Case series: Gamna-Gandy bodies of the spleen: a supportive finding for portal hypertension

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Gamna-Gandy bodies (GGB) of the spleen, which are mainly found in patients with portal hypertension, represent the end-result of hemorrhages in the splenic follicles or adjacent trabeculae.[1] It is known that GGB contain hemosiderin, fibrous tissue and calcium.[2] Although ultrasonography (USG) and CT scan have been shown to be useful modalities for the detection of these lesions, MRI has been regarded to be a very sensitive imaging modality for depicting these nodules, due to their iron content.[3] We would like to report three cases diagnosed to have portal hypertension due to liver cirrhosis, in whom MRI revealed GGB in their enlarged spleens.

Case Report

Three patients with a known history of liver cirrhosis were referred for MRI. They were two men, aged 20 and 60 and one woman, who was 30 years old. All patients presented with portal hypertension findings and had a history of cryptogenic liver cirrhosis. Their laboratory tests demonstrated abnormal liver function.

MRI was performed using a 1.5 Tesla superconducting magnet (GE, Signa, Milwaukee, Wisconsin, USA) with a phased- array body coil. The imaging protocol included T1W axial and coronal breath-hold fast-spoiled, gradient-echo (FSPGR / 80) (TR: 185 msec, TE: 2.3 msec), T2W axial fast spin-echo (FSE) with fat saturation (TR: 5454 msec, TE: 104 msec) sequences and post-contrast axial and coronal breath-hold T1W FSPGR / 80 (TR: 185 msec, TE: 2.3 m) sequences after intravenous injection of 0.1 mmol / kg gadolinium.

These images demonstrated characteristic findings of liver cirrhosis and also revealed an enlarged spleen due to portal hypertension. Furthermore, all images showed multiple, tiny, low signal intensity, sharply delineated nodules measuring only a few millimeters in diameter, scattered throughout the spleen [Figure 1]. Although these small nodules were distributed diffusely in the spleen parenchyma, they were most conspicuous at the periphery. T1W gradient-echo images were superior to T2W FSE images for detecting these lesions, which showed no enhancement though they were more conspicuous on the post-contrast images [Figure 2]. In addition, due to their iron content that causes a blooming effect on gradient-echo images, they were more apparent on these sequences. Thus, they were regarded as siderotic nodules, the so- called GGB.
Discussion

Portal hypertension leads to splenomegaly with hyperplasia of the cells of the reticulo-endothelial system, which cover the sinusoids. Prolonged transit time of the blood and pressure increase disintegration of cells, leading to bleeding into the red pulp with deposition of siderin adjacent to the thickened collagen tissue, forming the so-called GGB. Therefore, GGB contain fibrous tissue, hemosiderin and calcium.

These nodules show signal voids on all pulse sequences, due to their hemosiderin content. Consequently, gradient-echo sequences are considered the most sensitive for their detection. The lesions vary in size, but generally have a diameter ranging from a few millimeters up to at the most 1 cm. Lesion depiction improves on post-gadolinium-contrast images, because the spleen enhances and leads to increased signal-to-noise, therefore improving GGB detection.

Minami et al retrospectively evaluated the MRI examinations of 233 patients with portal hypertension and found siderotic nodules in 21 of them. Yasuhara et al detected splenic nodules in 8 of 62 patients in a post-mortem histologic study.

Unenhanced CT may detect GGB as multiple faint high-attenuation spots in the spleen. These spots represent calcification in siderotic nodules; CT however is far inferior to MRI in detecting GGB.

USG features of GGB include multiple, punctate, hyperechoic foci. Chan et al found the sensitivity of USG to be 70.6%, the specificity 78.9% and the positive predictive value to be 85.7%, for the diagnosis of GGB. The detection of punctate hyperechoic foci on USG in the clinical setting of cirrhosis is sufficient for the diagnosis of splenic siderosis complicating portal hypertension.

Although GGB are the end-result of hemorrhages in the spleen caused by portal hypertension, they are not specific for portal hypertension. They are also seen in patients with portal vein or splenic vein thrombosis, hemolytic anemia, leukemia or lymphoma, acquired hemochromatosis, paroxysmal nocturnal hemoglobinuria and patients receiving blood transfusions, although they are rare findings in these diseases.

In conclusion, we have discussed the MRI appearances of GGB, which are seen in some patients with portal hypertension.

References


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