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Chapter 7

Splenic Artery Aneurysms

Ahmad Alsheikhly

Additional information is available at the end of the chapter

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1. Introduction

The spleen is a wedge-shaped organ that lies in relation to the 9th and 11th ribs, located in the left upper quadrant of the abdomen (left hypochondrium), and partly in the epigastrium; thus, it is situated between the fundus of the stomach and the diaphragm (see the following image). The spleen is highly vascular and reddish purple; its size and weight are variable. Normally spleen is not palpable.

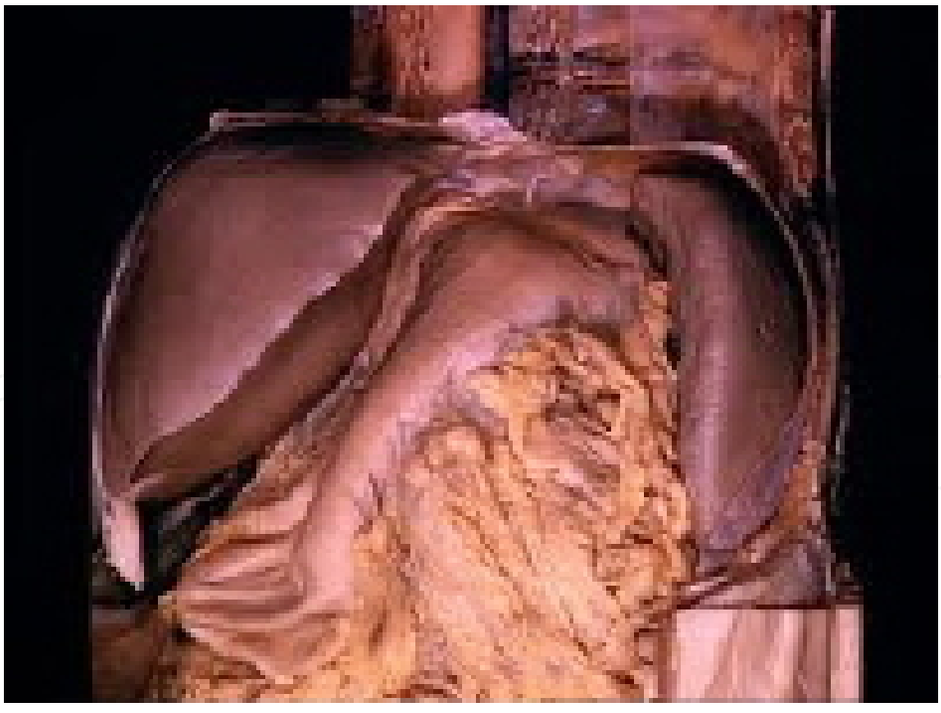

Figure 1.

The spleen develops in the cephalic part of dorsal mesogastrium (from its left layer; during the sixth week of intrauterine life) into a number of nodules that soon fuse to form a

lobulated spleen. Notching of the superior border of the adult spleen is evidence of its multiple origins.[1]

The spleen has 2 ends, 3 borders, and 4 surfaces, as follows:

The 2 ends

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

The 3 borders

The superior border of the spleen is notched near the anterior end, the inferior border is rounded, and the intermediate border is directed toward the right.

The 2 surfaces

There are 2 surfaces: diaphragmatic and visceral. The diaphragmatic surface is smooth and convex. The visceral surface is irregular and concave and has impressions. The gastric impression is for the fundus of the stomach; this 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 (see the image below).

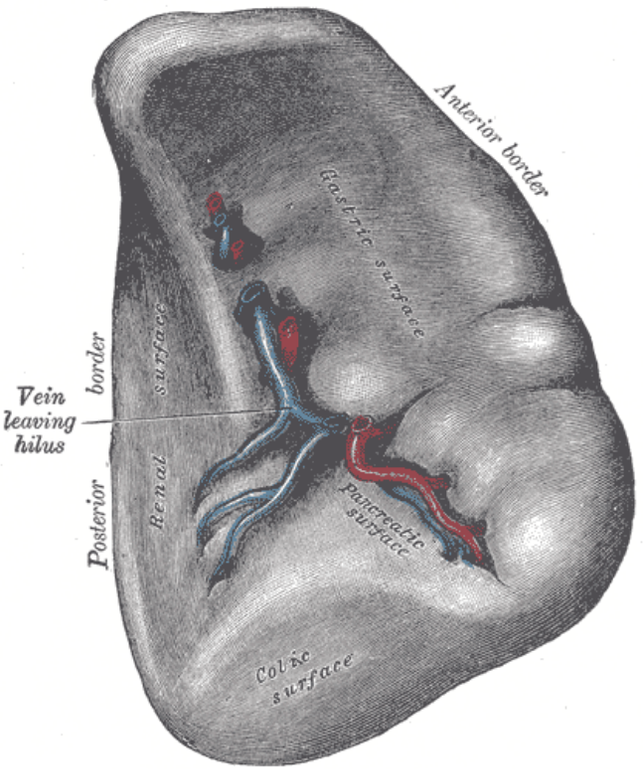

Figure 2. Spleen showing the different surfaces and impressions caused by different organs with relation to the hilum of the spleen.

1.1. Hilum

The hilum lies on the inferomedial part of the gastric impression. It transmits the splenic vessels and nerves and provides attachment to the gastrosplenic and splenorenal (lienorenal) ligaments.

