JBR–BTR, 2012, 95: 61-65.

THE ACCESSORY SPLEEN: PREVALENCE AND IMAGING FINDINGS IN 1,735 CONSECUTIVE PATIENTS EXAMINED BY MULTIDETECTOR COMPUTED TOMOGRAPHY

T. Romer, W. Wiesner[1,2]

Objective: **To analyze the prevalence and CT findings of accessory spleens in the normal population.**
Material and methods: **CT-examinations of 1735 consecutive patients, all examined by triple phase 16-row multidetector computed tomography (MDCT) of the abdomen, were retrospectively analyzed with special emphasis on the presence, location and imaging aspects of accessory spleens.**
Results: **199 patients showed an accessory spleen (11%). Size of accessory spleens ranged from 3 to 20 mm (mean 10 mm). In 60% the accessory spleen was located at the level of the splenic hilum and in 33% at the level of the lower pole. In 46% the accessory spleen was located medially and in 43% ventrally. 19 patients presented with two (1.1%) and seven patients with three accessory spleens (0.4%), respectively. One patient showed splenosis and one patient showed an enlarged accessory spleen (5 cm) secondary to a splenic apoplexy (i.e. hemorrhagic infarction) of the accessory spleen, caused by torsion.**
Conclusion: **Accessory spleens may be identified by MDCT in about 11% of patients. Familiarity with normal imaging findings and knowledge on differential diagnoses, possible pathologies and potential pitfalls helps to differentiate from other findings in the upper abdomen.**

Key-words: Spleen, abnormalities.

Accessory spleens represent congenital variants of small isles of normally encapsulated splenic tissue with its own arterial and venous vascularisation and their prevalence seems to range between 10 and 20% (1-5). Accessory spleens do undergo the same pathophysiological mechanisms as the main spleen and, therefore, they may show all the potential lesions that may be observed in the main spleen, ranging from shrinking to swelling, from calcification to cyst or tumor formation and from haemorrhage to infarction. Most accessory spleens are located near the pancreatic tail and the splenic hilum and accurate identification of an accessory spleen may be important to avoid misinterpretation and differentiation from pathologic findings in the upper abdomen (6-8).

The only study on accessory spleens – detected by CT – reported even a prevalence of 16% around ten years ago (9). However, this study was performed at the beginning of the era of multidetector computed tomography - using dual phase CT with a collimation of 8 mm for both, the unenhanced and the enhanced portal venous phase scans. Therefore, the present study was initiated with the intention to analyze if an increased spatial resolution by using triple phase MDCT with a slice thickness of 3.75-5 mm would be able to detect more accessory spleens in the normal human population than reported in the literature until present. The present study addresses the CT-prevalence of accessory spleens in the normal population followed by a discussion of normal imaging findings, differential diagnoses and potential pitfalls that all may be helpful for their correct identification and differentiation from other findings in the upper abdomen.

Material and methods

1735 consecutive patients, all examined over a time period of two years using 16-row MDCT (LightSpeed, GE, Milwaukee, USA) by triple phase CT (including unenhanced, arterial (35 sec.) and portal-venous phase (70 sec.) CT) were included into the study. Patients who have undergone splenectomy and patients with a potential reason for a splenic enlargement (liver cirrhosis, severe infection, sepsis, haematologic disorders etc.) were excluded from the study.

All patients received positive oral contrast medium before CT (1 litre of Telebrix Gastro, orally taken over 1 hour, Guerbet, France). Triple phase MDCT was performed using 16-row MDCT. 100 ml of intravenous contrast material (Visipaque 320, GE Medical, Milwaukee, USA) was injected with a flow rate of 2-3 ml/sec. Arterial phase CT data acquisition started 35-40 seconds and portal-venous phase CT started 70-75 seconds after the intravenous administration of 100 ml of contrast material. CT parameters were as follows: Rotation time 0.8, helical thickness: 1.25 mm, pitch: 1.375:1, speed 27.5 mm, interval: 0.6 mm, KV 120, mAs (auto-mA): 400. Reconstructed slice thickness (and interval) was 5 mm for the unenhanced and the portal-venous phase images and 3.75 mm for the arterial phase images. Image analysis was performed on an integrated RIS/PACS work station (Centricity, GE Milwaukee, USA).

