INTRODUCTION

Gastric vascular ectasia lesions are recognized as an increasingly important source of upper GIT bleeding. The clinical presentation could be iron deficiency anemia due to chronic blood loss or acute bleeding with hematemesis and melena. Patients usually have recurrent episodes of bleeding and require multiple transfusion and in some cases may become transfusion dependant *(Herrera et al., 2008)*.

Upper GI endoscopy is the criterion standard diagnostic test for gastric vascular ectasia (GVE) lesions. The usual appearance of gastric vascular ectasia on endoscopy includes different forms as portal hypertensive gastropathy (PHG) which appears as mosaic like or reticular pattern of gastric mucosa. A similar pattern can be seen through the stomach called gastric antral vascular ectasia (GAVE) or watermelon stomach. Also focal vascular ectasia (FVE) considered when a limited number of red flat spots or reticulated vascular areas present in gastric mucosa without a mosaic pattern *(Fuccio et al., 2009)*.

Various endoscopic techniques are used to treat GVE in the setting of GIT bleeding, heat and multipolar probes are used, but their efficacy depends on the number and extension of lesions. argon plasma coagulation (APC) appears to be easier to use and is safe and more effective than others *(Probest et al., ٢٠٠١)*.

Despite different forms of treatment, few studies have been formed on the outcome and prognosis of patients admitted with upper GIT bleeding secondary to GVE. As it is easy and has low complication rates, APC is the most common method of endoscopic treatment for GVE *(Baudet et al., 2009)*.

AIM OF THE WORK

To study the role of argon plasma coagulation in managing different types of GVE lesions in patients admitted for upper GIT bleeding.

Chapter ١

ANATOMY OF STOMACH AND PORTAL VENOUS SYSTEM

The stomach is an expanded section of the digestive tube between the esophagus and small intestine.The stomach is composed of a cardia, fundus, body, and pyloric part.The greater and lesser curvatures extend between the gastro-esophageal and pyloric openings. The greater is on the left and is convex and longer; the lesser is on the right and is concave and shorter. The lesser curvature usually has an angular notch (incisura angularis). The stomach, which is sometimes J-shaped when empty, is very variable in shape, capacity, and position. The front of the organ faces the greater sac; the back forms the anterior border of the lesser sac. The stomach lies on a variable visceral bed that includes the diaphragm, pancreas, and transverse mesocolon *(O'Rahilly et al., ٢٠٠٤).*

Blood Supply of stomach:

The blood supply of the stomach and duodenum is illustrated in Fig. (1). The left gastric artery

supplies the lesser curvature and connects with the right gastric artery, a branch of the common hepatic artery. In 60% of persons, a posterior gastric artery arises from the middle third of the splenic artery and terminates in branches on the posterior surface of the body and the fundus. The greater curvature is supplied by the right gastroepiploic artery (a branch of the gastroduodenal artery) and the left gastroepiploic artery (a branch of the splenic artery). The mid portion of the greater curvature corresponds to a point at which the gastric branches of this vascular arcade change direction. The fundus of the stomach along the greater curvature is supplied by the vasa brevia, branches of the splenic and left gastroepiploic arteries *(Gerard, 2009)*.