1.2. Peritoneal relations

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

The gastrosplenic ligament: This 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: This 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: This ligament is a horizontal fold of peritoneum extending from the splenic flexure of the colon to the diaphragm in the midaxillary line; it forms the upper end of the left paracolic gutter.

1.3. Visceral relations

The visceral surface of the spleen is related to the following organs:

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

The diaphragmatic surface is related to the diaphragm, which separates the spleen from the pleura and the lung.

1.4. Blood supply

The blood supply of the spleen is by the splenic artery (in the past called the lienal artery), which is the largest branch of the celiac trunk. The artery passes through the splenorenal ligament to reach the hilum of the spleen. At the hilum, it divides into multiple branches. Within the spleen, it divides into straight vessels called penicillin, ellipsoids, and arterial capillaries.

The splenic circulation is adapted for the mechanism of separation and storage of the red blood cells. On the basis of the blood supply, the spleen has superior and inferior vascular segments. The 2 segments are separated by an avascular plane.

Apart from its terminal branches, the splenic artery gives off branches to the pancreas, 5-7 short gastric branches, and the left gastro-omental (gastroepiploic) artery (see the image below).

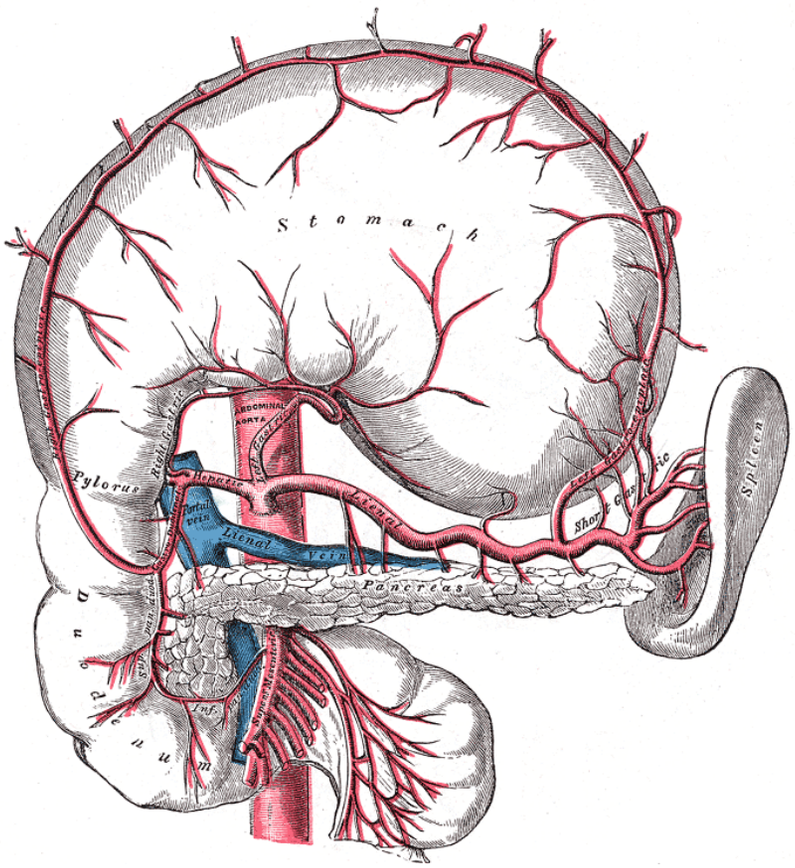

Figure 3.

1.5. Venous drainage

The principal venous drainage of the spleen is through the splenic vein. It is formed at the hilum and runs behind the pancreas then joins the superior mesenteric vein behind the neck of the pancreas to form the portal vein. Its tributaries are the short gastric, left gastro-omental, pancreatic, and inferior mesenteric veins.

1.6. Lymphatic drainage

Splenic tissue proper has no lymphatics. However, a few arise from the capsule and trabeculae and drain to the pancreaticosplenic lymph nodes.

1.7. Nerve supply

Sympathetic fibers are derived from the celiac plexus.[2, 3, 4]

1.8. Surface marking

The spleen is marked on the left side of the back with the long axis of the 10th rib. The upper border is marked along the upper border of the 9th rib; the lower border, along the 11th rib. The medial end lies 5 cm from the midline, and the lateral extension is to the midaxillary line.[5]

Microscopically, the spleen is made up of 4 components: (1) supporting tissue, (2) white pulp, (3) red pulp, and (4) vascular system.

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

The white pulp consists of lymphatic nodules 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 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. The vascular system traverses the spleen and permeates it.[3]

2. Physiologyof the spleen

The spleen is a major hematopoietic organ containing approximately 25 percent of the total lymphoid mass of the body; and it is capable of supporting elements of the erythroid, myeloid, megakaryocytic, lymphoid, monocytic, and macrophagic (reticuloendothelial) systems. As such, it is important in the following situations:

2.1. Phagocytosis

Phagocytosis is one of the most important functions of the spleen. The spleen forms 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. The phagocytes present in the organ remove debris, old and effete red blood cells (RBCs), other blood cells, and microorganisms; thus, the splenic phagocytes filter the blood. Phagocytosis of circulating antigens initiates the humoral and cellular immune responses.

This function is most apparent when the spleen has been removed, since splenectomized patients are susceptible to bacterial sepsis, especially with encapsulated organisms.

