procedure. Sclerotherapy of gastric varices has been attempted with variable results. However, it is associated with complications like bleeding and is technically difficult to perform.

(b) Lowering of Portal Pressure

Propranolol is reported to reduce the chances of rebleed in adults with cirrhosis. It causes vasoconstriction of splanchnic vessel and lowers the portal blood flow, cardiac output and portocollateral flow. Results are quite variable and some authors believe that there is no difference between propranolol and placebo groups. Good controlled studies are required.

(c) Prophylactic Endoscopic Sclerotherapy

Prophylactic endoscopic sclerotherapy is not practised in many centres of the world. There are few reports with contradictory statements. Recently, Paquet and Roussouris have shown from prospective controlled randomized trial that prophylactic endoscopic paravariceal injection sclerotherapy of esophageal Grade III-IV varices with telangectasia and/or poor coagulation reserves of liver can largely prevent esophageal variceal hemorrhage and is able to prolong the life of these chronically ill patients to an appreciable extent(26). In an another controlled study, role of prophylactic EST could not be established(27).

Management of portal hypertension requires an expert team work. Sclerotherapy of esophageal varices seems to be the mainstay of treatment in the pediatric age group. However, the long term effect of sclerotherapy in young children has yet to be defined. Possibly drugs like propranolol may be helpful in controlling rebleed. In children, EHVO is more common, so the initial medical management in early age gives time to grow and to tolerate surgical risks. The chances of blockage of shunt also decrease.

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NOTES AND NEWS

68TH ALL INDIA MEDICAL CONFERENCE 1992 VARANASI

The 68th All India Medical Conference and the Platinum Jubilee of IMA Banaras Branch is being organized by Banaras Branch of Indian Medical Association from *25th to 30th December, 1992.* The central theme of scientific deliberation would be "Health by 2000 AD in developing countries".

The scientific programme will include key-note address, symposia and panel discussion on all aspects of Medical Sciences. For free paper presentation submit 3 copies of abstract of not more than 200 words to Dr. K. Tripathi, Chairman, Scientific Committee, Department of Medicine, Institute of Medical Sciences, BHU, Varanasi 221005, latest by 31st September, 1992.

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Dr. P.N. Singh,
Organizing Secretary,
68th All India Medical Conference,
IMA House,
C.7/31, Chetganj, Varanasi 221001.
Tel: 64561, 62756.