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Acute, complete splenic infarction in cancer patient is associated with a fatal outcome

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Abstract

Splenic infarction frequently occurs in patients with myeloproliferative diseases, endocarditis, and sickle cell anemia. Various sonographic patterns of splenic infarction do exist, but little is known about tumor associated splenic infarction in cancer patients. Between January 1992 and December 2002, 66 patients were diagnosed with splenic infarction by color Doppler sonography (CDS). Ten patients had an underlying solid cancer. Clinical and sonographic data of cancer patients were evaluated retrospectively with regard to age, sex, frequency of thrombotic episodes, splenic size, echomorphology and vascularity of splenic lesions, and follow-up examination. The median age was 53 years (range, 16–73 years). Nine of 10 patients had abdominal metastases, four had evidence of a hypercoagulable state, five had a small spleen ($< 7 \times 3$ cm), and seven had acute complete infarction of the spleen without hilar and parenchymal vessels on CDS. Survival of six patients with acute complete infarction ranged from 1 to 30 days. In cancer patients with splenic infarction, an acute complete infarction is the most common pattern. It is caused predominantly by a hypercoagulable state and is associated with an extremely short survival.

Key words: Cancer—Splenic infarction—Ultrasound

Patients with cancer are frequently in a hypercoagulable state. The most common thrombotic episodes are migratory superficial thrombophlebitis, venous thrombosis marantic endocarditis, disseminated intravascular coagulation, and thrombotic microangiopathy [1]. In contrast, tumor-associated arterial thrombosis is much less common in cancer patients.

Splenic infarctions frequently occur in patients with myeloproliferative disorders, endocarditis, and sickle cell anemia [2, 3]. The striking clinical feature of splenic infarction is sudden onset of pain in the left upper abdomen; however, clinical diagnosis can be difficult because infarcts may be silent [4].

Ultrasound enables evaluation of splenic tissue texture and identification of focal and diffuse abnormalities [5]. Splenic infarcts can be visualized at ultrasound and color Doppler sonography (CDS) because the echographic texture and vascular flow pattern on CDS is different from that of the surrounding normal tissue [6, 7]. The aim of this report is to describe clinical data, sonographic patterns, and prognosis of cancer patients with splenic infarction.

Patients and methods

Between January 1992 and December 2002, 119,000 patients were documented by abdominal ultrasound at the department of sonography in a comprehensive medical center. Sixty-six patients received a final diagnosis of splenic infarction. Ten patients had an underlying solid cancer. Clinical and sonographic data of these cancer patients were evaluated retrospectively in a single-center study.

Male to female ratio was 6 to 4. The median age was 53 years, with a range between 16 and 73 years. The underlying cancer was histologically confirmed and included adenocarcinoma of the pancreas ($n = 3$) adenocarcinoma of the lung ($n = 2$), adenocarcinoma of unknown primary origin ($n = 2$), adenocarcinoma of the colon ($n = 1$), hypernephroma ($n = 1$), and adenocarcinoma of the bile duct ($n = 1$).

The following clinical data were obtained: evidence of metastatic disease, evidence of sepsis, evidence of migratory superficial thrombophlebitis, evidence of idiopathic venous thrombosis, evidence of non-bacterial thrombotic endocarditis, evidence of disseminated intravascular coagulation, evidence of thrombotic microangiopathy, and evidence of arterial thrombosis other than splenic infarction.

In all patients, the indication for abdominal ultrasound was tumor staging. None of the patients had left upper abdominal pain. The following sonographic data were determined: splenic size, echomorphology of splenic tissue, and echomorphology of splenic lesions.

