Liver is the organ that cleanses toxin materials from our body through blood. The portal vein delivers blood to the liver. Esophageal varices occur usually in people with liver disease. Blood flow through the liver slows down in people who have liver disease. The pressure in the portal vein goes up when that happens. Because of High blood pressure in the portal vein (portal hypertension) pushes blood into surrounding blood vessels, including the oesophagus. These blood vessels have thin. The extra blood causes them to expand and swell causing varices. If the pressure caused by the extra blood gets too high, it breaks open and bleeds. Bleeding is a medical emergency that requires urgent treatment. Uncontrolled bleeding leads to shock and death. Thrombosis or blood clotting in the portal vein or the splenic vein can cause esophageal varices. The Budd-Chiari syndrome (blockage of certain veins in the liver) and infection with the parasite schistosomiasis are two rare conditions that may cause oesophageal varices.

Cirrhosis of liver is the most common type and a major issue in the western world. More than 90% of these patients develop esophageal varices sometime in their lifetime, and about 30% will bleed. Large sections of scar tissue develop throughout the liver in patients who have cirrhosis and cause slowing blood flow. Cirrhosis can be caused by alcoholic liver disease, fatty liver disease, viral hepatitis or other diseases of the liver.

Portal hypertension is one of the significant complications of both acute and chronic cirrhosis. It can lead to a multitude of pathology of which include the development of porto-systemic collaterals. It generally develops from an increase in prehepatic, intrahepatic, or post herpetic vascular resistance level. Varices are veins that are enlarged or swollen. When an enlarged vein develops on the lining of the esophagus, they are called esophageal varices. Varices may be life-threatening if they break open and bleed. The most significant complication of portal hypertension is life-threatening bleeding from any breakage of gastrointestinal varices, which is associated with substantial morbidity and mortality. There has been considerable progress in the natural history, pathophysiology of portal hypertension but despite the development medical treatments and equipments, early mortality because of variceal bleeding remains high. The incidence of varices is high due to alcohol and obesity.

Esophageal varices: Esophageal varices are dilated sub mucosal distal esophageal veins connecting the portal and systemic circulations. This happens due to portal hypertension (most commonly a result of cirrhosis), resistance to portal blood flow and increased portal venous blood inflow. The most common fatal complication of cirrhosis is variceal rupture; the severity of liver disease correlates with the presence of varices and risk of bleeding.

Bleeding esophageal varices: No single treatment for bleeding esophageal varices is appropriate for all patients and situations. An algorithm for management of the patient with acute bleeding is presented in this article. The options for long-term, definitive therapy and the criteria for selection of each are discussed.

Pathophysiology and management of esophageal varices: Esophageal varices are one of the most common and severe complications of chronic liver diseases. New aspects in epidemiology, pathogenesis and treatment of varices are reviewed. Sclerotherapy is the first-line treatment for acute haemorrhage. Prevention of first or recurrent bleeding is still unsatisfactory. β-Blockers are slightly superior to sclerotherapy with regard to prophylaxis of first bleeding. β-Blockers or sclerotherapy may be used for prophylaxis of recurrent bleeding. However, prophylactic treatment regimens do not have a major impact on survival. Combination treatment, new drugs
or new devices may help to improve the efficacy of prophylactic measures.

Endoscopic therapy for esophageal varices: Among therapeutic endoscopic options for Esophageal varices (EV), Endoscopic variceal ligation (EVL) has proven more effectiveness and safety compared with endoscopic sclerotherapy and is currently considered as the first choice. In acute EV bleeding, vasoactive therapy (either with terlipressin or somatostatin) prior to endoscopy improves outcomes; moreover, antibiotic prophylaxis has to be generally adopted. Variceal glue injection (cyanoacrylates) seems to be effective in the treatment of esophageal as well as in gastric varices. Prevention of rebleeding can be provided both by EVL alone or combined with non-selective β-blockers. Moreover, EVL can be adopted for primary prophylaxis, with no differences in mortality compared with drugs, in subjects with large varices and unfit for a β-blocker regimen. A meta-analysis of endoscopic variceal ligation for primary prophylaxis of esophageal variceal bleeding: Despite publication of several randomized trials of prophylactic variceal ligation, the effect on bleeding-related outcomes is unclear. We performed a meta-analysis of the trials, as identified by electronic database searching and cross-referencing. Both investigators independently applied inclusion and exclusion criteria and abstracted data from each trial. Standard meta-analytic techniques were used to compute relative risks and the number needed to treat (NNT) for first variceal bleed, bleed-related mortality and all-cause mortality. Among 601 patients in 5 homogeneous trials comparing prophylactic ligation with untreated controls, relative risks of first variceal bleed, bleed-related mortality and all-cause mortality were 0.36 (0.26-0.50), 0.20 (0.11-0.39) and 0.55 (0.43-0.71), with respective NNTs of 4.1, 6.7 and 5.3. Among 283 subjects from 4 trials comparing ligation with β-blocker therapy, the relative risk of first variceal bleed was 0.48 (0.24-0.96), with NNT of 13; However, there was no effect on either bleed-related mortality (relative risk [RR], 0.61).

Symptoms of esophageal varices: Most people do not know they have esophageal varices until it starts bleeding. The person vomits large amounts of blood when bleeding is both sudden and severe. When less severe, the person may swallow the blood, which can cause black, tarry stools. If bleeding isn't controlled, the person may develop shock signs, including pale, clammy skin, irregular breathing and loss of consciousness.

Diagnosis and Treatment: Regular screening for esophageal varices is recommended for people who have advanced liver disease. Screening is done by endoscopy. An endoscope is a thin, flexible tube with a light on the tip, and a small camera. The doctor passes the endoscope down the esophagus, and the camera sends images of the esophagus inside to a monitor. The physician looks at the images to detect and grade the enlarged veins by size. Red lines on the veins symbolize bleeding. The physician may also use the endoscope to examine the stomach and the upper part of the small intestine. This is called an esophagogastroduodenoscopy (EGD). CT or MRI imaging scans are often used to treat oesophageal varicose veins, often in conjunction with endoscopy. The CT or MRI images show the esophagus, the liver and the portal and the splenic veins. They give the physician more information about the liver’s health than endoscopy alone.