Introduction:
Vascular diseases of the spleen are relatively uncommon in clinical practice. Nevertheless, the reported incidence is increasing every year, possibly due to advances in the imaging techniques and increased attention towards disorders of the spleen. Among all splenic vascular diseases, aneurysms and pseudoaneurysms are of major clinical significance due to their complications. In this study, we present five cases of splenic artery aneurysm, which presents 4.63% out of 108 organ complexes. We also provide a detailed description of the cases therefore demonstrating their variability. The splenic artery branched into two arteries of the I order in 75%, three arteries – 11.11%, four arteries – 4.63%, without branching – 9.26%. There were superior polar arteries in 37.96% and inferior polar arteries in 42.59%. There was no correlation between the presence of SAAs and the length or width of the spleen, splenic artery diameter, number of terminal branches or polar arteries (p>0.05).

Key Words: Dissection, Spleen, Splenic artery, Splenic artery aneurysms

Material and Methods
The study was conducted using macroscopic dissection. The total number of organs was 108 and included the spleen, pancreas and duodenum. The organs for macroscopic dissection were preserved in 10% formaldehyde solution and then carefully dissected. The organs complexes where the splenic artery (SA) had a localized enlargement of more than 50% of its diameter were sectioned and subjected to hematoxylin and eosin staining. The results of histology then were analyzed using microscopy. Statistical analysis was performed using SPSS 20. The data is presented in means with standard deviations. Spearman’s correlation was used to assess during necropsy.(3) Nevertheless, centuries later the true prevalence of SAAs is still controversial. They are considered the most frequent type of visceral aneurysms.(4) They can be seen in 0.098% of cases during autopsy and in 0.78% of cases during arteriograms.(3, 5) The prevalence seems to vary and they become more frequent as patients get older. In patients older than 60 years they can be seen in up to 10.4% of cases.(6) Since SAAs are associated with a high mortality rate (25-95%), prompt diagnosis and management of SAAs is of prime importance.(7) It is also essential to differentiate aneurysms with a low risk of rupture which may be treated conservatively but require regular imaging to evaluate their progress. This is done based on the assessment of risk factors such as SAAs that are symptomatic, enlarging more than 2 cm in diameter or those detected in pregnancy, childbearing age or following liver transplantation which have higher risk of rupture.(8)

Abdominal surgery is founded on a profound understanding of the blood supply to the abdominal organs.(9) Detailed knowledge of the regional anatomy and pathology as well as peculiarities of the vascular supply, collateral paths of vascularization are essential for general, vascular and endovascular surgery.(10)
the relationship between the presence of SAAs and the length and width of the spleen, splenic artery diameter, number of terminal branches or polar arteries. A p value of less than 0.05 was considered statistically significant.

Results
Out of 108 organ complexes, there were five cases (4.63%) of SAAs. The splenic length was 9.53±3.63 cm, splenic width 6.73±2.71 mm, SA diameter 5.65±1.70 mm. The SA branched into two arteries of the 1 order in 75%, three arteries – 11.11%, four arteries – 4.63%, without branching – 9.26%. There were superior polar arteries in 37.96% and inferior polar arteries in 42.59%. There was no correlation between the presence of SAAs and the length or width of the spleen, SA diameter, number of terminal branches or polar arteries (p>0.05).

In the first case, an eccentric round bag-shaped aneurysm was located on the anterior semicircle of the SA, in its middle third. It had a wide neck and a body with a diameter of 0.5 cm. The SA had a sinus shape, located on the upper edge of the pancreas and had a distributed branching pattern (Fig.1).

In the second case, the scaphoid oval aneurysm had a longitudinal length of 1.1 cm and a transverse size of 0.7 cm. The aneurysm was located between the proximal and middle third of the SA, immediately after branching of great pancreatic artery of Popova. The SA had a sinusoidal trajectory, passing along the posterior surface of the pancreas; branched into 2 branches of the first order, and also gave a large superior polar artery and several arteries to the tail of the pancreas (Fig.2).

In the third case, the aneurysm had a mixed form (both saccular and fusiform aneurysm). It was localized in the proximal third of the SA and measured 1.3 x 0.7 cm. The SA had a direct trajectory and magistral branching pattern (Fig.3).

The fourth case was represented by a large spindle-shaped aneurysm with dimensions of 2 and 1.5 cm, located transversely, at the site of the splenic artery bifurcation. From the side walls of the aneurysm 2 vessels emerged, with a diameter of 0.5 cm, which were branches of the first order of the SA. The artery itself had a spiral trajectory, thickened walls with diffuse atherosclerotic lesions and multiple calcifications (Fig.4).