All CT examinations were retrospectively analyzed by a radiologist, specialized in abdominal imaging on a double monitor work station of an integrated RIS/PACS System (Centricity, GE, Milwaukee) using both, slice after slice and cine-viewing – technique in the axial plane, whereas multiplanar reconstructions were used only in selected and unclear cases. The reader was unaware of the reasons for the CT examination and of the clinical histories of the patients. Special emphasis was given on the presence, size, number and location, and if present, on visible pathologies of accessory spleens.

The presence of an accessory spleen was confirmed only if it could be clearly depicted as a well circumscribed mass with similar attenuation and contrast enhancement as

From: 1. Department of Radiology, University Hospital Basel, Basel, Switzerland, 2. Institute for Diagnostic Radiology, Clinic Stephanshorn, St. Gallen, Switzerland.
Address for correspondence: PD Dr. med. W. Wiesner, Clinic Stephanshorn, Brauerstrasse 95, 9016 St. Gallen, Switzerland.. E-mail: walter.wiesner@stephanshorn.ch

Table I. — Location of accessory spleen.

Location	%
Upper pole	
dorsal	0.0%
ventral	3.5%
lateral	2.5%
medial	0.0%
	6.0%
Splenic hilum	
dorsal	2.5%
ventral	22.6%
lateral	1.5%
medial	33.7%
	60.3%
Lower pole	
dorsal	3.0%
ventral	17.1%
lateral	1.0%
medial	12.6%
	33.7%

the main spleen on all three phases. Two patients (one with splenosis following traumatic rupture of the spleen and one with apoplexy of his accessory spleen (i.e. haemorrhagic infarction due to torsion) were not excluded from the study according to the importance of the imaging findings, but they were excluded from the statistical analyses regarding number of accessory spleens and size, respectively.

Results

199 patients (11.5%) showed at least one accessory spleen, which could clearly be depicted on all appropriate CT images, but usually was seen best on the thin collimated arterial phase CT. 19 patients had two accessory spleens (1.1%) and seven patients three accessory spleens (0.4%).

45,5% of the patients were male and 54,5% of the patients were female. Female patients showed accessory spleens in 11.7% and male patients in 11.2% of cases. The size of accessory spleens ranged from 3 mm to 20 mm (mean 10.3 mm) in female patients and from 3-24 mm (mean 9.8 mm) in male patients.

In 60.3% of patients the accessory spleen was located near the splenic hilum and the pancreatic tail, and in 33.7% at the level of the splenic lower pole – usually ventrally (43.1%) and medially (46.4%) to the spleen, respectively, whereas only 6% of accessory spleens were found at the level of the upper pole (Table I, Fig. 1).

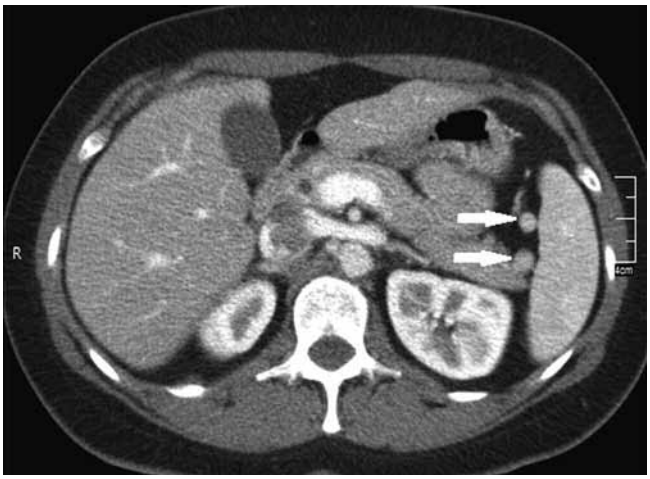

Fig. 1. — Two accessory spleens (arrows) with a typical location near the splenic hilum.