2.2. Hematopoiesis

The spleen is an important hematopoietic 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.

2.3. Active immune responses

Following antigenic stimulation, increased lymphopoiesis for cellular responses and increased formation of plasma cells for humoral responses occurs.

2.4. Storage of erythrocytes

The RBCs are stored in the spleen. Approximately 8% of the circulating RBCs are present within the spleen. However, this function is seen well in animals than humans.[6]

3. Splenic Artery Aneurysms (SAAs)

3.1. General considerations

An arterial aneurysm is one of the most common vascular disorders causing morbidity and mortality in humans. It occurs in most arteries of the body and is especially common in the elderly. They have a variable sizes, shapes, and locations.

An aneurysm is defined as a permanent localized dilatation of an artery having at least a 50% increase in diameter compared to the expected normal arterial diameter, so clinicians should know the normal arterial diameters throughout the body to decide the presence or absence of an aneurysm. [7]

Splenic artery aneurysms are a type of splanchnic arteries aneurysm, although the later are rare but clinically very important vascular conditions. These interesting lesions have been recognized since more than 200 years. [8, 9]

Splanchnic artery aneurysms represent intra-abdominal aneurysms that are not part of the aorto-iliac system and include aneurysms of the celiac, superior and inferior mesenteric arteries with their branches.

Of all intra-abdominal aneurysms, only approximately 5% affect the splanchnic arteries. (10) In general population, their prevalence has been estimated to be varying from 0.1% to 2 %. [11]

The frequency of the anatomic distribution of the splanchnic arteries aneurysms is estimated to be the following:

1. Splenic artery aneurysms (SAAs), 60% (see the image below).
2. Hepatic artery, 20%
3. Superior mesenteric artery, 6%
4. Celiac artery, 4%
5. Gastric and gasrtoepiploic arteries, 4%
6. Jejunal, ileal, and colic arteries, 3%
7. Pancreaticoduodenal and pancreatic arteries, 2%
8. Gastroduodenal artery, less than 1.5%
9. Inferior mesenteric artery, less than 1%. (11)

3.2. Prevalence and epidemiology

SAAs are the most common of the splanchnic artery aneurysms and account for as many as 60% of all reported splanchnic aneurysms. They are recognized for their significant potential

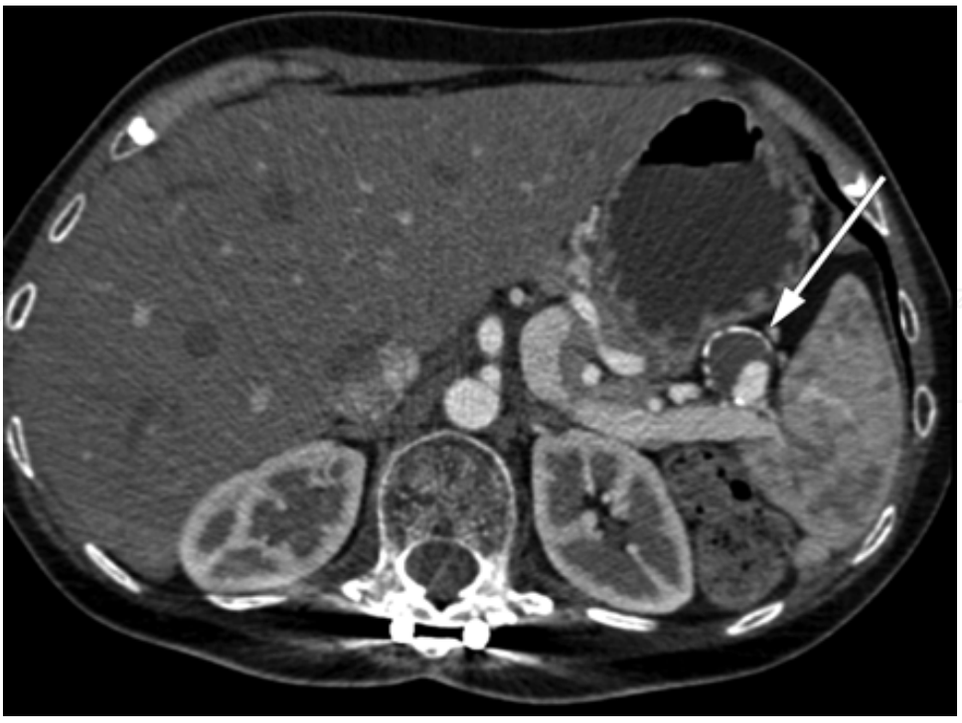

Figure 4. A Peripherally calcified, and thrombosed splenic artery aneurysm, (CT view)

to rupture. In spite of their relatively high prevalence in comparison to other splanchnic aneurysms, there are few large series in the literature. The prevalence of the lesion in the general population is low. A large general autopsy study estimated their all incidence to be 0.01 %(12), whereas more specific examination of the splenic arteries in an autopsy study of patients older than 60 years revealed an incidence of 10 %(13).

The prevalence of incidentally noted aneurysmal changes in the splenic artery on arteriographic studies was reported to be 0.78%, and such changes have been found incidentally in 0.1% to 10% of autopsies. (11).