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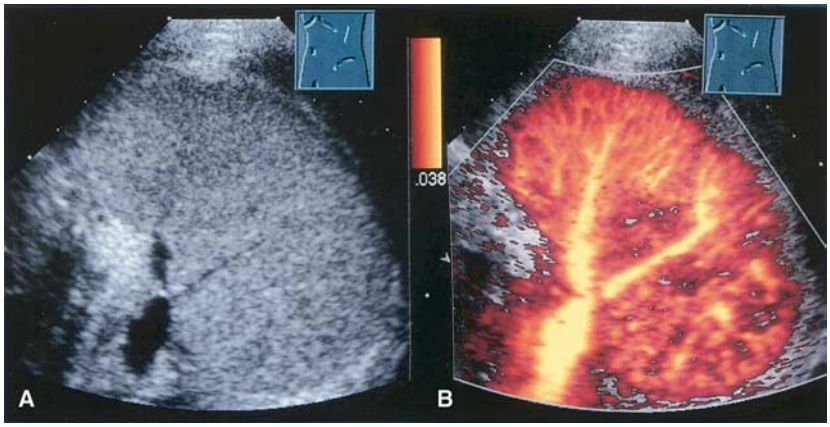

Fig. 1. **A** Normal splenic echomorphology is characterized by a homogeneous parenchyma. **B** On CDS normal splenic vascularity is characterized by treelike branches.

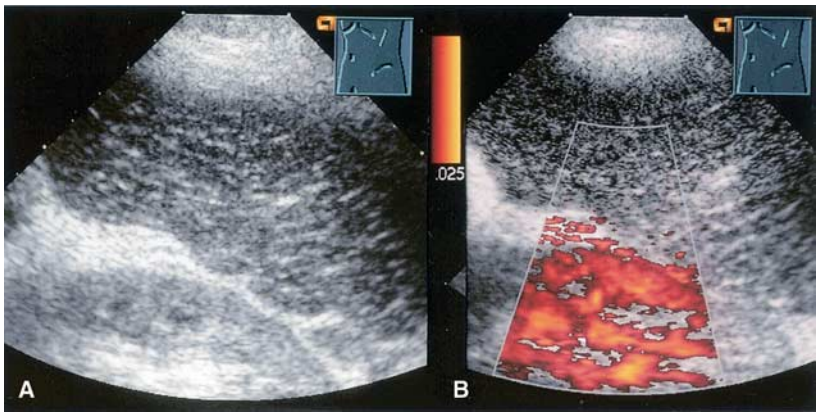

Fig. 2. **A** Patient 7 shows a hypoechoic and inhomogeneous splenic parenchyma 1 day after abdominal surgery. **B** On CDS no hilar and parenchymal vascularity is seen in contrast to visualization of regular kidney vessels.

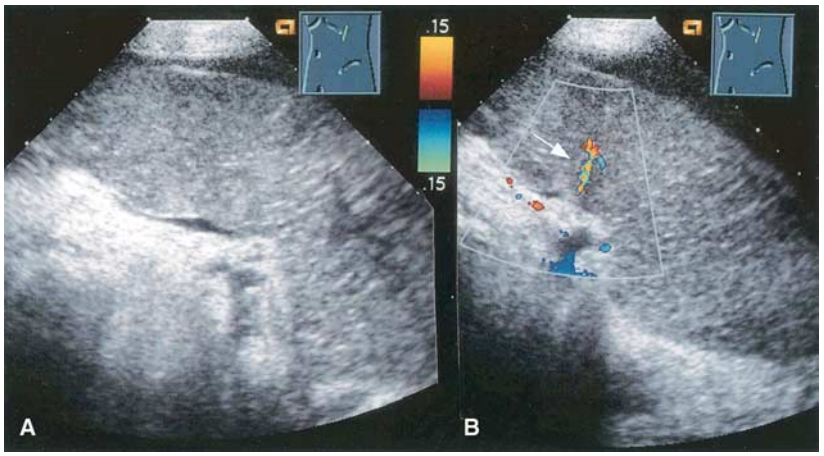

Fig. 3. **A** Patient 4 shows a hypoechoic and inhomogeneous splenic parenchyma. **B** On CDS a small nutritive vessel (*arrow*) is seen without evidence of regular hilar and parenchymal vascularities.

1. Splenic size. The size of the spleen was estimated with the patient in the right lateral position to scan the maximum length of the spleen and the width was measured between the hilus and the cortex. Splenic size was classified as follows: small spleen ($< 3 \times 8$ cm), normal-size spleen ($< 5 \times 11$ cm), moderate splenomegaly (5×11 to 8×20 cm), and severe splenomegaly ($> 8 \times 20$ cm).
2. Echomorphology of splenic tissue texture. Normal hepatic echomorphology was used as an internal standard, and splenic tissue was classified as hypoechoic or isoechoic and as homogeneous or inhomogeneous. Normal splenic vascularity was characterized by treelike branches on CDS (Fig. 1). CDS evaluation of splenic tissue was performed in all patients and classified as absent or present hilar and parenchymal vascularities.
3. Echomorphology of splenic lesions. Healthy splenic tissue was used as an in vivo reference, and focal lesions were classified as hypoechoic, isoechoic, or hyperechoic.