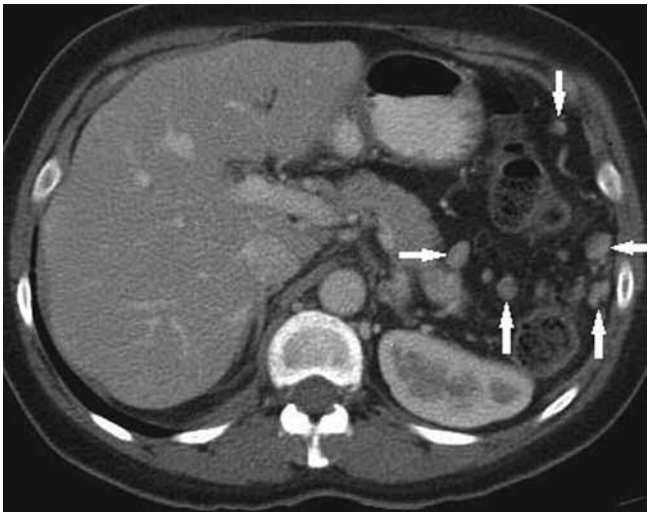

Fig. 2. — Multiple nodules spread within the peritoneal cavity in the left upper abdominal quadrant (arrows) in a patient with splenosis – mimicking nodular peritoneal carcinomatosis.

80% of the accessory spleens were round, whereas 20% showed a more oval shape. Attenuation and contrast enhancement paralleled the values of the normal spleen in all cases, in which the accessory spleen was larger than 5 mm, whereas accessory spleens measuring less than 5 mm usually seemed to show less enhancement than the spleen - probably due to partial volume effects.

One patient showed splenosis with multiple spleens throughout the entire abdomen, measuring between 1 mm and 20 mm and mainly located along the greater omentum (Fig. 2). Another patient showed a single, but enlarged accessory spleen, measuring 5 cm with inhomogeneous attenuation and absence of contrast enhancement (despite the capsule) due to torsion and subsequent venous haemorrhagic infarction, (i.e. apoplexy of the accessory spleen) (Fig. 3).

Discussion

Accessory spleens may be found in up to 23.8% of golden hamsters (1), whereas their prevalence in the human seems to range between 10% to 20% as published by several studies based on anatomical and pathological examinations (2-5). However, the only publication that reported a prevalence of 20% was based on a quite small anatomical study including only 250 patients (6), whereas all other studies, and especially the largest and surely

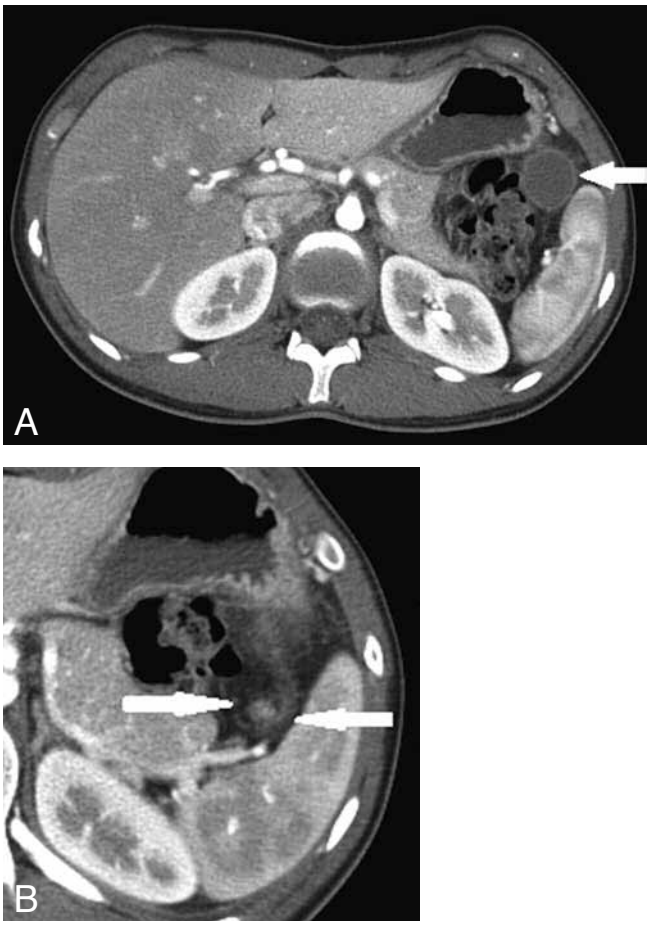

Fig. 3. — A. Large round lesion superior to the spleen with low attenuation. Except for some peripheral rim enhancement (arrow) the lesion does not enhance after contrast administration. B: Whirl sign (arrows), which shows torsion of the vessels supplying this mass and deriving from the splenic vasculature, confirms haemorrhagic infarction (i.e. apoplexy) of an accessory spleen.

most representative anatomical study, including 3000 patients (2), showed accessory spleens only in 311 cases – i.e. in around 10% of patients (2-5).

