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Review

Hepatic capsular retraction: An updated MR imaging review

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ABSTRACT

Hepatic capsular retraction is a morphologic descriptor that refers to invagination or focal flattening of the typical smooth contour of the liver capsule. It is an uncommon finding that, when combined with other imaging features and clinical context, can help to refine the differential diagnosis in patients with liver lesions. Although this descriptor has historically been used in reference to a small subset of benign and malignant lesions, the differential has since been expanded with the discovery of new entities causing capsular retraction as well as with novel and increased use of liver-directed treatment techniques. Additionally, modern imaging techniques now allow for improved detection and characterization of capsular retraction. In this review, we discuss these common and uncommon causes of capsular retraction, with an emphasis on findings from body MRI.

1. Background

Hepatic capsular retraction is a morphologic descriptor referring to invagination or focal flattening of the typical smooth contour of the liver capsule. It is a rare finding on cross-sectional imaging, seen in approximately 2% of patients [1]. While once believed to be exclusively correlated with hepatic malignancy, capsular retraction is in fact also associated with several types of benign lesions and with post-treatment changes [2–4]. Although nonspecific, the finding of hepatic capsular retraction can help the practicing radiologist to formulate and refine a differential diagnosis in the setting of other imaging findings and clinical context.

The underlying cause of capsular retraction is specific to the type of lesion. For example, malignancies may cause intralesional or adjacent inflammatory fibrosis, and treatment may cause tumoral necrosis and desmoplastic reaction to the hepatic parenchyma. Benign processes may cause localized atrophy due to biliary stasis or vascular insult [4,5]. Extrinsic etiologies may also cause compressive force on the liver capsule, yielding a pseudoretraction appearance from distortion of the hepatic parenchyma.

Though the phenomenon of capsular retraction and its findings on CT have been reviewed in prior work, new and more ubiquitous use of liver-directed treatment techniques such as transjugular intrahepatic portosystemic shunt (TIPS) placement and transarterial chemoembolization have expanded the differential for capsular retraction [3,4,6–8]. Additionally, new entities have been described to cause capsular retraction and modern imaging techniques such as high magnetic field strength body magnetic resonance imaging (MRI) with dynamic contrast-enhanced imaging, now also allow for improved detection and characterization. Thus, the unique contribution of this review is to provide an updated and comprehensive review of the various etiologies of hepatic capsular retraction in the context of modern MRI techniques (Table 1).

2. Malignant lesions

2.1. Intrahepatic cholangiocarcinoma

Cholangiocarcinoma is the second most common primary liver cancer and can arise from either the intrahepatic or extrahepatic bile ducts. Intrahepatic cholangiocarcinoma is less common than the extrahepatic form, although its prevalence in the United States is increasing [9]. Chronic biliary inflammation is the most common risk factor for the development of intrahepatic cholangiocarcinoma; this chronic inflammation may be caused by inflammatory conditions such as primary sclerosing cholangitis in the United States and *Clonorchis* infection in endemic regions [9].

Capsular retraction is observed in 20% of cholangiocarcinoma tumors and is most commonly associated with the intrahepatic variant [10,11]. Intrahepatic cholangiocarcinoma is frequently mass-forming

Abbreviations: CT, computed tomography; EHE, epithelioid hemangioendothelioma; HCC, hepatocellular carcinoma; MRI, magnetic resonance imaging; TE, echo time; TIPS, transjugular intrahepatic portosystemic shunt

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Table 1
Conditions associated with capsular retraction.

Malignant lesions	Benign lesions	Iatrogenic and post-treatment changes	Mimickers and other conditions
• Intrahepatic cholangiocarcinoma • Epithelioid hemangioendothelioma • Metastatic disease • Hepatocellular carcinoma (including fibrolamellar subtype)	• Sclerosed/sclerosing hemangioma • Inflammatory pseudotumor • Confluent hepatic fibrosis	• Pseudocirrhosis • Changes after transarterial chemoembolization • Changes after radiation therapy • Changes after transjugular intrahepatic portosystemic shunt placement	• Biliary obstruction (benign or malignant) • Normal variants • Trauma • Pseudomyxoma peritonei

with a fibrotic component, often inducing chronic bile duct obstruction and atrophy of the adjacent liver parenchyma, collectively contributing to retraction of the hepatic capsule [11–13]. Additionally, capsular retraction is more likely to be seen when the lesion is peripherally located, given the proximity of the capsule.