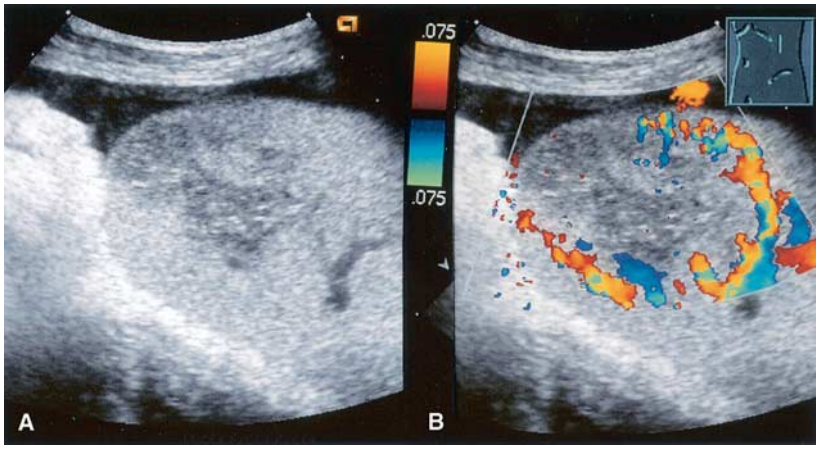

Fig. 4. **A** Patient 10 shows a focal, wedge-shaped, hypoechoic, irregular, delineated lesion. **B** On CDS no vascularity is seen within the infarcted area.

Table 1. Clinical and sonographic data in 10 patients with solid cancers and splenic infarction

Patient number, age (years), sex	Diagnosis	Metastases	Splenic size	Splenic infarct	CDS	Time to death after diagnosis of splenic infarct
1, 73, M	Hypernephroma	Peritoneal	SP	Complete	No hilar and no parenchymal vessels	2 days
2, 60, M	Colon cancer	Peritoneal, liver	MS	Complete	No hilar and no parenchymal vessels	1 day
3, 47, M	CUP	Peritoneal, liver	SP	Complete	No hilar and no parenchymal vessels	10 days
4, 54, M (Fig. 3)	CUP	Peritoneal liver	MS	Complete	No hilar and no parenchymal vessels	3 days
5, 73, F	Bile duct cancer	Peritoneal	SP	Complete	No hilar and no parenchymal vessels	1 day
6, 70, F	Pancreatic cancer	Peritoneal, liver	SP	Complete	No hilar and no parenchymal vessels	30 days
7, 16, F (Fig. 2)	Pancreatic cancer	Peritoneal, liver	SP	Complete	No hilar and no parenchymal vessels	Still alive at 8 months
8, 43, M	NSCLC	Liver, adrenal, bone	MS	Focal	Hilar vessels, infarct area without vessels	6 months
9, 47, F	NSCLC		MS	Focal	Hilar vessels, infarct area without vessels	24 months
10, 45, M (Fig. 4)	NET of the pancreas	Liver, lung, bone	MS	Focal	Hilar vessels, infarct area without vessels	Still alive at 10 months

CDS, color Dopper sonography; CUP, carcinoma of unknown primary cause; F, female; M, male; MS, moderate splenomegaly; NET, neuroendocrine tumor; NSCLC, non-small cell lung cancer; SSP, small spleen

In addition, the number (solitary or multiple), configuration (wedge shape or round), margination (smooth or irregular), and maximum diameter of splenic infarcts were determined.

Clinical and sonographic follow-up was performed in all patients.

Sonographic studies were performed with sector 3.5- and 5-MHz transducers (Picker LSC 7000, Highland Heights, OH, USA; Acuson 128, Mountain View, CA, USA; Sequoia Acuson, Mountain View, CA, USA). The color Doppler velocity scale and level were adjusted to show the slowest blood flow.

