Introduction

I any patients with advanced liver disease (especially Leirrhotic patients) develop portal hypertension that results in an enlarged spleen and subsequent platelet sequestration. The increase in resistance to portal blood flow (i.e. increased portal pressure) causes redistribution of blood to the spleen, subsequent pooling of platelets, and the increased clearance of platelets from the circulation (Adinolfi et al., 2001).

Hypersplenism is an overactive spleen. The spleen is an organ found in the upper left side of your abdomen. The spleen helps filter old and damaged cells from your bloodstream. If the spleen is overactive, it removes the blood cells too early and too quickly. The spleen plays a key role in helping your body fight infections. Problems with the spleen can make you more likely to develop infections (Connell et al., 2013).

Symptoms of hypersplenism include easy bruising, easy co ntracting of bacterial diseases, fever, weakness, heartpalpitations, and ulcerations of the mouth, legs and feet. Individuals may also b leed unexpectedly and heavily from the nose or other mucous membranes, and from the gastrointestinal or urinary tracts. Most patients will develop an enlarged spleen, anemia, leukopenia, or abnormally low white blood cell counts, or thrombocytopenia, a deficiency of circulating platelets in the blood.

Other symptoms may be present that reflect the underlyin g disease that has caused hypersplenism (Ferri et al., 2016).

Hypersplenism occurs in patients with chronic liver disease, and splenectomy is the definitive treatment. However, the operation may be hazardous in patients with poor liver function. In recent years, partial splenic embolization (PSE) has been widely used in patients with hypersplenism and cirrhosis. This study was conducted to assess the safety and efficacy of PSAE in the management of hypersplenism in cirrhotic patients.

Splenic embolization was first introduced in 1973, when autologous blood clot was used by Maddison to produce splenic artery embolization for hypersplenism treatment. Seven years later, transcatheter partial splenic embolization (PSE) was developed by Spigos et al., which has been proved as a safe and effective method of vascular occlusion. Since then, PSE has ever been gaining its indications and is popularly used in the world, nowadays, and increasingly performed to treat various clinical conditions from salvaging patients with blunt splenic injury to facilitating interferon therapy in patients with chronic hepatitis virus infection (Palsson et al., 2005).

Partial splenic embolization is an effective therapeutic modality for the treatment of hypersplenism secondary to chronic liver disease. It is a simple, rapid procedure that is easily performed under local anesthesia; and it allows preservation of adequate splenic tissue to safeguard against overwhelming infection (Amin et al., 2009).

AIM OF THE WORK

This study aims to discuss the role of partial splenic artery embolization in the treatment of the decreased hematologic indices including thrombocytopenia and leukopenia in chronic liver disease and the efficacy of this method in increasing of hematologic indices in cirrhotic patients with splenomegaly.



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ANATOMY OF THE SPLEEN

The spleen is an organ shaped like a shoe that lies relative to the 9th and 11th ribs and is located in the left hypochondrium and partly in the epigastrium. Thus, the spleen is situated between the fundus of the stomach and the diaphragm. The spleen is very vascular and reddish purple in color; its size and weight vary. A healthy spleen is not palpable (Sadler, 2009).

Gross Anatomy

The spleen's 2 ends are the anterior and posterior end. The anterior end of the spleen is expanded and is more like a border; it is directed forward and downward to reach the mid axillary line. The posterior end is rounded and is directed upward and backward; it rests on the upper pole of the left kidney.

The spleen's 3 borders are the superior, inferior, and intermediate. The superior border of the spleen is notched by the anterior end. The inferior border is rounded. The intermediate border directs toward the right.

The 2 surfaces of the spleen are the diaphragmatic and visceral. The diaphragmatic surface is smooth and convex, and the visceral surface is irregular and concave and has impressions. The gastric impression is for the fundus of the stomach, which is the largest and most concave impression on



the spleen. The renal impression is for the left kidney and lies between the inferior and intermediate borders. The colic impression is for the splenic flexure of the colon; its lower part is related to the phrenicocolic ligament. The pancreatic impression for the tail of the pancreas lies between the hilum and colic impression (*Gray's*, 2010).

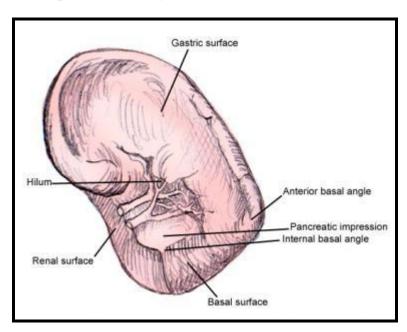


Figure (1): Shows different surfaces and impressions caused by different organs in relation to the spleen's hilum (*Imaging Atlas of Human Anatomy*, 2013).

