Inflammatory Liver Diseases

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Contents

Abstract

Liver inflammation may be acute or chronic form which leads to fibrosis and cirrhosis. The diagnosis depends on histopathologic evaluation which can be confounding especially in cases with heterogeneous involvement. In this chapter, we reviewed inflammatory liver diseases such as autoimmune hepatitis, overlap syndromes, sarcoidosis, drug disease, Wilson's disease, alfa 1 antitripsin deficiency, and radiation injury with imaging findings.

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Several etiological factors cause liver inflammation that is characterized by acute or chronic inflammatory cell accumulation within the liver. Liver inflammation may be in acute or chronic form that leads to fibrosis and cirrhosis. The diagnosis of liver inflammation primarily depends on liver biopsy and histopathologic evaluation. However, imaging findings may guide both clinician and pathologist especially in cases with confounding laboratory or pathologic findings. In this chapter, we review inflammatory liver diseases such as autoimmune hepatitis, overlap syndromes, sarcoidosis, drug disease, Wilson's disease, alpha-1 antitrypsin deficiency, and radiation injury with imaging findings.

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1 Autoimmune Hepatitis

Autoimmune hepatitis (AIH) is a chronic liver disease that is seen in all ages and races with a female predominance (Manns et al. 2015). Genetic susceptibility with an exposure to a trigger environmental agent such as herbs, microbes, immunization, and drugs is a blamed factor in the etiopathogenesis of AIH. In general, AIH present with an episode of acute hepatitis with subsequent chronic course. Clinical manifestation of AIH may vary such as mildly elevated liver function tests in asymptomatic patient, acute fulminant hepatitis, or cirrhosis. The diagnosis of the disease primarily depends on clinical, laboratory, and liver histology findings. Interface hepatitis with lymphoplasmocytic infiltrates and varying degrees of lobular inflammation is a histopathologic finding in AIH (Manns et al. 2015).

Imaging findings of AIH may change according to the severity of the disease. Ultrasonography (US) may reveal no abnormal finding in the early stage of the disease as well as features of chronic liver disease such as coarsening of hepatic echotexture, nodularity, and volume redistribution (Malik and Venkatesh 2017). The main role of US in AIH is HCC surveillance. Transient US elastography was shown to be a reliable method for estimation of hepatic fibrosis in AIH (Obara et al. 2008). Computed tomography may reveal normal findings or findings associated with chronic liver disease. Benign hypervascular nodules can be observed in AIH that may enhance after contrast administration, some with delayed washout (Qayyum et al. 2004). Nonspecific findings such as hepatic surface nodularity, ascites, and splenomegaly were found to be the most common findings on CT (Sahni et al. 2010).

Magnetic resonance imaging (MRI) may demonstrate similar findings as CT. Hepatic surface nodularity that is associated with fibrosis is the most common finding observed in AIH on MRI (Bilaj et al. 2005; Sahni et al. 2010) (Fig. 1). Reticular fibrosis that is defined as fine lines that had low signal intensity at out-of-phase MR images with prominent enhancement on post-contrast images is another finding observed on MRI (Bilaj et al. 2005). Global atrophy was the main volumetric change observed in AIH (Obara et al. 2008). Lymphadenopathy is not common as other autoimmune liver diseases such as primary sclerosing cholangitis (PSC) and primary biliary cholangitis (PBC). MR elastography is a promising method in the evaluation of fibrosis in patients with AIH. It was shown that a liver stiffness threshold of >4.1 kPa predicted advanced fibrosis with 89.5% sensitivity and 100% specificity, and a threshold of >4.5 kPa predicted cirrhosis with 92% sensitivity and 96% specificity (Wang et al. 2017).

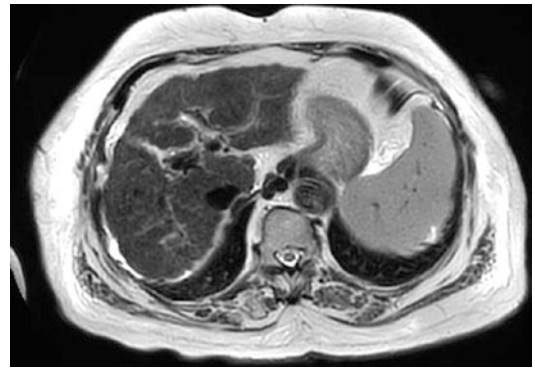

Fig. 1 A 70-year-old female patient with autoimmune hepatitis. T2W imaging demonstrates hepatic surface nodularity, which is the most common imaging finding

1.1 Overlap Syndromes

Overlap syndromes are variant forms of autoimmune liver diseases, which share histological, biochemical, serological, and imaging characteristics of AIH, primary biliary cholangitis (PBC), and primary sclerosing cholangitis (PSC). It was reported that 7–13% of patients with AIH have overlapping features of PBC and 6–11% of patients have features of PSC (Czaja 2013). Biochemical and imaging findings of overlap syndromes may vary according to the difference of coexistence disease.