On MRI, cholangiocarcinoma often demonstrates heterogeneous hyperintense signal on T2-weighted images relative to background hepatic parenchyma, though the mass-forming variant may be hypointense on T2-weighted images due to its fibrotic components. On dynamic postgadolinium T1-weighted images using traditional extracellular contrast agents, cholangiocarcinoma demonstrates a contrast-enhancement pattern similar to the pattern seen on CT, including irregular continuous rim enhancement on the arterial phase followed by progressive intralesional accumulation of contrast (Fig. 1) [14]. When novel hepatobiliary contrast agents such as Gd-EOB-DTPA (Eovist/Primovist, Bayer HealthCare Pharmaceuticals Inc.) are used, cholangiocarcinoma is typically hypointense to background hepatic parenchyma on delayed hepatobiliary phase images, although the degree of hypointensity can be somewhat heterogeneous. On hepatobiliary phase, a target appearance may be seen in the context of cloud-like central enhancement with peripheral hypointense rim [15]. Typically, intrahepatic cholangiocarcinoma demonstrates impeded diffusion with lower apparent diffusion coefficient values than the values seen with benign lesions. Up to 75% of mass-forming intrahepatic cholangiocarcinomas may also demonstrate a target appearance on diffusion weighted images, thought to be due to alternating bands of areas of fibrosis or necrosis adjacent to the cellular tumor cells [16,17].

Intrahepatic cholangiocarcinoma is often associated with upstream biliary duct dilation, the extent of which is more appreciated on heavily T2-weighted MRI sequences or dedicated MR cholangiopancreatography sequences. The growth pattern of intrahepatic cholangiocarcinoma is typically aggressive, with involvement of the intrahepatic vasculature in up to 50% of tumors [11]. Regional lymph node involvement is often present, with distant metastasis seen on autopsy.

In a cirrhotic liver with capsular retraction, the primary differential consideration is hepatocellular carcinoma (HCC), which generally shows enhancement on arterial phase imaging with intralesional washout and capsule-like enhancement on later phase [14,18,19]. Because small cholangiocarcinomas can be diffusely hypervascular, the lack of washout is a key feature differentiating cholangiocarcinoma from HCC. In contrast to cholangiocarcinoma, HCC is rarely associated with biliary duct dilation; if this finding is present, advanced HCC is usually evident [14,19].

2.2. *Epithelioid hemangioendothelioma*

Epithelioid hemangioendothelioma (EHE) is a rare, low-grade primary malignant vascular neoplasm of the liver that occurs primarily in young female patients with no identifiable risk factors. This malignant tumor is relatively slow growing and generally low grade. Treatment includes resection and/or transplant, which makes accurate diagnosis essential [20]. EHE arises from the epithelioid cells and generates a

