



Anatomy and Role of Spleen

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The first case of laparoscopic splenectomy was described by Delaitre et al in 1991. This was soon followed by the first case report of laparoscopic splenectomy in USA. These reports were followed by numerous reports from all parts of the world indicating the growing popularity of laparoscopic splenectomy. As experience increased and technological advances took place, the indications and safety profile of the surgery laparoscopic splenectomy also rose. In the initial years, all laparoscopic surgeons adopted the traditional anterior approach with the patient in the semi-lithotomy position. The proponents of this approach cite several advantages. The lateral approach was first mooted by Delaitre again in 1995, the so called “hanged spleen” approach. It was enthusiastically adopted by practitioners of this art, notably Park et al. Finally, the “leaning spleen” technique was adopted by Richardson et al which combined the advantages of both the previous approaches.

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1. Anatomy

The spleen is the second largest organ of the reticuloendothelial system. It has two major ligaments the gastrosplenic ligament carrying the short gastric vessels and the splenorenal (lienorenal) ligament, which contains the splenic artery. It has several minor ligaments the splenophrenic, the splenocolic, the pancreaticosplenic, the presplenic fold, the phrenocolic, and the pancreaticocolic ligaments. It is usually about $3 \times 8 \times 14$ cm, and weighs about 200 gms. Its lateral surface is separated from the left ninth, tenth and eleventh ribs by the diaphragm. Its medial surface relates to the stomach, tail of pancreas and left kidney. It is entirely encapsulated by the peritoneum except at its hilum.

The splenic artery arises as a branch of the celiac trunk in the majority of the cases or as a branch of the gastrosplenic or hepatoesplenic trunk or directly from the aorta. It then runs along the superior border of the pancreas in the posterior leaf of the peritoneum, crosses the upper pole of the kidney, and enters the hilum. The splenic hilum most commonly divides into two major branches but occasionally into three branches. These further divide into 3 to 38 branches. Two or three vasa brevia or the short gastric arteries arise from the splenic artery or the left gastroepiploic artery.

The tail of the pancreas lies in direct contact with the spleen in 30 percent of cadavers. The splenic vein is formed by joining of the intralobar veins near the splenic hilum and is usually joined by the left gastroepiploic vein but not the short gastric veins, which enter the upper part of the spleen in the majority of patients. It has a more direct course than the artery, passing through the splenorenal ligament to the right and usually inferior to the artery and posterior to the pancreas, ending by joining the superior mesenteric vein to form the portal vein.

2. Function

The spleen has important hematopoietic functions during early fetal development, with both red and white blood cell production. By the fifth month of gestation, the bone marrow assumes the predominant role in hematopoiesis, and normally there is no significant hematopoietic function left in the spleen. Under certain pathologic conditions, however, such as myelodysplasia, the spleen can reacquire its hematopoietic function. Removal of the spleen does not usually result in anemia or leukopenia in an otherwise healthy person. Although the hematopoietic function is usually lost during fetal development, the spleen continues to function as a sophisticated filter because of the unique circulatory system and lymphoid organization, and it has blood cell monitoring and management functions as well as important immune functions throughout life.

The functions of the spleen are closely linked to splenic structure and its unique circulatory system. The arteries flow through the white pulp (lymphoid tissues), after which part of the blood flow goes directly through endothelial cell-lined capillaries into the venous system ("closed" theory). Most of the blood flow, however, enters the macrophage-lined reticular meshwork, and the blood flows slowly back to the venous circulation through the venous sinuses ("open" theory). The formed blood elements must pass through slits in the lining of the venous sinuses; if they cannot pass, they are trapped in the spleen and ingested by splenic phagocytes.

The most important function of the spleen is probably its mechanical filtration, which removes senescent erythrocytes and likely contributes to control of infection. The spleen is important in clearing circulating pathogens that reside within erythrocytes, for example, malarial parasites, or bacteria such as Bartonella species. Mechanical filtration by the spleen may also be important for removal of unopsonized, noningested bacteria from the circulation. It may be particularly important for clearing microorganisms for which the host has no specific antibody.

Splenic filtering function is important for maintaining normal erythrocyte morphology and function. Normal red blood cells are biconcave and deform relatively easily to facilitate both passage through the microvasculature and optimal oxygen and carbon dioxide exchange. The spleen is an important site for the processing of immature erythrocytes and for repair or destruction of deformed or aged erythrocytes. As immature red blood cells pass through the spleen, they may undergo several types of repair, including removal of nuclei and excessive cell membrane from immature cells to convert them from a spherical nucleated to a biconcave anucleated mature morphology. Erythrocytes may also undergo repair by having surface abnormalities such as pits or spurs removed. In the asplenic condition, there are

several characteristic alterations in the morphologic appearance of the peripheral red blood cells, with the presence of target cells (immature cells), Howell-Jolly bodies (nuclear remnant), Heinz bodies (denatured hemoglobin), Pappenheimer bodies (iron granules), stippling, and spur cells. Aged red blood cells (120 days) that have lost enzymatic activity and membrane plasticity are trapped and destroyed in the spleen.