In the fifth case, a giant dissecting aneurysm (longitudinal size - 2.7 cm, transverse size in the widest part – 2.1 cm) had a convoluted shape, and was located at the level of the pancreatic head, at the site of artery of the pancreatic head, extending to the proximal part vessel (at the distance of 1.2 cm). The aneurysm had 2 chambers, since there were 2 microruptured media vessels. The large chamber was located proximally, closer to the branching site. The splenic artery had a direct trajectory, located in the proximal intraorganic part and then above the surface of the pancreas (Fig.5).
Discussion

There are several physiological and pathological risk factors contributing to SAAs formation. Patients with SAAs tend to have a longer splenic artery and more prominent curvature compared to the non-vascular control patients. The average curvature of the SA in patients is associated with the dilatation rate in females.(11) Aneurysm growth is also strongly associated with an increased diameter of the SA trunk, which depends on the arterial flow.(12) Up to 50% of cases occur in the region of the splenic hilum.(12) The increased pressure, turbulent blood flow at the branching point of the artery and uneven pressure along the splenic artery can all contribute to SAA formation. Although this is not demonstrated in the current study due to the limited number of cases of SAAs. Although the true etiology of SAAs remains unknown, it has been associated with several conditions as hypertension, portal hypertension, cirrhosis, liver transplantation, and pregnancy.(13-15) Enlargement of the splenic vein seemed to be a predictive factor for the development of SAA as it is seen in a significant proportion of patients with cirrhosis.(16) Other conditions may include arterial fibrolympasia, arteritis, collagen vascular disease, AI-antitrypsin deficiency, and inflammatory and infectious disorders.(13) SAAs are also frequently seen in children with underlying conditions are presented. In children with chronic liver disease and long-standing portal hypertension, they can be seen in up to 10.4%. There is also a statistically significant difference in regards to the size of spleen and patient age between children with SAAs and without.(17) In patients with Wilson’s disease, SAAs are seen in up to 61.1% which is higher compared to other causes of cirrhosis and portal hypertension. There were significant differences between the patients with SAA and those without SAA with respect to splenic artery diameter, portosystemic collateral vessel diameter, and spleen volume (p=0.007, p=0.001, and p=0.006, respectively).(18) Approximately 80% of SAAs show atherosclerotic changes including calcification and mural thrombus may be present.(19) Risk of rupture of SAAs is 2 to 3%. (20, 21) However, this can be variable, for instance the prevalence of SAAs in childhood-aged females and incidence of rupture during pregnancy are reported to be less than 0.1%. (22) Typical clinical presentations of ruptured SAAs include abdominal pain, hemodynamic instability, and gastrointestinal bleeding. The sudden onset of left upper quadrant pain usually indicates rupture of the aneurysm.(23) Ruptured splenic aneurysm can rapidly lead to hemopteroneum and hemorrhagic shock.(24) There can be spontaneous stabilization and subsequent sudden circulatory collapse. This is termed the “double rupture” phenomenon, when initial bleeding tamponades into the lesser sac, followed by flooding into the peritoneal cavity.(25) The morphology and the site of origin of the splenic aneurysm should also be taken into account. The artery can frequently have other sites of origin besides the celiac trunk, which may complicate the management of the disease.(26) As uncommon as true SAAs are, pseudoaneurysms are even rarer. In a large series from the Mayo Clinic, 10 SA pseudoaneurysms were compiled over 18 years.(27) The risk of rupture of a SA pseudoaneurysm is higher and can be up to 37%, with the mortality rate approaching 90% when left untreated.(28, 29) Unlike SAAs, SA pseudoaneurysms are practically always symptomatic and only 2.5% of cases presented incidentally.(27) The most common presentations are abdominal pain (29.5%), hematochezia or melena (26.2%), hemorrhage into the pancreatic duct (20.3%), and hematemesis (14.8%).(27) In a study performed in the Mayo Clinic over a period of two decades. This analysis of the patient data lead to the conclusion that although SAAs may rupture, not all intact aneurysms need intervention. Calcification did not appear to protect against rupture, although beta-blockade may be protective. Growth rates of SAA are slow and growth is infrequent. The recommendations were that open ligation or transcathether embolization should be considered for symptomatic aneurysms, for aneurysms equal or more than 2 cm in size, or for any SAA in women of childbearing years.(13) Another study advocates that for patients aged over 60 years with no symptoms and with aneurysms less than 20 mm in diameter, conservative management with CT scans every six to twelve months is advocated.(30) MDCT is a noninvasive and valuable method in diagnosis of SAAs and has higher value than ultrasonography in determination of treatment plan.(31) The primary therapeutic approach should be endovascular therapy by either embolization or stent grafting.(8) In embolization of the SAA there can be complications as splenic infarction, pancreatitis and postembolization syndrome.(32) The complication of stenting mostly include stent migrations and thrombosis.(33) End-to-end anastomosis can be used in cases where stenting or coiling is unavailable or cannot be used.(34) It is also often used in resource-limited settings.