In contrast to routine atherosclerotic or degenerative aneurismal diseases, SAAs are found much more commonly in women than in men with an approximate ratio of 4:1. (11), they are also noted to occur in younger patients at a mean age of 52 years. (14)

SAAs are usually saccular and less than 2 cm in diameter, with the majority being in the mid or distal portion of the splenic artery or at its bifurcation points. (14)

Giant SAAs with diameter larger than 10 cm have been reported, and in contrast to smaller SAAs, these lesions appear to be more common in men. (15)

3.3. Pathogenesis and aetiology

The most clinical risk factors are the following:

1. Female gender.
2. Multiple pregnancies.

3. Portal hypertension.
4. Systemic hypertension.
5. Arterial fibrodysplasia.
6. chronic inflammatory processes
7. Arteriosclerosis.
8. Less commonly, polyarteritis nodosa, systemic lupus erythematosus, and anomalous splenic artery origin. (16).

In one reported series, it was noted that 80% of the patients with SAAs were females who had an average of 4.5 pregnancies and 50% of females with SAAs had more than 6 pregnancies. (17, 18).

Portal hypertension may be present in 25% of patients with SAAs, while about 10% are awaiting liver transplantation. (19).

Blunt splenic trauma and pancreatitis frequently noted in association with SAAs. Local hemodynamic aspects, hormonal factors, and medial degeneration have all been considered as causative factors in the development of SAAs. (20).

Increased blood volume which results into increased cardiac output, and portal congestion are thought to be related to an increased splenic artery blood flow and SAAs formation. (21).

Impaired elastin formation and degeneration of the internal elastic lamina could be added as hormonal factors which contribute to SAAs formation during pregnancy; It seems that splenic artery is more susceptible to these changes than other vessels. (22).

Histological changes which are noted microscopically during SAAs formation include calcifications, intimal hyperplasia, arterial dysplasia, fibromuscular dysplasia, and medial degeneration. (23).

3.4. Clinical and diagnostic aspects of splenic artery aneurysms

Most SAAs are found incidentally at the time of first presentation during abdominal imaging examination for unrelated disorders. A classic calcified ring may be noted in the left upper abdominal quadrant on a plain x—ray film of the abdomen. (see the image below):

There may be an abdominal bruit, but the majority of cases are showing normal physical examinations especially with asymptomatic patients.

Symptoms are including the following:

1. Vague abdominal pain, nausea and vomiting.(24).
2. Symptoms related to compression of adjacent organs.
3. Sever left-sided pain due to rupture or acute aneurysm expansion.
4. Shock, abdominal distension, and death due to intraperitoneal rupture.
5. Double-rupture phenomenon which may occur in bout 20% to 30% of cases provides a proper diagnosis of rupture into the lesser sac, before free intraperitoneal rupture diagnosed.(25,26).
6. Gastrointestinal tract, pancreatic ducts, or splenic vein rupture.(27).

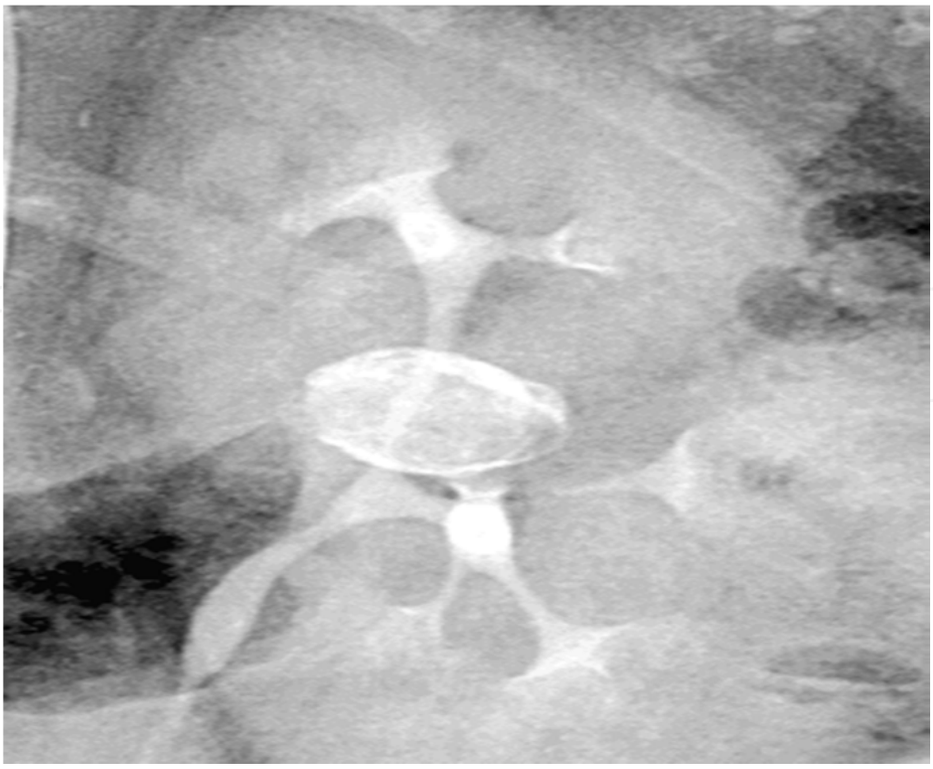

Figure 5.

The overall mortality of ruptured SAAs is about 25%.(26). Pregnancy may be associated with a rate of 20% to 50% of all ruptures.(28).