Embryology and anatomy

Accessory spleens are little islands of encapsulated splenic tissue with its own arterial and venous vascularisation and therefore they represent a kind of simple heterotopy. Accessory spleens develop during the fifth fetal week of life in the dorsal mesogastrium and move to the left upper abdomen due to the rotation of the stomach, finally fixed at this position by the gastrosplenic ligament ventrally and the splenorenal ligament dorsally (10, 11). Therefore, accessory spleens are usually located near the splenic hilum and close to the spleen, but sometimes they may be found also in the pancreatic tail, in the wall of the stomach, the large bowel, the omentum, the mesentery and rarely even in the scrotum (6, 12, 13).

Diagnosis and pitfalls

An exact knowledge on the presence of an accessory spleen may be of great value – not only in cases in which splenectomy is necessary due to splenic rupture and where an accessory spleen should be preserved but also in cases of certain hematologic disorders, where all splenic tissue should be removed. Therefore, accessory spleens should be differentiated from other developmental anomalies such as splenic clefts and lobulations and one should be aware that sometimes venous collaterals in portal venous hypertension and rarely even aneurysms of the splenic artery may mimic accessory spleens at first sight, especially if only portal venous phase is acquired.

Correct identification of an accessory spleen is also important in order to avoid misinterpretation of an accessory spleen for lymphadenopathy, a metastasis or an adrenal or renal tumor – and this is especially challenging when accessory spleens are found in an atypical locations – as for example in the pancreatic tail – where they may mimick a pancreatic neoplasm (14-19). Such cases were not present in our study, but with modern CT- and MR-imaging this differentiation is usually possible nowadays.

Lesions and complications

It is well known, that accessory spleens undergo the same pathophysiologic changes as the normal spleen: they may shrink in case of infarction, as for example in sickle cell disease and they may show a swelling, as for example in case of portal venous hypertension or in certain haematologic disorders causing splenomegaly (2, 7).

This is especially evident after splenectomy - when the accessory spleen takes over the physiologic function of the main spleen, leading to an incredible enlargement of the accessory spleen, which may grow under these circumstances to a size of up to 7 cm although still keeping its original round shape (5, 20). Accessory spleens may show also the same focal lesions as the main spleeen – ranging from cyst or even tumour and abscess formation to rupture, haemorrhage and infarction (21-23).

Although these findings are extremely rare according to the small size of accessory spleens, splenic apoplexy (i.e. haemorrhagic infarction of the accessory spleen) has occurred once in our series. Such apoplexy of an accessory spleen typically leads to unspecific subacute pain in the upper left abdominal quadrant and imaging findings consist of a mass with diffusely reduced and inhomogeneous attenuation and reduced or even absent contrast enhancement, which may be preserved only along the capsule (21-23). Although these imaging findings are not specific, identification of a "whirl sign" along the vessels leading to the lesion is almost pathognomonic for this rare condition (Fig. 3).

Differential diagnoses

A rare differential diagnosis of multiple spleens is splenosis, an acquired finding that was found

once in our study population. Splenosis results from seeding and implantation (auto transplantation) of splenic tissue following splenic rupture, where the small and mostly multiple nodules, whose blood supply – unlike in an accessory spleen – derives from neovascularisation, may be found throughout the abdominal cavity - mostly adjacent to the greater omentum, the parietal peritoneum, the small bowel serosa or the diaphragm (24). Although these imaging findings may sometimes lead to a misdiagnosis according to the fact that they may mimic nodular peritoneal carcinomatosis knowledge about this entity, together with the patient's history, usually allows one to make the correct diagnosis by CT (24).