Results

Clinical and sonographic data are shown in Table 1. Evidence for a hypercoagulable state included: metastatic disease (n = 9), sepsis (n = 1, patient 5), migratory superficial thrombophlebitis (n = 1, patient 4); venous thrombosis (n = 3, patients 3, 4, and 6), disseminated intravascular coagulation (n = 1, patient 5), and thrombotic microangiopathy (n = 1, patient 3). No patient had evidence of non-bacterial thrombotic endocarditis or arterial thrombosis other than splenic infarction. The splenic vein was patent in all cases without evidence of peripancreatic tumor masses.

Splenic size

In patients with an acute complete splenic infarction (n = 7), a small spleen was found in five cases. All three patients with a focal splenic infarction had a moderate splenomegaly.

Echomorphology of splenic tissue texture

Patients 1 to 7 had a hypoechoic, inhomogeneous splenic tissue texture with absent hilar and parenchymal vessels (Fig. 2). Small nutritive vessels due to short gastric arteries were seen in patients 1, 2, 4, and 5 (Fig. 3). Patients 8 to 10 had an isoechoic, homogeneous splenic tissue texture with

regular hilar and parenchymal vessels in the noninfarcted area (Fig. 4).

Echomorphology of splenic lesions

Patients 8 to 10 had focal hypoechoic lesions that were solitary (patients 8 and 9) or multiple (patient 10). All lesions were wedge shaped and irregularly delineated. The maximum diameters were 4 cm (patient 8), 6 cm (patient 9), and 5 cm (patient 10).

Follow-up

In patient 7, acute complete splenic infarction was diagnosed 1 day after pancreatic surgery; for this reason, splenic infarction most probably was due to surgical complication. In patients 1 to 6 acute complete splenic infarction developed spontaneously in end-stage disease.

Survival of patients 1 to 6 with acute complete infarction ranged from 1 to 30 days. Follow-ups after diagnosis of splenic infarction were 6 months in patient 7 and 12 months in patient 10 (Table 1).

Discussion

Compared with lesions of the liver, focal and diffuse lesions of the spleen are uncommon in patients with solid cancers. Splenic metastases were found in autopsy studies with a frequency of 0.6% [8] and were sonographically characterized by round intrasplenic lesions of various echomorphologies [9]. A sonographic pattern of diffuse splenic involvement has been described for patients with malignant lymphoma but not for cancer patients with splenic metastases [10].

The acute focal splenic infarct is frequently associated with myeloproliferative diseases and endocarditis and appear as a focal wedge-shape, hypoechoic, well-demarcated lesions at sonography [4, 6]. On CDS acute focal splenic infarction is characterized by absent flow signals within the infarcted area [7]. Splenomegaly is a usual concomitant of focal infarction. There is a high self-healing tendency in acute splenic infarction [4].

Chronic, recurring splenic infarction is commonly associated with sickle cell anemia and leads to a functional hyposplenia or asplenia. The spleen is generally small, with a complex, predominantly hyperechoic texture of the parenchyma on ultrasound [9]. On CDS chronic splenic infarction is characterized by absent or reduced flow signals within the infarcted area [9].

The acute infarction of the entire spleen is an extremely rare event and has been described in case reports [11, 12]. It is related to splenic artery occlusion, which is principally caused by torsion, tumor invasion, compression, thrombosis, or thromboembolism. In acute complete infarction, the spleen is small, with a diffuse inhomogenity and a hypoechoic texture on ultrasound. On CDS acute complete splenic infarction is characterized by absent flow signals within the entire spleen [11, 12].

Similar to diffuse liver disease, diffuse splenic pathology is difficult to assess [13]. It should be mentioned that there is no histologic confirmation of sonographically diagnosed acute complete splenic infarction. In our series, the diagnosis was made predominantly on the basis of CDS examination. In noncancer patients with splenic infarction, an acute, complete infarction is an extremly rare event [4]. As shown on our results, there was a strong association between complete splenic infarction with metastatic disease and a fatal outcome. Four (3, 4, 5, and 6) of our seven patients with complete infarction had some evidence of a hypercoagulable state.

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