CLINICAL IMAGE

Hypointense Spleen Associated with Severe Acute Pancreatitis on MR Imaging

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Acute pancreatitis is an acute-onset pancreatic or peripancreatic inflammatory disease caused by gall stones, alcohol addiction, fatty diet, viral infection, and so on. In severe acute pancreatitis, systemic inflammatory response syndrome can occur, which is associated with renal impairment, intraperitoneal abscess, sepsis, and multiorgan failure. Recently, splenic density on CT is reported to decrease in patients with severe acute pancreatitis.¹ This CT finding may be induced by splenocyte reduction, which is a compensatory reaction to peripheral lymphocyte reduction, damaged B-lymphocytes, and fibroblast infiltration in the spleen.^{1,2} The spleen shows hypointensity in the neonate on the T_1 - and T_2 -weighted MR images, whereas the signal intensity markedly increases by 8 months of age on the T₂-weighted images in proportion to the development of white pulp and maturation of lymphocytic systems.³ Thus, the splenic signal intensity may change if the lymphocytic systems within or outside the spleen are affected by severe acute pancreatitis.

A 66-year-old obese man presented with severe abdominal pain about 2 hours after eating ramen noodles. He had a history of repeated pancreatitis, gall stones, and hepatic abscess, but no hematologic diseases, blood transfusions, granulomatous diseases, or amyloidosis.

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Laboratory examinations (e.g., serum amylase, 991 U/L; urine amylase, 5435 U/L) and CT made a diagnosis of acute pancreatitis at the grade 2. The spleen showed normal enhancement on CT. Abdominal MR imaging study was performed on a 3T 2 days after the onset. The spleen showed hypointensity on all of the breath-hold T₁-weighted imaging (TR, 256 ms; TE, 2.3, 3.5, 4.6 ms) and non-fatsuppressed and fat-suppressed T₂-weighted imaging (TR, 4745 and 6725 ms; TE, 90 ms) and diffusion-weighted imaging (TR, infinite; TE, 70 ms, b value = $600 \text{ mm}^2/\text{s}$) (Fig. 1), especially on the fat-suppressed T₂-weighted and diffusion-weighted imaging (Fig. 1E and 1F). The size of the spleen appeared normal. It took 7 weeks to normalize amylase and CT with intensive care (e.g. gabexate mesylate, meropenem hydrate). Among our 24 cases of acute pancreatitis who underwent MR imaging, the present case was one of the two most serious.

We have demonstrated hypointense spleen associated with severe acute pancreatitis on MR imaging. Hemosiderosis and amyloidosis also leads to hypointense spleen, but the clinical backgrounds, laboratory data, and contrastenhanced CT and MR imaging of the liver, bone marrow, and intestines can exclude these particular diseases. To our knowledge, this is the first report to indicate the utility of MR imaging for evaluating the inflammatory response syndrome of the spleen in acute pancreatitis. Further investigation is required to confirm the significance of hypointense spleen associated with severe acute pancreatitis on MR imaging.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Fig. 1 In-phase (**A**), out-of-phase (**B**), and the second in-phase (**C**) T_1 -weighted imaging, non-fat-suppressed (**D**) and fat-suppressed T_2 -weighted imaging (**E**), and diffusion-weighted imaging (**F**) demonstrate hypointense spleen. Especially, fat-suppressed T_2 -weighted imaging (**E**) and diffusion-weighted imaging (**F**) shows inflammatory lesions (arrows) and hypointense spleen clearly (arrowheads).

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