AIH-PBC overlap syndrome presents with serum liver tests typically showing a hepatitic pattern in AIH and a cholestatic pattern with predominant elevation of alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT)

and only mild elevation of serum transaminases in PBC. Primary biliary cirrhosis is a slow progressive disease with destruction of small intrahepatic bile ducts ending up with chronic cholestasis and cirrhosis. Imaging findings of PBC include heterogeneous attenuating parenchyma and segmental atrophy on CT (Blachar et al. 2001). Atrophy or hypertrophy may be observed as well as smooth or nodular liver contour (Blachar et al. 2001). Findings of portal hypertension are commonly seen in advanced PBC. Lymphadenopathy is an expected finding in PBC with most commonly in portacaval region and porta hepatis (Blachar et al. 2001). Conspicuous low signal intensity around portal vein branches on T1 and T2 images was defined for MRI finding of PBC (Wenzel et al. 2001). "Periportal halo sign" was described as a finding of PBC consisting of a rounded lesion centered on a portal venous branch 5 mm–1 cm in size, and numerous lesions involving all hepatic segments, with low signal intensity on T1- and T2-weighted images, and no mass effect (Fig. 2) (Wenzel et al. 2001). In AIH-PBC overlap, imaging findings may be consistent with either isolated AIH or isolated PBC (Hyslop et al. 2010).

AIH-PSC overlap syndrome is diagnosed with characteristic changes in biliary tree observed in imaging such as strictures and segmental dilatations in a patient with AIH. PSC is a chronic inflammatory large duct cholangiopathy, which results with fibrotic biliary duct strictures, cholestasis, and biliary cirrhosis. Diffuse involvement of both intrahepatic and extrahepatic bile ducts is the commonest form (75%) followed by isolated intrahepatic bile duct involvement (15%) and isolated extrahepatic bile duct involvement (10%) (Tischendorf et al. 2007). Central lobe enlargement with left lateral hypertrophy predominates in PSC (Bilaj et al. 2005). Imaging findings in AIH-PSC overlap syndrome on MRI include central macroregenerative nodules, peripheral atrophy, biliary ductal obstruction, and biliary ductal beading (Hyslop et al. 2010). The presence of macroregenerative nodules, peripheral atrophy, and biliary ductal irregularity, alone or in combination, had 100% specificity for PSC-type overlap syndrome.

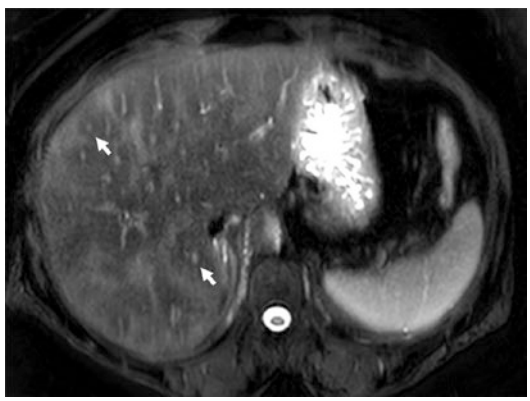

Fig. 2 A 48-year-old female patient with overlap syndrome (autoimmune hepatitis—primary biliary cholangitis). T2W images show periportal halo sign (arrows)

2 Sarcoidosis

Sarcoidosis is a multisystem disease characterized with noncaseating granulomas in affected organs. The prevalence of the disease is 2–60 per 100,000 people with a predominance in African American populations (Baughman and Lower 2008). The etiopathogenesis of the disease remains unclear with blamed genetic susceptibility and environmental factors. Liver is one of the most affected organs after lung and lymph node involvement. The disease may involve all compartments of the liver and results in various patterns of liver injury such as portal and lobular inflammation of hepatocytes, biliary involvement with cholestasis, or sinusoidal dilatation as a result of compressive effect of granulomas (Deutsch-Link et al. 2018). The majority of patients with hepatic involvement are asymptomatic. Clinical symptoms include fatigue, abdominal pain, and fever (Tadros et al. 2013). The diagnosis generally depends on clinical and radiological findings with histopathological sarcoid granuloma identification.