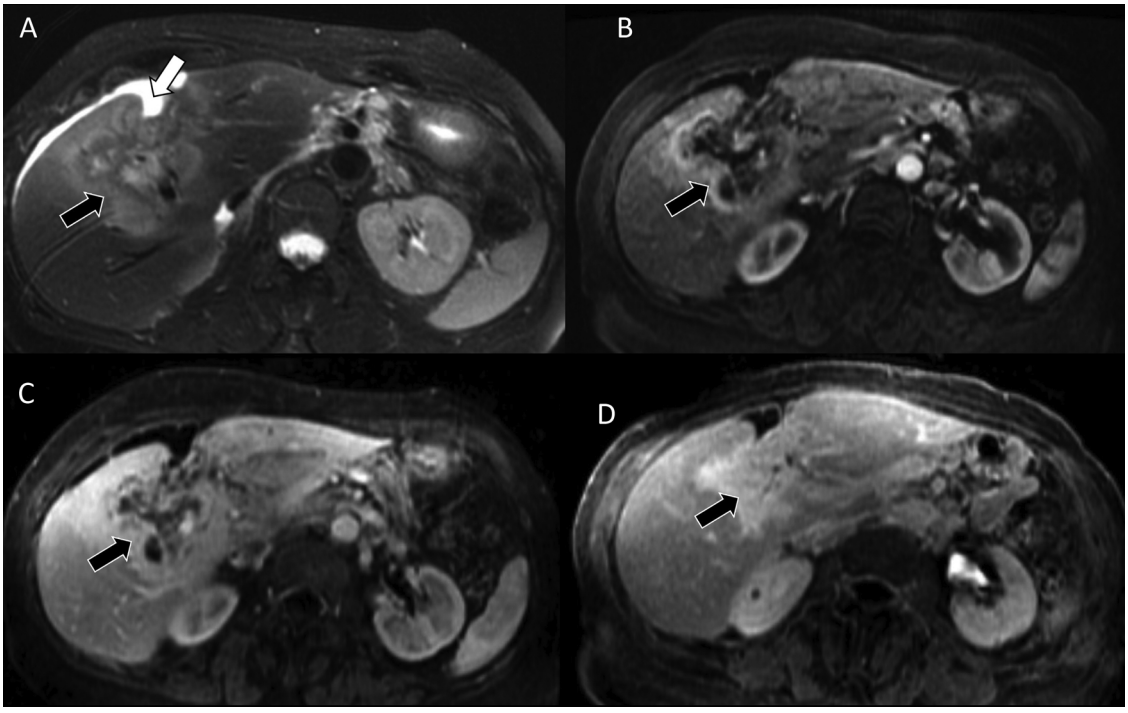

Fig. 1. Intrahepatic cholangiocarcinoma (unknown history). (a) T2-weighted imaging demonstrates a heterogeneously hyperintense mass (black arrow) in the right hepatic lobe that is associated with marked overlying capsular retraction (white arrow). The mass (black arrow) demonstrates progressive enhancement throughout dynamic postcontrast T1-weighted imaging, including arterial (b), portal venous (c), and delayed (d) phases. Biliary ductal dilation is also present.

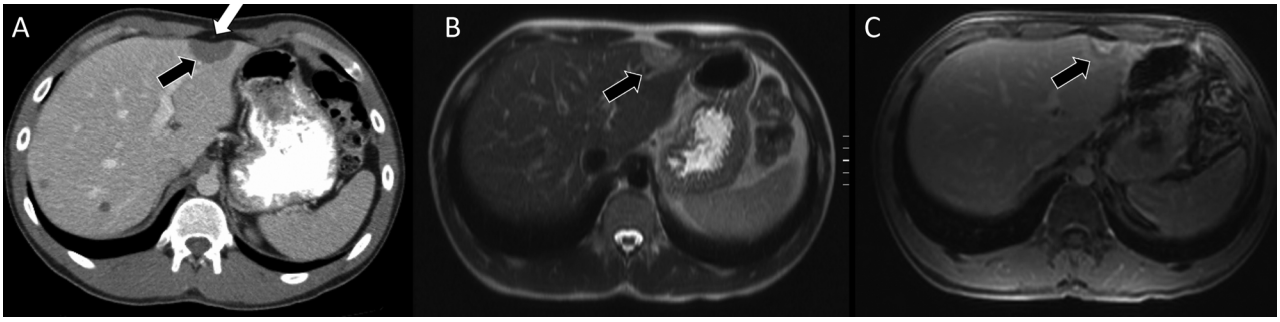

Fig. 2. 23 year-old male with epithelioid hemangioendothelioma. (a) Initial CT shows a peripheral subcapsular lesion (black arrow) in the left hepatic lobe with focal capsular retraction (white arrow). (b) Subsequent MRI shows that the lesion is hyperintense on T2-weighted imaging. (c) Postcontrast T1-weighted imaging demonstrates a "targetoid" pattern (ie, hypointense center with concentric outer layers of high and low signal) as well as marginal hypervascularity.

fibrous, myxoid stroma typically occurring in subcapsular regions [20]. These factors account for its association with capsular retraction, which is seen in 10% to 50% of tumors [21,22].

EHE is often composed of multiple round lesions with increased signal intensity on T2-weighted images. When conventional gadolinium contrast agents are used, EHE demonstrates a "targetoid" enhancement pattern of early peripheral ring enhancement, a late-appearing thick inner border of low signal, and a central high-signal core (Fig. 2) [22]. When hepatobiliary contrast agents are used, a recently described core pattern may be present in more than 50% of patients, manifesting as a seed-like distinct center of low signal intensity [23]. Tumor involvement of EHE can become extensive, with multiple masses involving both lobes of the liver [22]. If imaged longitudinally, these discrete masses often progressively coalesce to form large conglomerate masses at later time points. In contrast to intrahepatic cholangiocarcinoma, EHE is not associated with biliary duct dilation.

