Hepatic hemangiomas and other hemangiomatous lesions: MR imaging manifestations, pitfalls and problem-solving MR techniques

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[Purpose] Hepatic hemangiomas and other hemangiomatous tumors and tumor-like lesions may show characteristic clinical and MR imaging manifestations reflecting their pathologic features. Various degeneration in hemangiomas, and surrounding parenchymal changes may influence the imaging manifestations of hemangiomas. In this exhibit we demonstrate usual, unusual MR imaging manifestations of hepatic hemangiomas and other hemangiomatous lesions, pitfalls and problem-solving MR techniques.

[Outline of Content] Various hemangiomas and other hemangiomatous lesions such as cavernous and sclerosed hemangiomas, solitary necrotic nodule as the end-stage of sclerosed hemangiomas, small high-flow hemangiomas, giant hemangiomas, pedunculated hemangiomas, hemangiomatosis, angiomylipomas, angiosarcomas, and peliosis hepatis may show characteristic clinical and MR imaging manifestations reflecting their pathologic features. Various degeneration such as hyalinization, cystic formation, fibrosis, calcification and thrombosis, and surrounding parenchymal changes such as co-existing fatty infiltration of the liver with peri-tumoral focal spared areas, peripheral parenchymal retraction, and arterial-portal venous shunts may influence the imaging manifestations. Problem-solving MR techniques such as chemical shift imaging (CSI), dynamic contrast-enhanced MRI (DCE-MRI), diffusion-weighted imaging (DWI), SPIO-MRI and Gd-EOB-MRI for the diagnosis of problematic cases are reviewed.

[Summary] To recognize various imaging manifestations of hemangiomas and other hemangiomatous lesions, and making accurate diagnosis by using problem-solving MR techniques are important for appropriate management of the patients.

Fig. 1: DCE-MRI of typical hemangioma: Characteristic early nodular CE, and prolonged CE on delayed phase due to pooling of Gd.

Fig. 2: EOB-DCE-MRI of typical hemangioma: No persisting CE on delayed phase (pseudo washout) and no EOB uptake on hepatobiliary phase.

Fig. 3: Characteristic signal increase on SPIO-T1WI due to the pooling of SPIO particles with T1-shortening paramagnetic effect.

Fig. 4: Small hemangioma may be clearly demonstrated on DWI.

Fig. 5: Giant hemangioma may cause cystic degeneration.

Fig. 6. Giant hemangioma may also cause hyalinized degeneration. Even a huge mass appears without bulging of liver contour.

Fig. 7: Sclerosed hemangioma contains T2-high hyalinization and T2-low fibrosis, and does not show typical CE pattern of usual hemangioma.

Fig. 8: Solitary necrotic nodule may be the end-stage of sclerosed hemangioma. Early ring CE may mimic metastatic liver tumor, however low intensity on T2WI/DWI may suggest its benignity.

Fig. 9: Peri-tumoral focal spared area in fatty liver may cause complex CE appearance of hemangioma mimicking malignancy. Opposed-phase image of chemical shift imaging (CSI) can reveal the focal spared area clearly.

Fig. 10: CSI is also helpful in diagnosing hepatic angiomylipoma by revealing the fatty components in the tumor.

Fig. 11: Epithelioid angiomylipoma is fat scanty mass and CSI cannot reveal fat. Marked hypervascularity with AP shunt may be suggestive finding.

Fig. 12: Angiosarcoma may show hemangioma-like CE pattern, however, bulging of liver contour and heterogeneous signal intensity with intratumoral hemorrhage on MRI are suggestive for its malignant nature.

Fig. 13: Peliosis hepatis is caused by extensive dilated sinusoid in the liver parenchyma as hemangioma-like appearance, and vessels may be involved in the peliotic area suggesting its non-neoplastic nature.