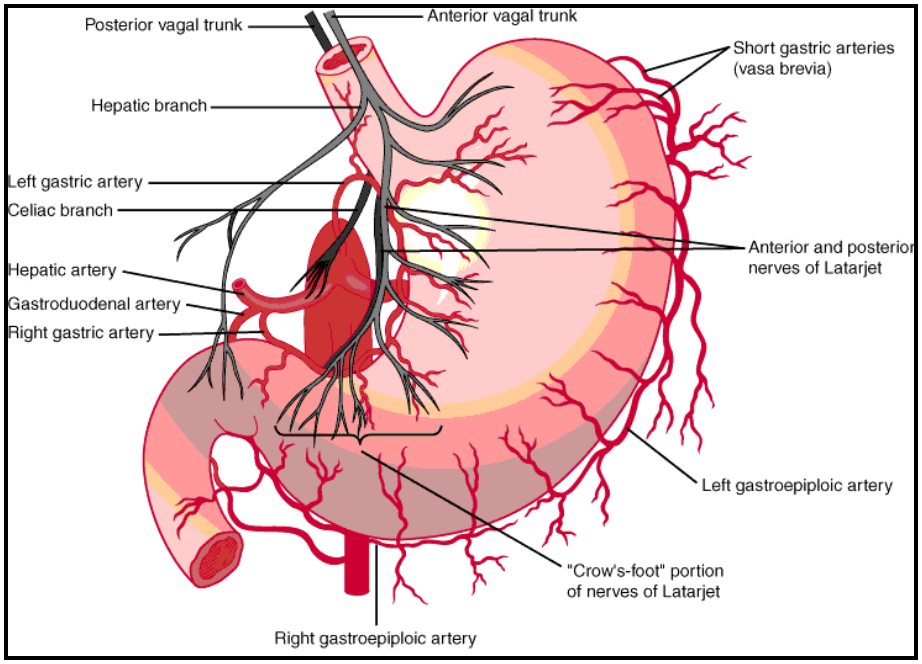

Fig. (1): Anatomy of stomach *(Dohrty, 2009)*.

The blood supply to the duodenum is from the superior and inferior pancreaticoduodenal arteries, which are branches of the gastroduodenal artery and the superior mesenteric artery, respectively. The stomach contains a rich submucosal vascular plexus. Venous blood from the stomach drains into the coronary, gastroepiploic, and splenic veins before entering the portal vein *(Gerard, 2009)*.

The Portal Venous System

The liver has a dual supply from both the hepatic artery and the portal vein *(Morris and Wood, ٢٠٠٠)*. The portal system includes all veins that carry

blood from the abdominal part of the alimentary tract, the spleen, pancreas and gallbladder. The portal vein enters the liver at the porta hepatis in two main branches, one to each lobe; it is without valves in its larger channels *(Douglass et al., 1950)*.

The portal vein is formed by the union of the superior mesenteric vein and the splenic vein just posterior to the head of the pancreas at about the level of the second lumbar vertebra. It extends slightly to the right of the mid-line for a distance of 5.5-8cm to the porta hepatis. The portal vein has a segmental intra-hepatic distribution accompanying the hepatic artery. The superior mesenteric vein is formed by tributaries from the small intestine, colon and head of the pancreas, and irregularly from the stomach via the right gastroepiploic vein *(Sherlock and Dooly, 2002)*.

The splenic veins (5-15 channels) originate at the splenic hilum and join near the tail of the pancreas with the short gastric vessels to form the main splenic vein. This proceeds in a transverse direction in the body and head of the pancreas, lying below and in front of the artery. It receives numerous tributaries from the head of the pancreas, and the left gastro-epiploic vein which enters portal vein near the

spleen. The inferior mesenteric vein bringing blood from the left part of the colon and rectum usually enters its medial third. Occasionally, however, it enters the junction of the superior mesenteric and splenic veins.

Portal blood flow in man is about 1000-١٢٠٠ml/min. The fasting arterio-portal oxygen difference is only 1.9 volumes per cent (range 0.4-3.3 volumes per cent) and the portal vein contributes 40ml/min or 72% of the total oxygen supply to the liver. During digestion, the arterio-portal venous oxygen difference increases due to increased intestinal utilization *(Sherlock and Dooly, 2002)*.

There is no consistent pattern of hepatic distribution of portal inflow. Sometimes splenic blood goes to the left and sometimes to the right. Crossing-over of the blood stream can occur in the portal vein. The flow of portal blood is probably stream-lined rather than turbulent. Portal pressure is about ٧mmHg *(Sherlock and Dooly, 2002)*.