The association of SAAs and pregnancy is very well documented, in addition to that rupture during pregnancy usually occurs at the third trimester which can lead to maternal and fetal death of 80% to 90%, respectively.(29). Actually this can lead to understand the misdiagnosis of the situation as an obstetric emergency.

Rupture due to portal hypertension is associated with a rate of about 20% .(30).

3.5. Treatment of splenic artery aneurysms

Ruptured , symptomatic SAAs, and those in pregnant women require urgent treatment. Enlarging or those greater than 2 cm in diameter SAAs have less stringent indications, although these criteria are not absolute. Patients with portal hypertension or waiting for liver transplantation should be treated as well.(31). Patient's medical condition and age could play a role the treatment option. Most vascular surgeons would consider suitable elective intervention for asymptomatic patients with lesions those diameter is greater than 2 cm when the surgical risk is thought to be low. If one estimates the incidence of rupture to be 2% with a death rate of at least 25% when rupture has occurred, operative mortality rates should be less than 0.5% to justify elective surgical treatment, in one author's study.(31).

Traditional operative therapy of SAAs includes proximal and distal ligation or aneurysmectomy or both modalities for lesions in the proximal or middle part of the splenic artery.(see the images below).

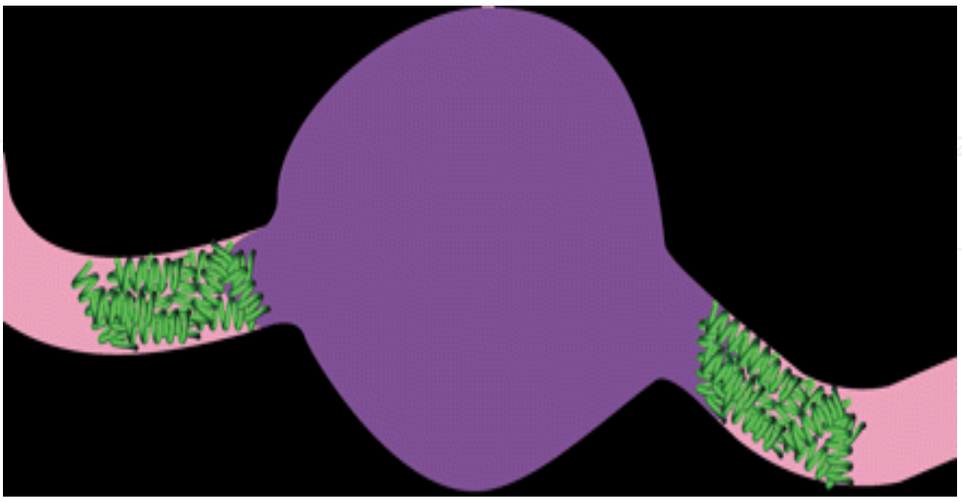

Figure 6. Drawing illustrates how coils are placed distal and then proximal to the aneurysm, thereby trapping the aneurysm and isolating it from the circulation, with resultant thrombosis of the aneurysm.

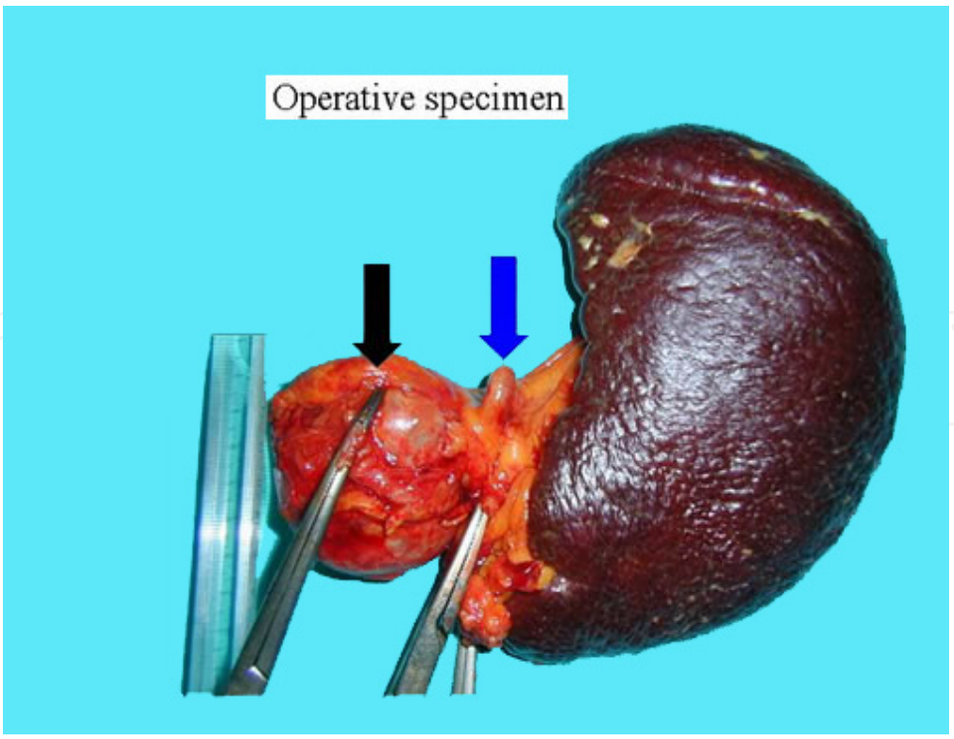

Figure 7.