Another very rare differential diagnosis, that has to be differentiated from multiple accessory spleens is Polysplenia – in which up to 16 spleens may be found in the left upper abdomen – measuring from one to several centimeters each (25,26). Polysplenia is a congenital syndrome (synonyme: Isomerie, Ivemark-Syndrome, bilateral left sideness) in which various anatomical and especially cardiovascular anomalies are found in combination with a quite variable situs and which belongs to the same family of malformations as asplenia (i.e. bilateral right sideness) in which no splenic tissue at all will be found due to total agenesis of the spleen (27, 28).

Prevalence and findings

Accessory spleens seem to be quite common in the human population. They usually do not exceed a size of 25-30 mm and they typically are found mainly at the level of the middle third and the lower pole of the spleen located mostly medially or ventrally (3, 7, 8, 9). Accessory spleens may therefore be identified with a high accuracy by CT when a small round or oval mass is detected at the mentioned regions which shows an attenuation and an enhancement pattern similar to that of the spleen.

Nevertheless, Mortele et al. investigated 1`000 patients by MDCT and they found accessory spleens in 16%, which stays in contrast to our results at first sight and especially to prior publications (9). Mortele et al. have therefore postulated that a thinner collimation at MDCT might probably lead to detection of even more accessory spleens – and this might even be right!

We did not use a slice thickness of 1 mm and we did not use a systematic postprocessing, which might be regarded as a certain limitation of our study, but although slice thickness was thinner (3.75-5 mm) in our study than in the study of Mortele (8 mm), and although all our patients were examined not only by dual phase but even by triple phase CT, we did not find more but less accessory spleens.

One explanation for this discrepancy could be that this difference derives just from varying prevalences of accessory spleens in varying study populations or even from ethnic differences and although one might argue that we probably just missed some small accessory spleens in our study despite the fact that we used a higher resolution, it is obvious that our results are almost identical with the results of the most representative study ever, published by Halpert et al. who found 311 patients with accessory spleens among a total of 3,000 autopsies (2).

Furthermore, there were no enlarged accessory spleens in our study according to our exclusion criteria which excluded patients with potential reasons for splenomegaly and, therefore, one might speculate about the varying number of very small accessory spleens that remained undetectable in the studies based on necropsies and also in our present study based on thin – slice MDCT – findings.

Hypothesis

As explained in the beginning, accessory spleens undergo the same pathophysiological mechanisms as the main spleen and, therefore, they may show an enlargement in any situation that leads to splenomegaly.

Certain authors have published an increased "incidence of accessory spleens" of up to 30% in patients with such haematologic disorders (5) and, although the term "incidence" is not appropriate in this setting (since accessory spleens are present or absent and since they never develop de novo!), these studies support the hypothesis, that even in the time of modern MDCT-scanning, probably more than 50% of all accessory spleens remain just undetectable during life due to their very small size as long as they are not forced to grow.

Conclusion

Even by using MDCT with a slice thickness of 3.75-5 mm accessory spleens may be identified only in around 11% of patients during routine scans. Although it is obvious that such results will never be able to represent the real prevalence of all accessory splenic tissue in the human being, a detailed knowledge on the embryology and pathophysiology of this entity, together with its differential diagnoses and potential pitfalls will surely be helpful for a correct interpretation of this interesting, but statistically underestimated congenital condition.

References

1. Yoon Y.S., Shin J.W., Park C.B., et al.: Morphological structure of accessory spleen in Chinese hamsters. *J Vet Sci*, 2000, 1: 73-75.
2. Halpert B., Gyorkey F.: Lesions observed in accessory spleens of 311 patients. *Am J Clin Pathol*, 1959, 32: 165-168.
3. Appel M.F., Bart J.B.: The surgical and hematologic significance of accessory spleens. *Surg Gynecol Obstet*, 1976, 143:, 191-192.
4. Curtis G.M., Movitz D.: The Surgical Significance of the Accessory Spleen. *Ann Surg*, 1946, 123: 276-298.
5. Olsen W.R., Beaudoin D.E.: Increased incidence of accessory spleens in hematologic disease. *Arch Surg*, 1969, 98: 762-763.
6. Wadham B.M., Adams P.B., Johnson M.A.: Incidence and location of accessory spleens. *N Engl J Med*, 1981, 304: 1111.
7. Hackl H.: Ueber Vorkommen und Entstehung von Nebenmilzen. *Bruns Beitr Klin Chir*, 1959,, 198: 128-138.
8. Barawi M., Bekal P., Gress F.: Accessory spleen: a potential cause of misdiagnosis at EUS. *Gastrointest Endosc*, 2000, 52: 769-772.
9. Mortelé K.J., Mortelé B., Silverman S.G.: CT features of the accessory spleen. *AJR*, 2004, 183: 1653-1657.
10. Varga I., Galfiova P., Adamkov M., et al.: Congenital anomalies of the spleen from an embryological point of view. *Med Sci Monit*, 2009, 15: 269-276.
11. Walther M.M., Trulock T.S., Finnerty D.P., Woodard J.: Splenic gonadal fusion. *Urology*, 1988, 32: 521-524.
12. Altaf A.M.S., Sawatzky M., Ellsmere J., et al.: Laparoscopic accessory splenectomy: the value of perioperative localization studies. *Surg Endosc*, 2009, 23: 2675-2679.
13. Ungör B., Malas M.A., Sulak O., Albay S.: Development of spleen during the fetal period. *Surg Radiol Anat*, 2007, 29: 543-550.
14. Spencer L.A., Spizarny D.L., Williams T.R.: Imaging features of intrapancreatic accessory spleen. *Br J Radiol*, 2010, 83: 668-673.