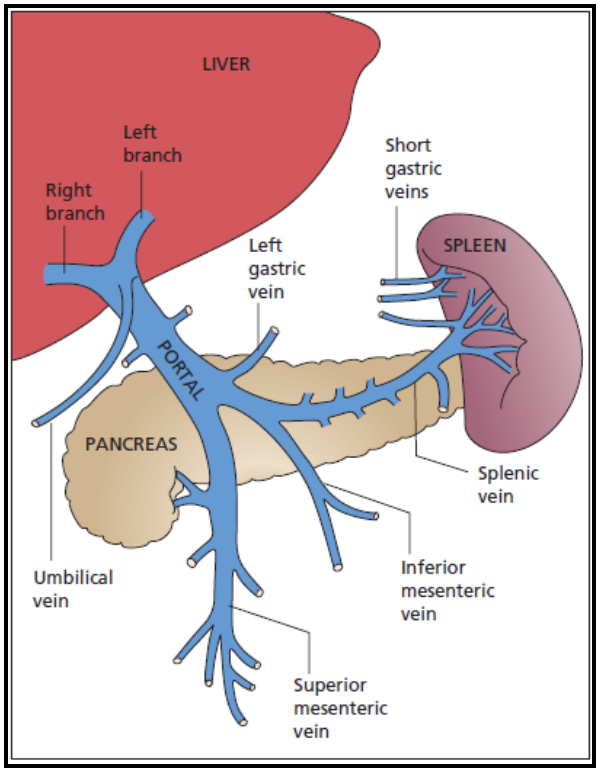

Fig. (2): The anatomy of the portal venous system. The portal vein is posterior to the pancreas.

Collateral circulation

When the portal circulation is obstructed, whether within or outside the liver, a remarkable collateral circulation develops to carry portal blood into the systemic veins.

Intra-hepatic obstruction (cirrhosis)

Normally 100% of the portal venous blood flow can be recovered from the hepatic veins, whereas in cirrhosis only 13% is obtained. The remainder enters collateral channels which form four main groups.

Group I: where protective epithelium adjoins absorptive epithelium

(A) At the cardia of the stomach, where the left gastric vein, posterior gastric and short gastric veins of the portal system anastomose with the intercostal, diaphragmo-oesophageal and azygos minor veins of the caval system. Deviation of blood into these channels leads to varicosities in the submucous layer of the lower end of the oesophagus and fundus of the stomach *(Kimura et al., 1990)*.

(B) At the anus, the superior haemorrhoidal vein of the portal system anastomoses with the middle and inferior haemorrhoidal veins of the caval system. Deviation of blood into these channels may lead to rectal varices.

Group II: In the falciform ligament through the paraumbilical veins, relics of the umbilical circulation of the fetus.

Group III: where the abdominal organs are in contact with retroperitoneal tissues or adherent to the abdominal wall. These collaterals run from the liver to diaphragm and in the spleno-renal ligament and omentum. They

include lumbar veins and veins developing in scars of previous operations or in small or large bowel stomas.

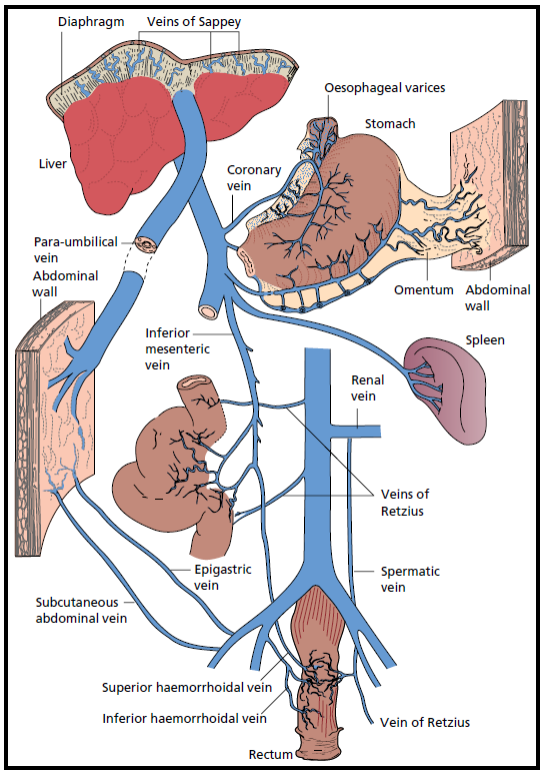

Fig. (3): The sites of the portal-systemic collateral circulation in cirrhosis of the liver ***(Sherlock and Dooly, 2002)***.

