FOLLOW-UP AFTER SPLENIC EMBOLIZATION IN PATIENTS WITH BLUNT SPLENIC INJURY: CAN CONTRAST-ENHANCED ULTRASOUND REPLACE CT?

PRELIMINARY RESULTS

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OBJECTIVE: To outline the contrast-enhanced sonographic appearances of traumatic lesions after splenic embolization and to compare these findings with CT and clinical outcome.

PATIENTS AND METHODS

From September 2003 to March 2004 in 16 patients 22 contrast-enhanced ultrasound (CEUS) examinations were carried out prospectively 0 to 408 days after splenic embolization and compared to computer tomography (CT) and clinical findings. Depending on the time interval after embolization the patients were classified into an early follow-up group and a medium and late follow-up group. After baseline ultrasound, a volume of 2.4 – 4.8 ml of a sulfur-hexafluoride-based microbubble contrast medium (Sonovue®) was administrated intravenously in all patients. Examination was performed with an Acuson Sequoia 512 ultrasound unit, a 2-4 MHz convex array probe, and the Cadence CPS software for low-mechanical index imaging. CT scans were carried out with 5.0 mm collimation, a table speed of 15 mm/rotation, slice thickness of 3.75 mm and a reconstruction interval of 2.5 mm delay. Perisplenic fluid, subcapsular and intraparenchymal hypoechoic areas, hyperechoic intraparenchymal spots and contrast medium pooling were registered. In cases of serial examinations, regression and progression of different lesions were considered. The results of ultrasound were compared to CT in form of agreement in detection rates. In group 2 without parallel CT and ultrasound examination, a simple clinical oral questionnaire was performed previous to the ultrasound study.

RESULTS

Group 1 (six patients 11 CEUS): Five patients were studied within three days and one patient eight days after embolization. There was an almost 100% agreement in the detection rates between the CT and CEUS examinations: Three patients showed perisplenic fluid, four patients had subcapsular clots, four patients developed post-embolization infarctions, two of them with intraparenchymal air. No pseudoaneurysm, no extravasation were seen. In one patient, a small amount of perisplenic fluid was undetected with CEUS, but clearly shown with unenhanced ultrasound.

In four patients serial ultrasound and CT was performed. Two patients were studied within one week after the primary studies. One of these patients had unchanged perisplenic fluid but showed clearly regression of subcapsular and intracapsular hematomas. The other patient showed increasing subcapsular hematoma and intrasplenic air on CT and CEUS and had also clinical findings of ongoing hemorrhage. This patient underwent a second embolization with selective coiling of an upper pole artery. Embolization was technically successful, but the patient developed clinical symptoms of infection. He was drained percutanously, but there was no evidence of splenic abscess. The patient was treated with antibiotics; he recovered and was transferred to the local hospital.

Two additional patients and one of the former mentioned patients underwent late follow-up between 9-12 weeks after embolization. Both modalities showed regression of intrasplenic lesions in all three patients, regression of subcapsular clots in two of them, disappearance of infarctions and of perisplenic fluid in one. In one patient with only peripheral coiling intrasplenic air had disappeared 9 weeks after embolization. On CEUS the small coils in the parenchyma were misinterpreted as intrasplenic gas in regression.

Group 2 (ten patients, 11 CEUS examinations):

In this group the CEUS was primary compared with the results of the questionnaire prior to the examination. In 9 cases, CEUS was performed 6-15 months (late follow-up), and in two cases two and three months (medium follow-up) after embolization. No patient complained of pain or other sequester after splenic trauma. Scars were seen in four patients of the late-follow-up subgroup and in one patient of the medium follow-up subgroup. In two of these

cases CEUS could not clearly differentiate between a small cystic lesion or scar, but the unenhanced examination showed marked hyperechoic changes, consistent with fibrotic tissue. Splenic size was at the lower limit or reduced (< 10 cm diameter) in five cases in the late follow-up group and in one in the medium follow-up group. One patient suffered from an almost unechoic lesion interpreted as a chronic hematoma in regression compared to prior CT.

A hypoperfusion area was detected in one patient 3 month after embolization consistent with traumatic infarction in regression. No perisplenic fluid or vascular complications were seen.

CONCLUSIONS

CEUS is a promising tool in the follow-up examination after splenic embolization to control the development of intrasplenic lesions and hypoperfusion areas. All complications of clinical relevance were detected both with CT and CEUS. Unenhanced ultrasound delineates perisplenic fluid slightly better than CEUS and helps in the late follow-up examination to differentiate between scar and remnants of intrasplenic hematomas.