

## **Introduction**

It Is Well Known That Egypt is one of the world's highest of hepatitis C virus (HCV) infection.

“Just about every family in Egypt is touched by hepatitis C,” says Dr. Henk Bekedam, who Representative in the country. The bloodborne virus, which is infectious, kills an estimated 40 000 Egyptians a year and at least 1 in 10 of the population aged 15 to 59 is infected. Estimates of HCV prevalence in Egypt range from 11% to 14% with 8 to 10 million having anti-HCV and 5 to 7 million having active infections (*WHO, 2014*).

According to the WHO data, Liver Disease Deaths in Egypt reached 41,400 or 7.9 % of total deaths. The Death Rate is 67.54 per 100,000 of population ranks Liver Cirrhosis as the Third cause of death in Egypt (*WHO, 2015*).

Along with HCV infection, Clinical studies showed 70% to 90% of patients with chronic hepatitis, cirrhosis, or hepatocellular carcinoma had HCV infections. Co-infections with schistosomiasis caused more severe liver disease than infection with HCV alone (*Haddock and McWilliams, 2104*).

Portal hypertension in the setting of cirrhosis commonly leads to splenomegaly (*Haddock and McWilliams, 2104*).

Hypersplenism refers to a clinical syndrome characterized by splenomegaly, a variable combination of anemia, leucopenia and/or thrombocytopenia. Thrombocytopenia in portal hypertension is due to the dual mechanism of splenic sequestration of platelets and reduced hepatocellular function. TIPS treats portal hypertension but does not improve liver synthetic function and has not been found effective in Hypersplenism (*Gowda et al., 2012*).

Cirrhosis is frequently associated with decreased hematologic indices, including thrombocytopenia and anemia. The prevalence of leukopenia amongst cirrhotic patients is more common than in the general population, and varies from 5% to 61% (*Bashour et al., 2000*).

The pathogenesis of each hematologic deficiency in cirrhotic patients is multi-factorial in nature (*Haddock and McWilliams, 2104*).

Decreased hematologic indices can have significant clinical Results. Thrombocytopenia increases a patient's risk of spontaneous bleeding, and may prevent surgical or endovascular interventions. Leukopenia decreases the

patient's ability to overcome infection, and may serve as a contraindication to the use of chemotherapy in hepatocellular carcinoma. Anemia places a patient at increased risk should bleeding occur, may prevent surgical or endovascular interventions and can leave a patient dependent on transfusions (*Bashour et al., 2000*).

Operative splenectomy can be used to treat splenomegaly in cirrhotic patients. While splenectomy is an effective treatment of splenomegaly in the setting of cirrhosis, it is not without risk. Major complications include portal vein thrombosis and sepsis. Additionally, some cirrhotic patients may be poor surgical candidates, thus necessitating alternative approaches to splenomegaly in some cirrhotic patients.

In 1973, Maddison performed the first splenic artery embolization. an intra-arterial embolization of the splenic artery utilizing autologous clot as the embolic agent. The patient responded well and no complications were reported at 5-months follow-up.

Despite Maddison's early success, numerous complications of total splenic artery embolization were soon discovered. Complications included splenic abscess, splenic rupture, pneumonia, septicemia, and death. In response to these

complications, Spigos et al transitioned to partial splenic embolization (PSE) paired with antibiotic prophylaxis and demonstrated significantly better outcomes (*Haddock and McWilliams, 2104*).

Now partial splenic embolization gained popularity and served as a therapeutic option for cirrhotic patients with Hypersplenism who were poor surgical candidates (*Haddock and McWilliams, 2104*).

PSE has a direct effect on the spleen and may cause improved hepatic function. The improvement may be due to an immunologic mechanism, or because of decreased splenic venous flow, leading to compensatory increase in flow in the hepatic artery and superior mesenteric and vein, which may result in more nutritious flow of blood to the liver (*Strickland, 2006*).

## **Aim of the Work**

The aim of this Work is to investigate the effect of partial splenic Artery embolization (PSE) on platelet Count in cirrhotic patients with Hypersplenism.