Group IV: Portal venous blood is carried to the left renal vein. This may be through blood entering directly from the splenic vein or via diaphragmatic, pancreatic, left adrenal or gastric veins.

Blood from gastro-oesophageal veins and other collaterals ultimately reaches the superior vena cava via the azygos or hemiazygos systems. A small volume

enters the inferior vena cava. An intra-hepatic shunt may run from the right branch of the portal vein to the inferior vena cava. Collaterals to the pulmonary veins have also been described *(Park et al., 1994)*.

Extra-hepatic obstruction

With extra-hepatic portal venous obstruction, additional collaterals form, attempting to bypass the block and return blood towards the liver. These enter the portal vein in the porta hepatis beyond the block. They include the veins at the hilum, venae comitantes of the portal vein and hepatic arteries, veins in the suspensory ligaments of the liver and diaphragmatic and omental veins. Lumbar collaterals may be very large.

Collaterals usually imply portal hypertension, although occasionally if the collateral circulation is very extensive portal pressure may fall. Conversely, portal hypertension of short duration can exist without a demonstrable collateral circulation. A large portal-systemic shunt may lead to hepatic encephalopathy, septicaemias due to intestinal organisms, and other circulatory and metabolic effects *(Sherlock and Dooly, 2002)*.

Chapter ٢

PORTAL HYPERTENSION

Portal hypertension, a major hallmark of cirrhosis, is defined as a portal pressure gradient exceeding 5 mm Hg.In portal hypertension, portosystemic collaterals decompress the portal circulation and give rise to varices. Successful management of portal hypertension and its complications requires knowledge of the underlying pathophysiology, the pertinent anatomy, and the natural history of the collateral circulation, particularly the gastroesophageal varices *(Bosch et al., 2000)*.

Physiology of the portal venous system:

The portal venous system is unique, being between two capillary beds: upstream, the sinusoids of the spleen and the capillaries of the digestive system and downstream, the sinusoidal bed of liver.

The portal blood differs from most of other venous blood in being:

١. Under slightly higher pressure in order to overcome the resistance of the hepatic sinusoids.

٢. Less depleted in oxygen because of the relatively higher blood flow through the splanchnic area due to multiple artriovenous fistula in the organs drained.

٣. Carring nutrients and bacterial waste products from the digesitive tract to the liver.

٤. Containing substnaces responsible for the maintenance of liver cells integrity,function and its capacity of regeneration.

(Jenkins and Billing, 1985)

Pathophysiology of portal hypertension:

Introduction:

Increased resistence to portal blood flow is the primary factor in the pathophysiology of portal hypertension, and is mainly determined by the morphological changes occuring on chronic liver disease. This is aggrevated by a dynamic component, due to the active-reversible-contraction of the different elements of the porto-hepatic bed. A decreased synthesis of nitric oxide (NO) in the entrahepatic circulation is the main determenant of this dynamic component. This is provides a national for the use of vasodilators to reduce intrahepatic resistance and portal pressure. Another factor contributing to

aggrevate the portal hypertension is a significant increase in portal blood flow, caused by arteriolar splanchnic vasodilation and hyperkinetic circulation *(Bosh and Garcia-Pagan, 2000)*.

The pathogenesis of portal hypertension involves the relationship between portal venous blood flow and the resistance offered to this blood flow within the liver (portohepatic resistance) and within portosystemic collateral blood vessels (the portocollateral resistance) that form during the evolution of portal hypertension *(Nathan and Francis, 2002)*.

Portal hypertension results when compensatory mechanisms are inadequate as consequence of pathological increase in either portal venous flow (Forward hypothesis) or resistance (backward hypothesis) and both will be explained as follows:

I) Backward flow theory:

For many years, it was thought that portal hypertension was merely due to an increase resistance to portal flow caused by prehepatic coma, intra-hepatic, or post hepatic "block" resulting in a "congested" portal venous system with a reduced portal flow. The increase of the portal venous