15. Meiler R., Dietl K., Novák K., Patzel C.: Intrapancreatic accessory spleen. *Int Surg*, 2010, 95: 183-187.
16. Schwartz T.L., Sterkel B.B., Meyer-Rochow G.Y., et al.: Accessory spleen masquerading as a pancreatic neoplasm. *Am J Surg*, 2009, 197: 61.
17. Schreiner A.M., Mansoor A., Faigel D.O., Morgan T.K.: Intrapancreatic accessory spleen: Mimic of pancreatic endocrine tumor diagnosed by endoscopic ultrasound-guided fine-needle aspiration biopsy. *Diagn Cytopathol*, 2008, 36: 262-265.
18. Meyer T., Maier M., Höller S., Fein M.: Intrapankreatische Nebenmilz als Differentialdiagnose des Pankreasschwanzkarzinomes. *Zentralbl Chir*, 2007, 132: 73-76.
19. Läuffer J.M., Baer H.U., Maurer C.A., Wagner M., Zimmermann A., Büchler M.W.: Intrapancreatic accessory spleen. A rare cause of a pancreatic mass. *Int J Pancreatol*, 1999, 25: 65-68.
20. Zwas S.T., Samra D., Samra Y., Sibber G.R.: Scintigraphic assessment of ectopic splenic tissue localization and function following splenectomy for trauma. *Eur J Nucl Med*, 1986, 12: 125-129.
21. Impellizzeri P., Montalto A.S., Borruto F.A., et al.: Accessory spleen torsion: rare cause of acute abdomen in children and review of literature. *J Pediatr Surg*, 2009, 44: 15-18.
22. Kim T.H., Kim J.K., Park M.J., Lee J.H.: Education and imaging. Hepatobiliary and pancreatic: torsion of an accessory spleen. *J Gastroenterol Hepatol*, 2009, 24: 1308.
23. Pérez Fontán F.J., Soler R., Santos M., Facio I.: Accessory spleen torsion: US, CT and MR findings. *Eur Radiol*, 2001, 11: 509-512.
24. Tsitouridis I., Michaelides M., Sotiriadis C., Arvaniti M.: CT and MRI of intraperitoneal splenosis. *Diagn Interv Radiol*, 2010, 16: 145-149.
25. Maier M., Wiesner W., Mengiardi B.: Annular pancreas and agenesis of the dorsal pancreas in a patient with polysplenia syndrome. *AJR*, 2007, 188: 150-153.
26. Seo H., Jeon T.Y., Sim M.S., Kim S.: Polysplenia syndrome with preduodenal portal vein detected in adults. *World J Gastroenterol*, 2008, 14: 6418-6420.
27. Gayer G., Zissin R., Apter S., Atar E., Portnoy O., Itzchak Y.: CT findings in congenital anomalies of the spleen. *Br J Radiol*, 2001, 74: 767-772.
28. Mahlaoui N., Minard-Colin V., Picard C., et al.: Isolated Congenital Asplenia: A French Nationwide Retrospective Survey of, 20 Cases. *J Pediatr*, 2011, 158: 142-148.