Revascularization of the distal splenic artery in not generally warranted due to that collateral flow to the spleen in maintained by the short gastric arteries. For those lesions near to the splenic hilum, splenectomy is the most common procedure. Distal pancreatectomy may occasionally be needed for the treatment of these distal lesions as well. (24, 31, 32 and 33).

Laparoscopic repair of SAAs by clipping or exclusion has been reported; intraoperative ultrasonography is believed to be an important adjunct to this procedure.(34). Laparoscopic occlusion combined with coil embolization has been proposed as a treatment for aberrant SAAs located in the retropancreatic position, for which traditional procedures would be exceptionally difficult.(24,35).

Endovascular exclusion of SAAs has been used more recently with good success. Treatment options include coil embolization of the splenic artery both proximal and distal to the aneurysm itself, thereby effectively trapping the lesion. Other options include embolization of the aneurysm sac with coils or cyanoacrylate glue or both modalities simultaneously or occlusion of the lesion with percutaneous or open thrombin injection.(24,36). In addition, stent-grafting has been performed, especially for saccular lesions of the mid splenic artery. There has been some concern regarding splenic infarction and pancreatitis when embolization of very distal splenic artery lesions has been performed. (24, 37 and 38).

The objective of splenic arterial embolization is to improve the results of nonsurgical management. Indications for splenic arterial embolization vary, depending on local management protocols. embolization is performed with microcoils as distally as possible, to preserve perfusion to the splenic parenchyma. Patients with a high risk for secondary rupture of the aneurysm should undergo embolization with coils in a more proximal segment of the splenic artery to reduce the pressure in the splenic parenchyma and help the reservation of the spleen. The placement of coils in a middle segment of the splenic artery allows reconstitution of the blood supply through collateral vessels, principally via the short gastric and gastroepiploic arteries, to the patent distal splenic, transgastric, and transpancreatic arteries. Proximal embolization performed exclusively with coils decreases the volume of splenic arterial blood flow and thereby produces relative hypotension in the splenic bed, which allows the spleen to repair itself without infarction (39)

In a review of 48 endovascular procedures for splanchnic artery pseudoaneurysms, 20 interventions on the splenic artery were performed. Six end-organ infarcts were noticed, all were within the splenic bed. Two additional patients developed splenic atrophy diagnosed on CT scanning after previous embolization of the splenic artery, without clear clinical evidence of initial splenic infarction. (40). In another study, one episode of splenic infarction associated with sever pancreatitis was noted after embolization of a distal splenic artery aneurysm. (37). (see the image below).

Post-embolization transverse contrast-enhanced CT scan obtained at follow-up shows a coil within the splenic artery (arrow), as well as complete infarction of the spleen, which is not contrast enhanced.

However, other authors have reported splenic infarction after embolization of even more proximal SAAs as well. (41).

3.6. Results of different treatment options for splenic artery aneurysms:

The results of open operative therapy are dependent on whether the procedure is an elective or emergency one, in addition to the anatomical complexity of the lesion and the nature of the required repair. Elective procedures have significantly lower perioperative morbidity and mortality compared to the emergency techniques for ruptured aneurysms which carries death rate greater than 50% in many reported series. (42).

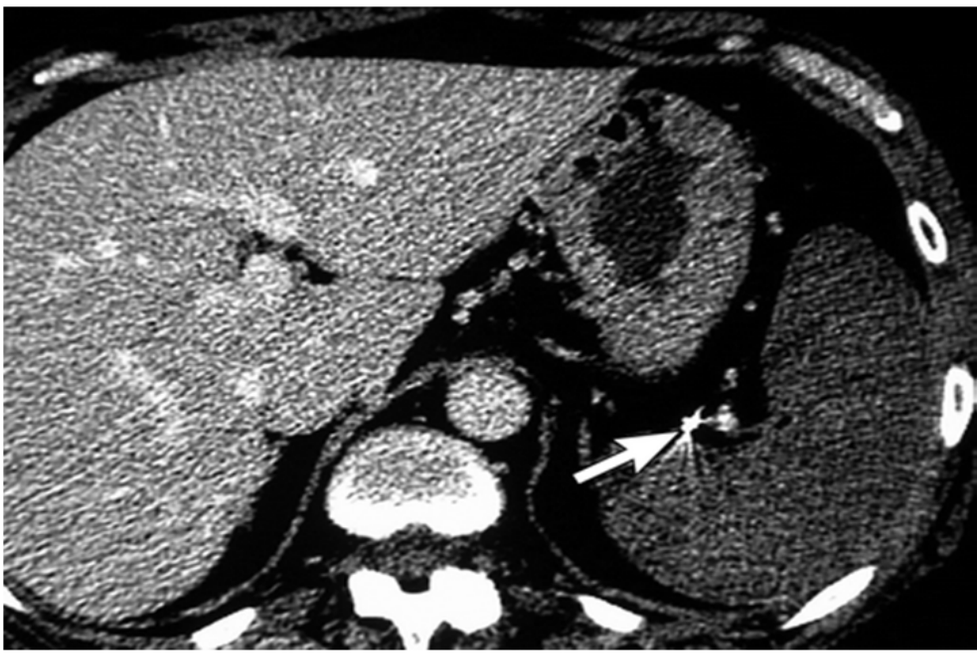

Figure 8.

Technical success after percutaneous coil embolization of SAAs is acceptable and ranges from 81% to 98%, although some studies showed that the presence of hemodynamic instability should not preclude endovascular management. (43,44).