Hilum

The hilum can be found on the inferomedial part of the gastric impression (see the image above). The hilum transmits the splenic vessels and nerves and provides attachment to the gastrosplenic and splenorenal (lienorenal) ligaments (*Gray's*, 2010).



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Peritoneal relations

The spleen is surrounded by peritoneum and is suspended by multiple ligaments, as follows:

- The gastrosplenic ligament extends from the hilum of the spleen to the greater curvature of the stomach; it contains short gastric vessels and associated lymphatics and sympathetic nerves.
- The splenorenal ligament extends from the hilum of the spleen to the anterior surface of the left kidney; it contains the tail of the pancreas and splenic vessels.
- The phrenicocolic ligament is a horizontal fold of peritoneum that extends from the splenic flexure of the colon to the diaphragm along the midaxillary line; it forms the upper end of the left paracolic gutter (*Gray's*, 2010).

Visceral relations

The visceral surface of the spleen contacts the following organs:

- Anterior surface of the left kidney
- Splenic flexure of the colon
- The fundus of the stomach
- Tail of the pancreas



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The diaphragmatic surface is related to the diaphragm; the diaphragm separates the spleen from the pleura and the lung (*Gray's*, 2010).

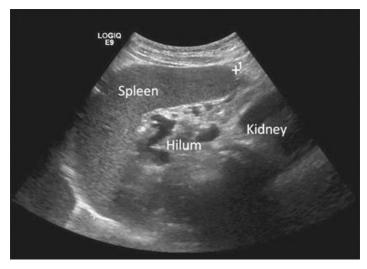


Figure (2): Shows the normal spleen with its vessels at the hilum (*Imaging Atlas of Human Anatomy*, 2013).

Vascular supply

Splenic artery

The **splenic artery** is one of three branches coeliac trunk and supplies the spleen as well as large parts of the stomach and pancreas (*McMinn*, 2003).

Gross anatomy

Origin and course

The splenic artery is one of the terminal branches of the coeliac trunk, passing from the coeliac axis toward the splenic

hilum, travelling superiorly to the pancreas with the splenic vein inferoposteriorly before entering the splenorenal ligament.

Near the splenic hilum the splenic artery divides into superior and inferior terminal branches, with each terminal branch further dividing into four-to-six intrasplenic segmental branches (*Madoff et al.*, 2005).

Branches and supply

- Pancreatic branches including the dorsal pancreatic artery and greater pancreatic artery (arteriapancreatica magna)
 - Supply neck, body and tail of the pancreas
- Short gastric arteries
 - Arising before the splenic artery enters the splenic hilum.
 - Run in the gastrosplenic ligament to supply the fundus and upper part of greater curvature of the stomach, and anastomose with the left gastric artery over the fundus.
 - o Supplies cardia and fundal regions of the stomach
- Left gastroepiploic artery
 - o Runs in the greater omentum along the greater curvature of the stomach to anastomose with the right gastroepiploic artery (*Madoff et al.*, 2005).



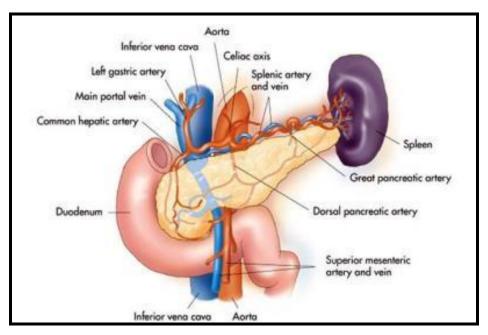


Figure (3): Shows splenic artery anatomy (Madoff et al., 2005).

Variant anatomy

 Separate origin from the abdominal aorta rather than the coelic trunk (~8%) (Pandey et al., 2004).

Related pathology

• Splenic artery aneurysm (Pandey et al., 2004).

Nerve supply

Sympathetic fibers are derived from the celiac plexus (Snell, 2007).

Venous drainage

The splenic vein provides the principal venous drainage of the spleen. It runs behind the pancreas (after forming at the



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hilum) before joining the superior mesenteric vein behind the neck of the pancreas to form the portal vein. The short gastric, left gastro-omental, pancreatic, and inferior mesenteric veins are its tributaries (*Snell*, 2007).

Lymphatic drainage

Proper splenic tissue has no lymphatics; however, some arise from the capsule and trabeculae and drain to the pancreaticosplenic lymph nodes (*Snell*, 2007).