## **Anatomy of the Spleen**

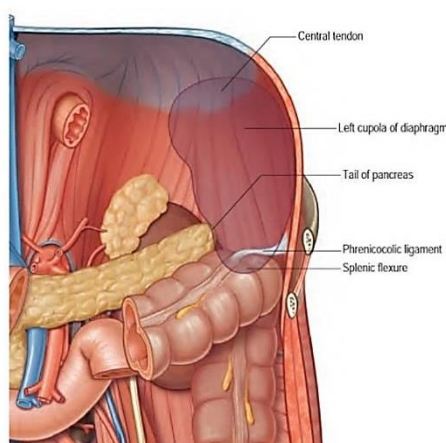
The spleen is considered the largest reticuloendothelial organ in the body. Although it has a characteristic shape, its appearance is variable, as manifested by clefts in the parenchyma of various depths and in various locations (*Ronald et al., 2002*).

The normal human spleen weighs approximately 150 to 250 g and is about the size of a clenched fist. Its juxtaposition in the left upper quadrant to the ninth, tenth, and 11th ribs renders it extremely vulnerable to injury when these ribs are fractured. There are two surfaces: the parietal surface is related to the diaphragm, while the visceral surface is related to the left colon, left kidney, pancreatic tail, and stomach (*Mebius and Kraal, 2005*).

The splenic capsule is relatively thin and is composed of a layer of mesothelial cells under which are several cell layers of fibroelastic tissue. The capsule is susceptible to tears or avulsion by either direct trauma or injudicious traction on adjacent structures. From the splenic capsule arise the trabeculae, which traverse the parenchyma. The capsule and trabeculae contain blood vessels, lymphatics, and nerves (*Morgenster and Skandalakis, 1997*).

## Anatomic Relationships

The spleen lies obliquely in the left hypochondrium. Its cranial pole may reach the epigastric region, and its caudal pole often extends into the lumbar region. It is located in a shallow pocket formed dorsally by the kidney and suprarenal gland, laterally by the costal part of the diaphragm (figure 1), cranially by the dome of the diaphragm, caudally by the left colic flexure and the phrenicocolic ligament, and ventromedially by the stomach. The long axis of the organ runs approximately in line with the tenth rib, with the projection of its margins reaching one rib width above and below this. The spleen lies lateral to a line drawn from the left sternoclavicular articulation to the tip of the eleventh rib (*Ronald et al., 2002*).



**Figure (1):** The posterior relations of the spleen (*Gray's Anatomy 2016*).

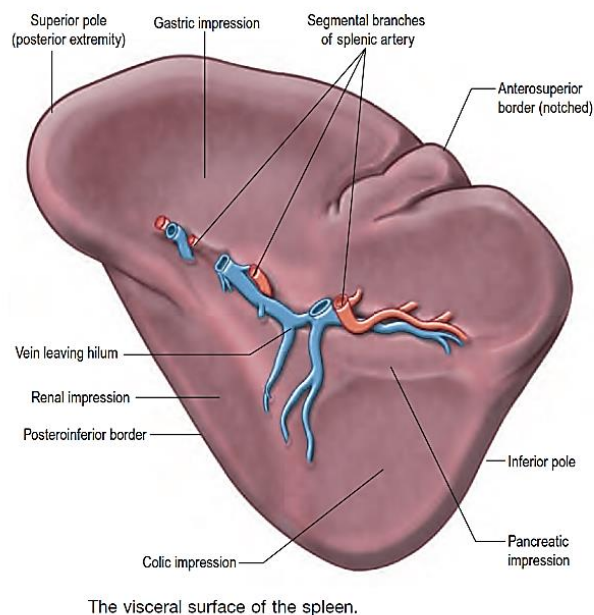
The spleen has superolateral diaphragmatic and inferomedial visceral surfaces, anterosuperior and posteroinferior borders, and superior and inferior poles.

The convex, smooth diaphragmatic surface faces mostly superiorly and laterally, although the posterior part may face posteriorly. The diaphragmatic surface is separated from the left pleural costodiaphragmatic recess, lower lobe of the left lung and the tenth to twelfth left ribs by the underside of the left dome of the diaphragm (*Coquet et al., 2010*).