The filtering function of the spleen is also an important factor in anemic conditions associated with abnormal red blood cell morphology. Abnormal erythrocytes that result from hereditary spherocytosis, sickle cell anemia, thalassemia, or pyruvate kinase deficiency are trapped by the splenic filtering mechanism, resulting in worsening anemia, symptomatic splenomegaly, and occasionally splenic infarction. In autoimmune hemolytic anemia, immunoglobulin G (IgG) bound to the cell membrane targets the red blood cells for splenic destruction by splenic macrophages. A similar IgG-dependent mechanism is involved in splenic platelet destruction in immune thrombocytopenic purpura (ITP).

Another major function of the spleen is the maintenance of normal immune function and host defenses against certain types of infectious agents. It is well established that people lacking a spleen are at a significantly higher risk for overwhelming postsplenectomy infection (OPSI) with fulminant bacteremia, pneumonia, or meningitis, as compared with those with normal splenic function. Major pathogens in OPSI are organisms such as *Streptococcus pneumoniae*, in which polysaccharide capsules requiring both antibody and complement are important in host defense against these organisms. Asplenic subjects have defective activation of complement by the alternative pathway, leaving them more susceptible to infection.

Asplenic patients have a normal response to reimmunization to an antigen first encountered before splenectomy but do not have an optimal response to new antigen exposure, especially if the antigen is administered intravenously (IV). For organisms such as the encapsulated bacteria, much higher quantities of antibody are necessary for effective clearance. The spleen, with its specialized circulatory system and large supply of macrophages that are capable of ingestion of organisms not optimally opsonized with antibody, greatly enhances their clearance. Asplenic subjects have been found to have subnormal IgM levels, and their peripheral blood mononuclear cells exhibit a suppressed immunoglobulin response.

References

1. Delaitre B, Maginien B. Splenectomy by the laparoscopic approach. Report of a case. *Presse Med* 1991; 20:2263.
2. Delaitre B, Maginien B. Laparoscopic splenectomy technical aspects. *Surg Endosc* 1992;6:305-308.
3. Carroll BJ, Phillips EH, Semel CJ, et al. Laparoscopic Splenectomy. *Surg Endosc* 1992;6:183-185.
4. Delaitre B. Laparoscopic splenectomy. The "hanged spleen" technique. *Surg Endosc* 1995;9:528-529.
5. Park A, Gagner M, Pomp A. The lateral approach to laparoscopic splenectomy. *Am J Surg* 1997;173:126-130.
6. Richardson WS, Smith D, Branum GD et al. Leaning spleen: a new approach to laparoscopic splenectomy. *JACS* 185:429-432.
7. Smith DL, Meyer AA. Anatomy, Immunology and Physiology of the spleen. In: Zuidema GD, Yeo CJ (eds). *Shackelford's Surgery of the Alimentary Tract*. W. B. Saunders Company, 5th edition, 2002, 542-549.
8. Michel NA. The variational Anatomy of the Spleen and Splenic Artery. *Am J Anat* 1942;70:21.

9. Schwartz SI, The spleen anatomy and splenectomy. In Nyhus LM, Baker RJ, Fischer JE (Ed.) Mastery of surgery. Third edition. Chapter 114. Little, Brown and Company, Boston 1997;1267-1275.
10. Rege RV. Laparoscopic splenectomy. In: Scott-Conner, CEH (Ed). The Sages Manual: fundamentals of laparoscopy and GI endoscopy. Springer, 2003;326-335.
11. Coon WW. Splenectomy for conditions other than trauma. In: Zuidema GD, Yeo CJ (eds). Shackelford's Surgery of the Alimentary Tract. W. B. Saunders Company, 5th edition, 2002, 550-561.
12. Park AE. Lateral approach to laparoscopic splenectomy. In: Zucker KA (Ed.): Surgical Laparoscopy, 2nd edition, Philadelphia, Lippincott Williams & Wilkins: 2001;625-634.
13. Schwartz P, Sterioff S, Mucha P, et al. Postsplenectomy sepsis and mortality in adult. JAMA 1982;248:2279.
14. Cullingford G, Watkins D, Watts A, et al. Severe late postsplenectomy infections. Br J Surg 1991;78:716.