Microscopic Anatomy

The spleen is made up of the following 4 components:

- Supporting tissue
- White pulp
- Red pulp
- Vascular system

Supporting tissue is fibroelastic and forms the capsule, coarse trabeculae, and a fine reticulum.

The white pulp consists of lymphatic nodules, which are arranged around an eccentric arteriole called the Malpighian corpuscle.

The red pulp is formed by a collection of cells in the interstices of the reticulum, in between the sinusoids. The cell



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population includes all types of lymphocytes, blood cells, and fixed and free macrophages. The lymphocytes are freely transformed into plasma cells, which can produce large amounts of antibodies and immunoglobulins (*Snell*, 2007).

Natural and Pathophysiologic Variants

- Accessory spleens or splenunculi are natural anatomic variants formed from nodules that fail to fuse during development. These are found in various locations such as the gastrosplenic ligament, splenorenal ligament, gastrophrenic ligament, and gastrocolic ligament. They have also been reported to have been found in the broad ligament of the uterus and in the spermatic cord.
- Pathophysiologic anatomic variants include splenomegaly, asplenia, and autosplenectomy. Splenomegaly is the enlargement of the spleen. It occurs due to various conditions, such as infections (eg, malaria, kalaazar), malignancies (eg, lymphomas, leukemias), and other conditions (eg, portal hypertension). The spleen then projects toward the right iliac fossa in the direction of axis of the 10th rib.
- Asplenia is a rare condition in which a congenital absence of the spleen occurs.
- Autosplenectomy is a condition in which splenic infarction occurs due to sickle cell anemia (*Gray's*, 2010).



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Functions of the Spleen

Immune responses

After antigenic stimulation, increased formation of plasma cells for humoral responses and increased lymphopoiesis for cellular responses occurs (*Guyton and Hall*, 2005).

Phagocytosis

One of the spleen's most important functions is phagocytosis. The spleen is a component of the reticuloendothelial system. The splenic phagocytes include reticular cells, free macrophages of the red pulp, and modified reticular cells of the ellipsoids. Phagocytes in the spleen remove debris, old and effete red blood cells (RBCs), other blood cells, and microorganisms, thereby filtering the blood. Phagocytosis of circulating antigens initiates the humoral and cellular immune responses (*Guyton and Hall, 2005*).

<u>Hematopoiesis</u>

The spleen is an important hemat 2005 opoietic organ during fetal life; lymphopoiesis continues throughout life. The manufactured lymphocytes take part in immune responses of the body. In the adult spleen, hematopoiesis can restart in certain diseases such as chronic myeloid leukemia and myelosclerosis (*Guyton and Hall, 2005*).



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Storage of red blood cells

The RBCs are stored in the spleen. Approximately 8% of the circulating RBCs are present within the spleen; however, this function is seen better in animals than humans (*Guyton and Hall*, 2005).

PATHOLOGICAL CONSIDERATION REGARDING SPLENOMEGALY AND HYPERSPLENISM

Splenomegaly:

plenomegaly is a condition that occurs when your spleen becomes enlarged. It is also commonly referred to as enlarged spleen or spleen enlargement. The spleen is a part of your lymphatic system. The spleen helps the immune system by storing white blood cells and helping in the creation of antibodies (*Poulin et al.*, 1998).

Etiology:

Many of the mechanisms leading to splenomegaly are exaggerated forms of normal splenic function. Although a wide variety of diseases are associated with enlargement of the spleen, the following six etiologies of splenomegaly are considered primary:

- Immune response work hypertrophy Such as in subacute bacterial endocarditis or infectious mononucleosis.
- RBC destruction work hypertrophy Such as in hereditary spherocytosis or thalassemia major.
- Congestive Such as in splenic vein thrombosis, portal hypertension, or Banti disease.

- Myeloproliferative Such as in chronic myeloid metaplasia.
- Infiltrative Such as in sarcoidosis and some neoplasms
- Neoplastic Such as in chronic lymphocytic leukemia and the lymphomas

Miscellaneous causes of splenomegaly include the following:

- Trauma
- Cysts
- Hemangiomas
- Metastasis
- Giant abscess

Inflammatory splenomegaly

Acute enlargement of the spleen due to various infections or inflammatory processes results from an increase in the defense activities of the organ. The demand for increased antigen clearance from the blood may lead to increased numbers of reticuloendothelial cells in the spleen and stimulate accelerated antibody production, with resultant lymphoid hyperplasia. Examples include splenomegaly from lupus and Felty syndrome, and from viral infections such as Epstein-Barr virus—induced mononucleosis.