Imaging findings are not evident in most of the cases. The most common imaging feature of hepatic sarcoidosis is hepatomegaly that is seen in more than 50% of the patients (Warshauer et al. 1995). Increased parenchymal echogenicity

may be observed in US examination. Heterogeneous echogenicity and coarse echo pattern may be detected in diffuse parenchymal involvement (Karaosmanoğlu et al. 2015). Contour irregularity and parenchymal calcifications are also expected findings in diffuse hepatic involvement (Kessler et al. 1993). Focal lesions that are consistent with granulomas are detected as well-defined hypoechoic or hyperechoic nodules in the liver (Gezer et al. 2015).

Cross-sectional imaging findings may demonstrate similar findings with US. The appearance of the liver may range from normal to chronic parenchymal liver disease in diffuse hepatic sarcoidosis. It was reported that both macroregenerative nodules and wedge-shaped peripheral atrophy presence may suggest the diagnosis of hepatic sarcoidosis in a patient with chronic parenchymal liver disease (Ferreira et al. 2013). Periportal thickening that is best seen on T2-weighted images can be observed in sarcoidosis. Imaging findings consistent with portal hypertension can be seen as a result of cirrhosis or compression effect of portal lymphadenopathies. Focal nodules in hepatic sarcoidosis are typically seen as hypo-attenuating subcentimeter focal lesions on CT (Fig. 3). On MR imaging, these are seen as T1 hypo-isointense and T2 hypointense foci with no evident contrast enhancement (Fig. 4).

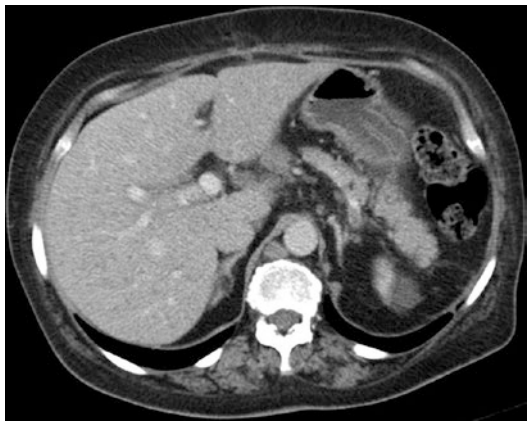

Fig. 3 A 54-year-old female patient with diagnosis of sarcoidosis. On contrast-enhanced CT images, multiple millimetric hypoattenuating nodules are seen

3 Drug Disease

Drug-induced liver injury (DILI) is an important cause of mortality around the world caused by various mechanisms causing liver injury. Hundreds of drugs have been identified causing DILI such as antibiotics (amoxicillin-clavulanate, isoniazid, doxycycline), diclofenac, acetaminophen, atorvastatin, and carbamazepine that are the commonest ones (Alempijevic et al. 2017). Besides, herbals and dietary supplements are used increasingly and cause increase in HDS-induced liver injury proportionally (Navarro et al.

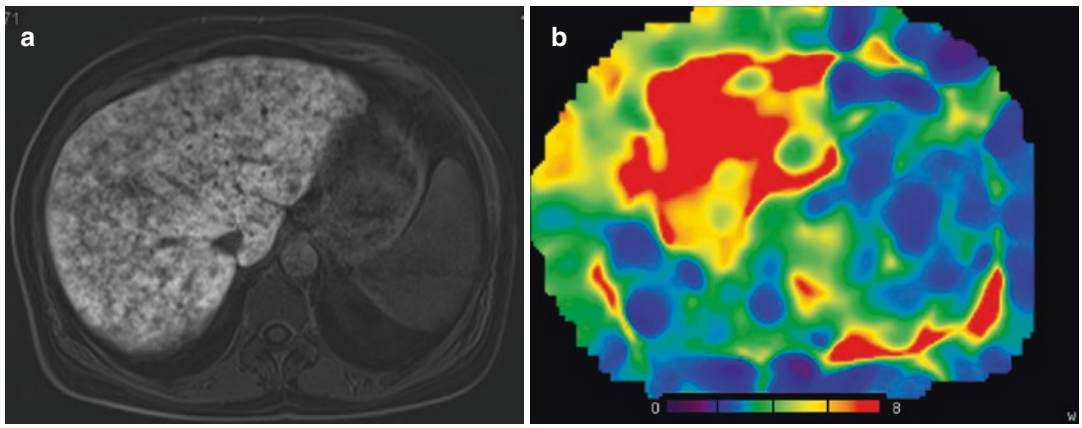

Fig. 4 A 47-year-old female patient with sarcoidosis. Postcontrast T1W MR images demonstrate heterogeneous enhancement of the liver (**a**). Liver stiffness was 9.9 kPa on MR elastography consistent with involvement (**b**)