Conclusions

Among all splenic vascular disease, aneurysms and pseudoaneurysms are of major clinical significance due to their complications. During the macroscopic dissection of 108 organ complexes, we have found five cases (4.63%) of SAAs. The SSAs had round bag form, scaphoid oval, spindle, and mixed form. One case of giant dissecting aneurysm was also detected. The splenic length was 9.3±3.63 cm, splenic width 6.73±2.71 mm, splenic artery diameter 5.65±1.70 mm. The longitudinal length varied from 0.5 to 2.7 cm, and the transverse length - from 0.5 to 2.1 cm. The splenic artery branched into two arteries of the I order in 75%, three arteries – 11.1%, four arteries – 4.63%, without branching – 9.26%. There were superior polar arteries in 37.96% and inferior polar arteries in 61.1% which is higher compared to other circumstances. There were significant differences as splenic artery and more prominent curvature of the SA in patients is associated with the curvature of the splenic hilum.(12) The increased pressure, turbulent blood flow at the branching point of the artery and uneven pressure along the splenic artery can all contribute to SAA formation. Although this is not demonstrated in the current study due to the limited number of cases of SAAs. Although the true etiology of SAAs remains unknown, it has been associated with several conditions as hypertension, portal hypertension, cirrhosis, liver transplantation, and pregnancy. SAAs are also frequently seen in children with underlying conditions are presented. In children with chronic liver disease and long-standing portal hypertension, they can be seen in up to 10.4%. There is also a statistically significant difference in regards to the size of spleen and patient age between children with SAAs and without. In patients with Wilson’s disease, SAAs are seen in up to 61.1% which is higher compared to other causes of cirrhosis and portal hypertension. There were significant differences between the patients with SAA and those without SAA with respect to splenic artery diameter, portosystemic collateral vessel diameter, and spleen volume (p=0.007, p=0.001, and p=0.006, respectively). Approximately 80% of SAAs show atherosclerotic changes including calcification and mural thrombus may be present. Risk of rupture of SAAs is 2 to 3%. However, this can be variable, for instance the prevalence of SAAs in childhood-aged females and incidence of rupture during pregnancy are reported to be less than 0.1%. Typical clinical presentations of ruptured SAAs include abdominal pain, hemodynamic instability, and gastrointestinal bleeding. The sudden onset of left upper quadrant pain usually indicates rupture of the aneurysm. Ruptured splenic aneurysm can rapidly lead to hemopteroneum and hemorrhagic shock. There can be spontaneous stabilization and subsequent sudden circulatory collapse. This is termed the “double rupture” phenomenon, when initial bleeding tamponades into the lesser sac, followed by flooding into the peritoneal cavity. The morphology and the site of origin of the splenic aneurysm should also be taken into account. The artery can frequently have other sites of origin besides the celiac trunk, which may complicate the management of the disease. As uncommon as true SAAs are, pseudoaneurysms are even rarer. In a large series from the Mayo Clinic, 10 SA pseudoaneurysms were compiled over 18 years. The risk of rupture of a SA pseudoaneurysm is higher and can be up to 37%, with the mortality rate approaching 90% when left untreated. Unlike SAAs, SA pseudoaneurysms are practically always symptomatic and only 2.5% of cases presented incidentally. 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MDCT is a noninvasive and valuable method in diagnosis of SAAs and has higher value than ultrasonography in determination of treatment plan. The primary therapeutic approach should be endovascular therapy by either embolization or stent grafting. In embolization of the SAA there can be complications as splenic infarction, pancreatitis and postembolization syndrome. The complication of stenting mostly include stent migrations and thrombosis. End-to-end anastomosis can be used in cases where stenting or coiling is unavailable or cannot be used. It is also often used in resource-limited settings.

References