The visceral surface (figure 2) is irregular, faces inferomedially towards the abdominal cavity and is marked by gastric, renal and colic impressions. It is separated from the stomach by a peritoneal recess, limited by the gastrosplenic ligament. The renal impression is slightly concave and lies on the posteroinferior part of the visceral surface, separated from the gastric impression above by a ridge of splenic tissue and the splenic hilum. It faces inferomedially and slightly backwards, and is related to the upper lateral area of the anterior surface of the left kidney and sometimes to the superior pole of the left suprarenal gland. The colic impression is usually flat; it lies at the inferior pole of the spleen and is related to the splenic flexure



of the colon and the phrenicocolic ligament. The hilum of the spleen is a long fissure pierced by the splenic vessels, nerves and lymphatics, and lies on the visceral surface closer to the posteroinferior border (*Petroianu, 2011*).



**Figure (2):** The visceral surface of the spleen (*Gray's Anatomy 2016*)

The anterosuperior border separates the diaphragmatic surface from the gastric impression and is usually convex. Inferiorly, it may bear one or two notches that have persisted from the lobulated form of the spleen in early fetal life. However, the notch may be absent and is not a completely

reliable guide to identification of the spleen during clinical examination (*Mirjalili et al., 2012*).

The posteroinferior border separates the renal impression from the diaphragmatic surface and is more rounded and blunt than the anterosuperior border. The superior pole corresponds to the posterior extremity and usually faces the vertebral column. The inferior pole is longer and less angulated than the superior pole and connects the anterosuperior and posteroinferior borders anteriorly; it is related to the colic impression and often lies adjacent to the splenic flexure and phrenicocolic ligament (*Petroianu, 2016*).

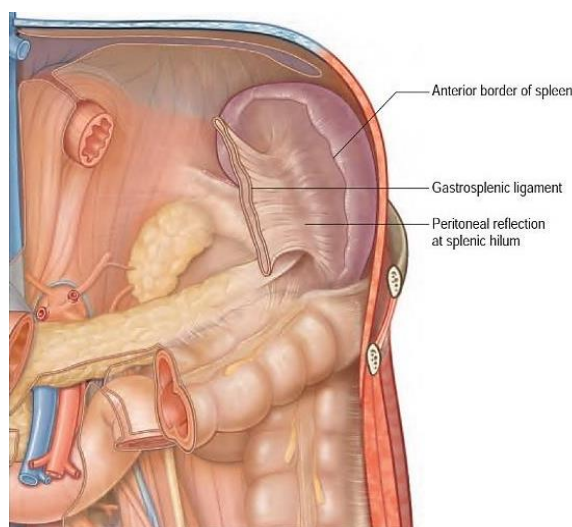
The inferior pole of the spleen is particularly at risk of injury from blunt abdominal trauma or during surgical procedures on the stomach (*Merchea et al., 2012*).

### **Splenic Ligaments**

The spleen develops between the two leaves of the dorsal mesogastrium and so is almost entirely invested in visceral peritoneum that is firmly adherent to its capsule. The dorsal mesogastric attachments persist as peritoneal ligaments. Each of these ligaments is made up of two layers of peritoneum containing fat, blood and lymphatic vessels and nerves (*Üngör et al., 2007*).

### The gastrosplenic Ligament:

The gastrosplenic ligament is synonymous with the gastrosplenic omentum. Its extent is variable, but usually it extends from the upper pole of the spleen to its lower third, sometimes leaving a portion of the lower third unattached to the gastrosplenic omentum. Occasionally, the entire medial surface of the spleen will be attached to the stomach in a broad band of vascular peritoneal reflection (figure 3). It contains the short gastric and superior polar arteries, and the left gastroepiploic artery, all of which arise from the splenic artery, and their corresponding veins (*Petroianu, 2016*).