2017). Acute and chronic hepatitic, acute and chronic cholestatic, and mixed hepatitis-cholestatic types are the most common forms of DILI within various histopathologic categories (Kleiner et al. 2014).

Imaging findings may vary according to the type of injury. Hepatic steatosis, which is defined as excessive triglyceride accumulation in the hepatocytes, is one of the possible mechanisms of DILI. Chemotherapeutic agents such as 5-fluorouracil, irinotecan, and methotrexate and amiodarone, tamoxifen, corticosteroids, and antipsychotics are frequently associated with steatosis or steatohepatitis (McGettigan et al. 2016). Increased echogenicity of the liver in US examination and decreased attenuation at CT are imaging findings of hepatic steatosis. Signal drop on out-of-phase image relative to in-phase image is present at MRI. Drugs such as immunosuppressive cyclosporine, the antibiotic trimethoprim-sulfamethoxazole, and the antipsychotic chlorpromazine cause cholestatic injury that predominantly affects bile ducts (Bhamidimarri and Schiff 2013; Padda et al. 2011). US and CT imaging have no specific finding in cholestatic injury and may demonstrate nonspecific findings of inflammation such as heterogeneous contrast enhancement or hepatomegaly (Fig. 5). Imaging modalities such as cholangiography and MRCP may demonstrate small bile ducts diminished in number (ElSayed et al. 2013; Gossard 2014, p. 1). Sclerosing cholangitis like changes such as intrahepatic and common hepatic duct strictures on MRCP was also defined in patients with DILI (Ahmad et al. 2019).

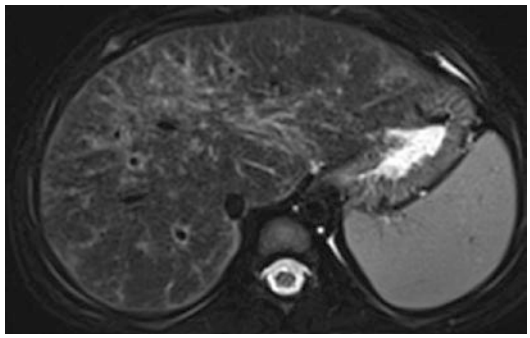

Fig. 5 Heterogeneous liver parenchyma and periportal thickening on T2W image are seen in a 2-year-old male patient with Wilms' tumor after chemotherapy. Follow-up MRI demonstrated resolution of the findings

4 Wilson's Disease

Wilson's disease is an autosomal recessively inherited disease with approximately 1/30,000 prevalence (Gitlin 2003). There is a defect in copper homeostasis which leads to accumulation of copper in different tissues such as liver, brain, and cornea. Liver is the main regulator of copper metabolism by storing both the copper and biliary excretion of this metal. There are different mutations reported in the ATP7G gene which give rise to dysfunction of the corresponding ATPase that transfers copper into the secretory pathway by incorporation into apoceruloplasmin and excretion into the bile (Lutsenko and Petris 2003; Schaefer and Gitlin 1999). This results in intrahepatocellular copper accumulation with resultant copper-mediated oxidative damage and activation of cell death pathways (Strand et al. 1998). Early diagnosis of Wilson's disease is important for complete recovery which might be fatal otherwise. Affected individuals mainly present with liver disease which may mimic different acute or chronic liver diseases. The presence of Kayser–Fleischer rings, decreased serum ceruloplasmin, and hepatic or neuropsychiatric symptoms are sufficient for the diagnosis of Wilson's disease.