End-organ ischaemia is an especial concern with regard to endovascular repair. Direct complications can result from this option of treatment such as arterial dissection, acute thrombosis, or embolization to nontarget tissues, or inadequate collateral circulation after deliberate vessel occlusion. It has been concluded that cases with aneurysmal lesion at the splenic hilum may be better managed by open repair and splenectomy.(45).

Although initial technical success rates with an endovascular procedure for treating SAAs approach 100%, the long-term success is less well defined.(46).

Ultrasound-guided percutaneous thrombin injection appears to be a viable method for treating failed endovascular interventions or even an alternative to initial endovascular treatment.(47). Actually, this technique is similar to thrombin injections for femoral artery pseudoaneurysms, were ultrasound or CT guidance or both are used to help delivering thrombin to the nidus of an aneurysm, thus facilitating thrombosis. This is particularly applicable to saccular aneurysms with a narrow neck arising from the parent vessel. Continued studies, even after secondary technical success, are imperative due to the natural history of SAAs after endovascular treatment remains unclear. This is true for saccular aneurysms treated by coil or thrombin embolization. Reports of reperfusion and even rupture after successful embolization support that a thrombosed aneurysm may not represent the definitive treatment in all cases.(47,48).

Author details

Ahmad Alsheikhly
Hamad Medical Corporation, Doha, Qatar

4. References

[1] Sadler TW. Chapter 14: Digestive system. In: *Langman's Medical Embryology*. 11th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2009:215-6.

[2] Gray H. Chapter 88: The spleen. In: Standring S, ed. *Gray's Anatomy: The Anatomical Basis of Clinical Practice*. 39th ed. Edinburgh, UK: Churchill Livingstone Elsevier; 1239-44.

[3] Snell RS. Chapter 5: The abdomen: part II. The abdominal cavity. *Clinical Anatomy by Regions*. 8th ed. Baltimore, Md: Lippincott, Williams & Wilkins; 2007:259-60.

[4] Lee McGregor A, Decker GAG, du Plessis DJ. Chapter 8: The spleen. *Lee McGregor's Synopsis of Surgical Anatomy*. 12th ed. Oxford, UK: Butterworth-Heinemann; 1986:106-13.

[5] Romanes GJ. Abdomen: spleen. In: *Cunningham's Manual of Practical Anatomy. Vol II: Thorax and Abdomen*. Vol 2. 15th ed. New York, NY: Oxford Medical Publications, Oxford University Press; 1986.

[6] Guyton AC, Hall JE. Chapter 15: Vascular distensibility and functions of arterial and venous systems. *Guyton and Hall Textbook of Medical Physiology*. 11th ed. Philadelphia, Pa: Saunders; 2005:179-80.

[7] Johnston KW, Tilson MD, Stanley JC. The subcommittee on reporting standards for arterial aneurysm, International society for cardiovascular surgery. Suggested standards for reporting on arterial aneurysm. *J Vass Surg,* 1991; 13:452-458.

[8] Abbas MA, Stone WM, Bower TC, Panneton Jm, Cherry KJ. Splenic artery aneurysms: two decades experience at Mayo clinic. *Ann Vass Surg.* 2002; 16: 442-449.

[9] Carr SC Nemcek AA Jr, Yao JS. Current management of visceral artery aneurysms. *Surgery.* 1996; 120:627-634.

[10] Graham LM, Lindenauer SM. Celiac artery aneurysms: historic (1745- 1949) versus contemporary (1950-1984) differences in etiology and clinical importance. *J Vass Surg*. 1985; 2: 757- 764.

[11] Messina LM, Shanley CJ. Visceral artery aneurysms. *Surg Clin North AM*. 1997; 77:425-442.

[12] Moore SW, Guida PM, Schumacher HW: Splenic artery aneurysm. *Bull Soc Int Chir* 1970; 29:210-218.

[13] Bedford PD, Lodge B: Aneurysm of the splenic artery. *Gut* 1960; 1: 312-320.

[14] Dave SP, Reis ED, Hossain A, et al: Splenic artery aneurysm in the 1990s. *Ann Vasc Surg* 2000; 14:223-229.

[15] Pescarus R, Montreuil B, Bendavid Y: Giant splenic artery aneurysms: case report and review of the literature. *J Vasc Surg* 2005; 42:344-347.

[16] Lee PC, Rhee RY, et al. Management of splenic artery aneurysms: The significance of portal and essential hypertension. *J Am Cool Surg* 1999; 189: 483-490.

[17] Stanley JC, Wakefield TW, Graham LM, et al: Clinical importance and management of splanchnic artery aneurysms. *J Vasc Sur* 1986; 3:836-840.

[18] Sellke FW, Williams GB, Donovan DL, Clarke RE: Management of intra-abdominal aneurysms associated with periarteritis nodosa. *J Vasc Surg* 1986; 4:294-298.

[19] Ayalon A, Wiesner RH, Perkins JD, et al: Splenic artery aneurysms in liver transplant patients. *Transplantation* 1988; 45:386-389.

[20] Trastek VF, Pairolero PC, Joyce JW, et al: Splenic artery aneurysms. *Surgery* 1982; 91:694-699.

[21] Sadat U, Noor N, Tang T, Varty K: Emergency endovascular repair of ruptured visceral artery aneurysms. *World J Emerg Surg* 2007; 2:17.