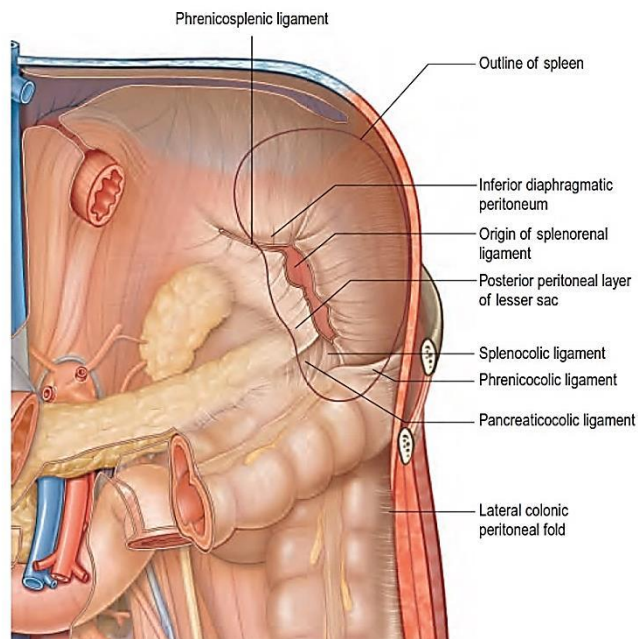
**Figure (3):** The anterior peritoneal connections, seen with the spleen in place, as if the stomach and greater omentum have been removed, with the folds fixed in their normal positions (*Gray's anatomy 2016*).

### **The Splenocolic Ligament:**

The peritoneal fold from the splenic flexure of the colon to the lower pole of the spleen is short and not as vascular as the gastrosplenic ligament (*Skandalakis et al., 1993*).

### **The splenorenal ligament:**

It is actually the posterior peritoneum (figure 4) which has parted above the underlying kidney to encompass The terminal portions of the splenic artery and vein, and, more inferiorly, the tail of the pancreas between its two peritoneal layers. It is the fold of peritoneum immediately posterior and parallel to the spleen. It is generally less vascular than either the gastrosplenic or splenocolic ligament in normal spleens, but may contain sizable vessels in cases of splenomegaly (*Merchea et al., 2012*).



**Figure (4):** Posterior peritoneal connections, seen as if the spleen has been removed, with the folds fixed in their normal positions (*Gray's anatomy 2016*)

### **The phrenicosplenic ligament:**

It runs between the spleen and the peritoneum of the undersurface of the diaphragm (*Petroianu, 2016*).

### **The Spleno-omental Ligament:**

A constant fold of peritoneum attaches to the lower pole of the spleen from the omentum, close to the splenic flexure of the colon (*Morgenster and Skandalakis, 1997*).

## **Vascular Anatomy**

A good knowledge of the vascular anatomy of the splenic artery and its branches is required to avoid potential complications resulting from inadvertent passage of the embolic material to the vessels of the non-target organs as it is one of the most dangerous complication. In partial splenic artery embolization, this complication may occur in pancreatic branches of the splenic artery. Thus the location of the last pancreatic branch in the splenic artery of particular importance, since the tip of the microcatheter should be distal to the origin of this vessel to achieve a safe embolization (*Sindel et al., 2001*).

## **Arterial supply**

The spleen is supplied by the splenic artery (figure 5), one of the most tortuous arteries in the body. The pathophysiology of the tortuosity of this vessel, which may become more pronounced with advancing age, is not understood, although several theories have been proposed (*üngör et al., 2007*).

Almost always, the splenic artery arises from the coeliac trunk, in common with the left gastric and common hepatic arteries. However, it may originate from the common

hepatic artery or the left gastric artery, or rarely directly from the aorta either in isolation or as a splenomesenteric trunk. From its origin, the artery runs a little way inferiorly before turning to the left behind the stomach to run horizontally posterior to the upper border of the body and tail of the pancreas. Multiple loops or even coils of the artery appear above the superior border of the pancreas. The splenic artery courses anterior to the left kidney and left suprarenal gland, and runs in the splenorenal ligament behind or above the tail of the pancreas (*Pandey et al., 2004*).

In its course, it gives off numerous branches to the pancreas (dorsal pancreatic, greater pancreatic artery, and arteries to the tail) and, near its termination, it gives off the short gastric arteries and the left gastroepiploic artery. Additional branches include a posterior gastric artery in 40% of individuals and small retroperitoneal branches (*Gürleyik et al., 2000*).

The splenic artery varies between 8 and 32 cm in length and its caliber usually exceeds that of the common hepatic and left gastric arteries, ranging from 3 to 12 mm.