There are different imaging findings of Wilson's disease according to the disease course. US is one of the most preferred imaging modalities for Wilson's disease. Increased hepatic echogenicity due to fatty change or fibrosis can be observed in individuals with Wilson's disease which is also the main US finding of nonalcoholic fatty liver disease and may be confusing (Akhan et al. 2009; Akpinar and Akhan 2007). Parenchymal heterogeneity was reported to be detected in most of the patients in Wilson's disease (Akhan et al. 2009). This heterogeneity can be diffuse or associated with multiple hypoechoic nodules or multiple hypoechoic and hyperechoic nodules (Akhan et al. 2009). Contour irregularity

and increased periportal thickness are other US findings seen in Wilson's disease. Perihepatic fat layer that is recognized by perihepatic hyperechogenic zone in US is a possible finding in Wilson's disease (Akhan et al. 2002, 2009; Akpinar and Akhan 2007). Cholelithiasis may be observed in individuals with Wilson's disease as a coexisting disease.

Diffuse increased hepatic attenuation due to copper accumulation on computed tomography (CT) can be detected in Wilson's disease. This may not be noticed due to opposing effect of fat deposition that decreases the attenuation on CT. Parenchymal heterogeneity and contour irregularity are other possible findings in Wilson's disease. Unenhanced CT generally reveals hyperdense nodules that will be hypodense on contrast-enhanced CT images and become more apparent on portal venous phase images (Akpinar and Akhan 2007; Li et al. 2011). The hyperdense nodules on unenhanced CT are seen hyperintense on T1W images and hypointense on T2W MR images due to paramagnetic effect of copper. Honeycomb pattern is defined as hyperdense nodules with hypodense septa on unenhanced CT with enhancement of septa after contrast administration (Li et al. 2011). This pattern is detected as hypointense nodules with surrounding hyperintense septa on T2W images (Fig. 6). Honeycomb pattern is proposed as the most sensitive imaging finding for Wilson's disease (Vargas et al. 2016). Perihepatic fat layer can also be observed as perihepatic hypodense zone on CT and hyperintense on MR images. In advanced stages of the disease, findings consistent with portal hypertension would

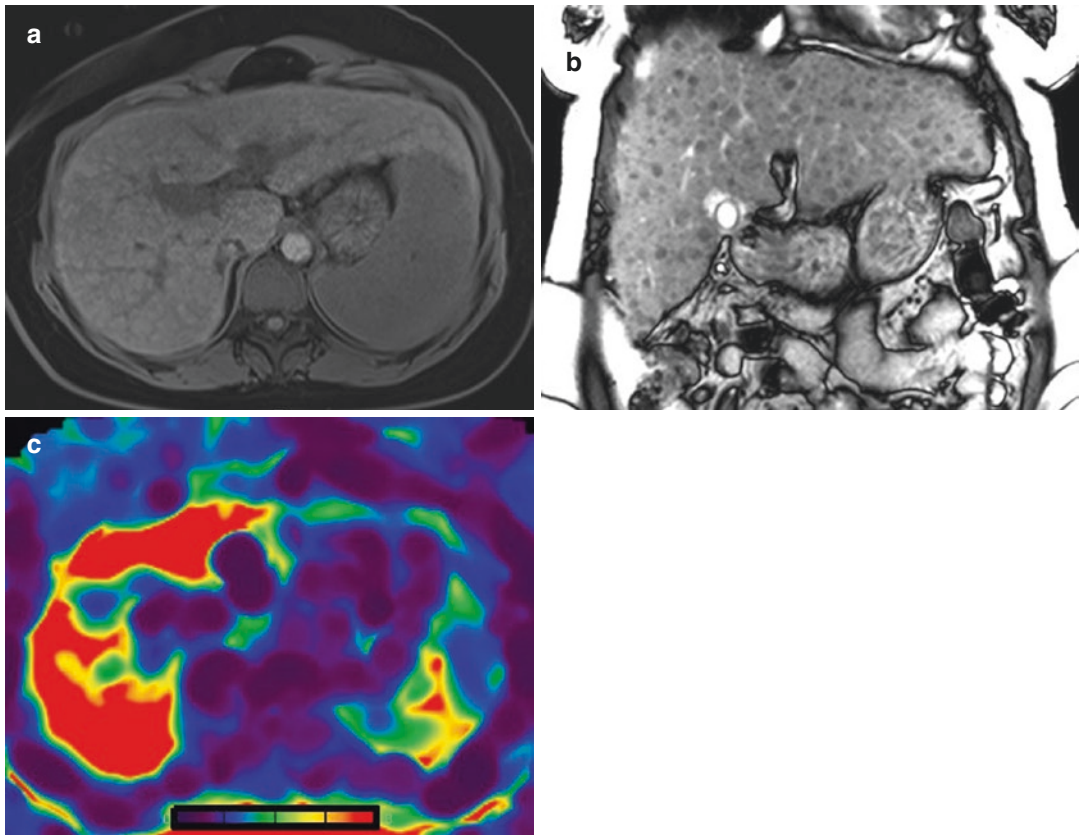

Fig. 6 A 34-year-old female patient with diagnosis of Wilson. T1W images demonstrate hypointense nodules (**a**) and T2W coronal image demonstrates hypointense nodules with surrounding hyperintense septa (**b**). MR elastography demonstrates severe fibrosis in this patient with liver stiffness value of 8 kPa (**c**)