[22] Hallett Jr JW: Splenic artery aneurysms. *Semin Vasc Surg* 1995; 8:321-326.

[23] Mattar SG, Lumsden AB: The management of splenic artery aneurysms: experience with 23 cases. *Am J Surg* 1995; 169:580-584.

[24] Alsheikhly A.S.: Ruptured True Aneurysm of the Splenic Artery: A Rare Cause of Haemoperitoneum: *The Middle East Journal of Trauma and Emergency medicine* 2008;8:197-199.

[25] Mattar SG, Lumsden AB: The management of splenic artery aneurysms: experience with 23 cases. *Am J Surg* 1995; 169:580-584.

[26] de Vries JE, Schattenkerk ME, Malt RA: Complications of splenic artery aneurysm other than intraperitonealrupture. *Surgery* 1982; 91:200-204.

[27] Wagner WH, Allins AD, Treiman RL, et al: Ruptured visceral artery aneurysms. *Ann Vasc Surg* 1997; 11:342-347.

[28] Trastek VF, Pairolero PC, Joyce JW, et al: Splenic artery aneurysm. *Surgery* 1982; 91:694-699.

[29] Barrett JM, Van Hooydonk JE, Boehm FH: Pregnancy-related rupture of arterial aneurysms. *Obstet Gynecol Surv* 1982; 37:557-566.

[30] Stanley JC, Fry WJ: Pathogenesis and clinical significance of splenic artery aneurysms. *Surgery* 1974; 76:898-909.

[31] Nosher JL, Chung J, Brevetti LS, et al: Visceral and renal artery aneurysms: a pictorial essay on endovascular therapy. *Radiographics* 2006; 26:1687-1704.

[32] Messina LM, Shanley CJ: Visceral artery aneurysms. *Surg Clin North Am* 1997; 77:425-442.

[33] Abbas MA, Stone WM, Fowl RJ, et al: Splenic artery aneurysms: two decades experience at Mayo Clinic. *Ann Vasc Surg* 2002; 16:442-449.

[34] Arca MJ, Gagner M, Heniford BT, et al: Splenic artery aneurysms: methods of laparoscopic repair. *J Vasc Surg* 1999; 30:184-188.

[35] Mastracci TM, Cadeddu M, Colopinto RF, Cina C: A minimally invasive approach to the treatment of aberrant splenic artery aneurysms: a report of two cases. *J Vasc Surg* 2005; 41:1053-1057.

[36] Huang IH, Zuckerman DA, Matthews JB: Occlusion of a giant splenic artery pseudoaneurysm with percutaneous thrombin-collagen injection. *J Vasc Surg* 2004; 40:574-577.

[37] Saltzberg SS, Maldonado TS, Lamparello PJ, et al: Is endovascular therapy the preferred treatment for all visceral artery aneurysms?. *Ann Vasc Surg* 2005; 19:507-515.

[38] Carroccio A, Jacobs TS, Faries P, et al: Endovascular treatment of visceral artery aneurysms. *Vasc Endovasc Surg* 2007; 41:373-382.

[39] LinkDP, Seibert JA, Gould J, Lantz BM. On-line monitoring of sequential blood flow reduction during splenic embolization. *Acta Radio* 1989; 30: 101–*103.*

[40] Tulsyan N, Kashyap VS, Greenberg RK, et al: The endovascular management of visceral artery aneurysms and pseudoaneurysms. *J Vasc Surg* 2007; 45:276-283.

[41] Sachdev U, Baril DT, Ellozy SH, et al: Management of aneurysms involving branches of the celiac and superior mesenteric arteries: a comparison of surgical and endovascular therapy. *J Vas Surg* 2006; 44:718-724.

[42] Pulli R, Dorigo W, Troisi N, et al: Surgical treatment of visceral artery aneurysms: a 25-year experience. *J Vasc Surg 2008*; 48:334-342.

[43] Gabelmann A, Gorich J, Merkle EM: Endovascular treatment of visceral artery aneurysms. *J Endovasc Ther* 2002; 9:38-47.

[44] Salam TA, Lumsden AB, Martin LG, Smith 3rd RB: Nonoperative management of visceral aneurysms and pseudoaneurysms. *Am J Surg* 1992; 164:215-219.

[45] Saltzberg SS, Maldonado TS, Lamparello PJ, et al: Is endovascular therapy the preferred treatment for all visceral artery aneurysms?. *Ann Vasc Surg* 2005; 19:507-515.

[46] Sachdev U, Baril DT, Ellozy SH, et al: Management of aneurysms involving branches of the celiac and superior mesenteric arteries: a comparison of surgical and endovascular therapy. *J Vasc Surg* 2006; 44:718-724.

[47] Szopinski P, Ciostek P, Pleban E, et al: Percutaneous thrombin injection to complete SMA pseudoaneurysm exclusion after failing of endograft placement. *Cardiovasc Intervent Radiol* 2005; 28:509-514.

[48] Carr SC, Pearce WH, Vogelzang RL, et al: Current management of visceral artery aneurysms. *Surgery* 1996; 120:627-633.

[49] Onohara T, Okadome K, Mii S, et al: Rupture of embolized coeliac artery pseudoaneurysm into the stomach: is coil embolization an effective treatment for coeliac anastomotic pseudoaneurysm?. *Eur J Vasc Surg* 1992; 6:330-332.

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