be evident. Contrary to other chronic liver diseases, caudate lobe hypertrophy is not an expected finding and normal caudate-to-right lobe ratio is reported as a diagnostic finding in Wilson's disease (Akpinar and Akhan 2007). Wilson's disease complicated by hepatocellular carcinoma is not an infrequent finding and should be evaluated in follow-up imaging.

5 Alpha-1 Antitrypsin Deficiency

Alpha-1 antitrypsin deficiency (AATD) is a rare hereditary disease with decreased production of alpha-1 antitrypsin that results in serious lung and/or liver disease. Aggregation of alpha-1 antitrypsin polymers within the endoplasmic reticulum of liver cells promotes hepatocyte injury and forms periodic acid-Schiff-positive inclusions, which are hallmark biopsy feature in AATD-related liver disease (Edgar et al. 2017). Phenotypic expression may vary based on genetic and environmental factors in both lung and liver diseases in AATD. The clinical course of AATD-related liver disease is poorly understood that affects about 10% of the patients with AATD (Townsend et al. 2018). There is no specific imaging finding defined for AATD-related liver disease in the literature. Imaging findings of chronic liver disease and portal hypertension may be detected in advanced liver disease. In recent studies, it was shown that elastography techniques performed with MRI and US have the potential to detect AATD-related liver fibrosis before development of cirrhosis (Kim et al. 2016; Reiter et al. 2018).

6 Radiation Injury

Radiation-induced liver disease (RILD), in other words radiation hepatitis, is a form of veno-occlusive disease with fibrous obliteration of hepatic venules. It is a complication of radiotherapy and is characterized with anicteric ascites, hepatomegaly, and elevated liver enzymes within 2–8 weeks after radiotherapy. Imaging finding characteristically demonstrates a sharp line of demarcation between normal and abnormal parenchyma compatible with radiation port (Fig. 7). Hypodense area at unenhanced CT and hypo- or hyperdense area at enhanced CT reveal RILD (Unger et al. 1987). In patients with hepatic steatosis, irradiated zone may demonstrate higher attenuation than fatty liver. Affected area appears hypointense on T1W images and hyperintense on T2-weighted images due to edema on MRI. Imaging findings usually regress within 4–6 months and atrophy of the irradiated area and compensatory hypertrophy of the remaining liver can be observed (Federle 2004).

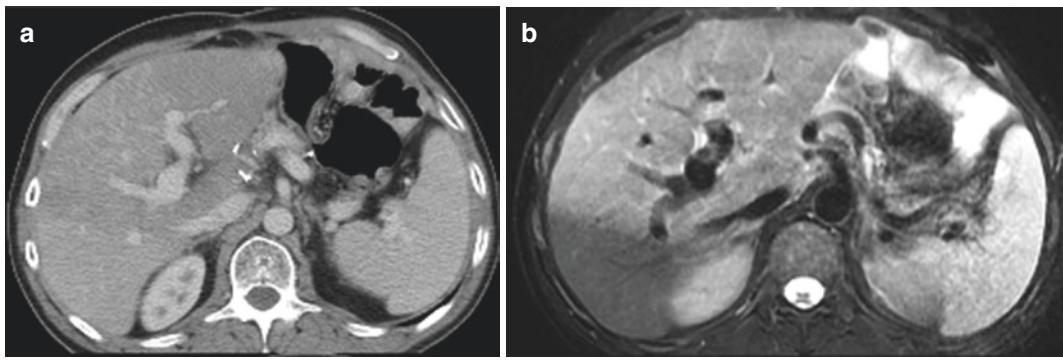

Fig. 7 A patient with breast carcinoma with a history of radiation therapy. A well-demarcated hypodense area on contrast-enhanced CT image (**a**) and hyperintense area on T2W image (**b**) that are consistent with radiation port are seen

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