2.3. Hepatic angiosarcoma

Primary hepatic angiosarcoma is a rare tumor, accounting for less than 2% of primary hepatic tumors; however, it is the most common primary malignant hepatic tumor of mesenchymal origin [24]. Historically, angiosarcoma was associated with environmental carcinogen exposure such as thorotrast, vinyl chloride, and arsenic, but more modern cases occur in the absence of clear risk factors [25]. It is a very aggressive tumor with a poor prognosis because of the high likelihood of multifocal involvement or metastatic disease at presentation. Additionally, angiosarcoma is relatively resistant to radiation, is often unresectable at diagnosis because of the presence of advanced disease, and has a high recurrence rate that generally precludes transplant.

Because of the poor prognosis associated with this condition, it is imperative to differentiate hepatic angiosarcoma from more common vascular lesions such as hemangioma. Although both entities can be associated with capsular retraction, distinctive features that favor a diagnosis of hepatic angiosarcoma include heterogeneous modestly increased intensity on T2-weighted images (as opposed to the more strongly hyperintense T2-weighted signal seen with conventional hemangiomas), the presence of multiple lesions, and the vascular hallmarks of prominent intratumoral vessels and irregularly shaped enhancing foci (Fig. 3) [26]. Several patterns of growth have been described, including multiple small nodules, a large solitary mass, and a diffuse infiltrating form. Regardless of the pattern of growth, aggressive temporal growth is highly suggestive of angiosarcoma.

2.4. Fibrolamellar and conventional HCC

Both fibrolamellar HCC and the more common conventional HCC have been described as causing capsular retraction, but the finding of capsular retraction is more specific for fibrolamellar HCC (Fig. 4). Capsular retraction in the context of fibrolamellar HCC is thought to occur in fewer than 10% of tumors [27]. Although these entities are similar in name, they have distinct imaging and demographic features that can be used to identify the lesions.

Fibrolamellar HCC is an uncommon form of HCC that is slow growing and predominantly found in patients without cirrhosis. Fibrolamellar HCC is equally common among men and women with an earlier average age of presentation than is seen with conventional HCC [28,29]. The prognosis of fibrolamellar HCC is better than that of conventional HCC if the lesion is deemed to be surgically resectable; however, fibrolamellar HCC may present later with more locally aggressive features and metastases [28,29]. It is therefore important to recognize this entity early.

Fibrolamellar HCC tends to show central scarring (sometimes with calcifications) with decreased signal on T2-weighted images, whereas conventional HCC often lacks a central scar [29]. Fibrolamellar HCC tumors also tend to be large (> 10 cm) solitary lesions that have distinct margins with more heterogeneous arterial enhancement [29]. Conventional HCC, on the other hand, has a characteristic enhancement pattern with avid arterial enhancement and washout with a capsule-like appearance on delayed phase imaging. Intralesional fat may also be present on T1-weighted in- and out-of-phase gradient echo imaging.

Lymphadenopathy is present in more than 60% of patients with fibrolamellar HCC and may or may not be present in those with conventional HCC [29,30]. Liver cirrhosis, while commonly associated with conventional HCC, is not a risk factor for fibrolamellar HCC. Finally, conventional HCC is more likely to feature portal vein invasion with tumor thrombus.

2.5. Untreated hepatic metastases

Metastatic disease is the most common malignant hepatic lesion, much more common than primary hepatic malignancies. Additionally, the liver is the second most common site of metastasis after the lymph nodes. Although relatively few metastases demonstrate capsular retraction, the comparatively high prevalence of metastases makes them the most common single cause of hepatic capsular retraction. One study found that of histologically proven metastatic lesions causing capsular retraction, 47% were colorectal primary lesions; 24%, pancreatic carcinomas; 6%, bronchogenic lesions; 6%, esophageal lesions; 6%, breast lesions; 6%, gallbladder lesions; and 6%, pancreatic neuroendocrine lesions [5]. Recently, capsular retraction has also been noted to occur in metastatic disease from basal cell carcinoma and hepatoid adenocarcinoma, a rare tumor that originates predominantly from the stomach [31,32]. Hepatic capsular retraction, although not specific for a particular primary neoplasm, generally indicates a desmoplastic or scirrhous component of the tumor and is therefore more frequently seen in these types of lesions [8].

Hepatic metastatic disease can be effectively assessed with cross-sectional imaging, particularly MRI, which is considered to have the highest diagnostic performance among modern cross-sectional

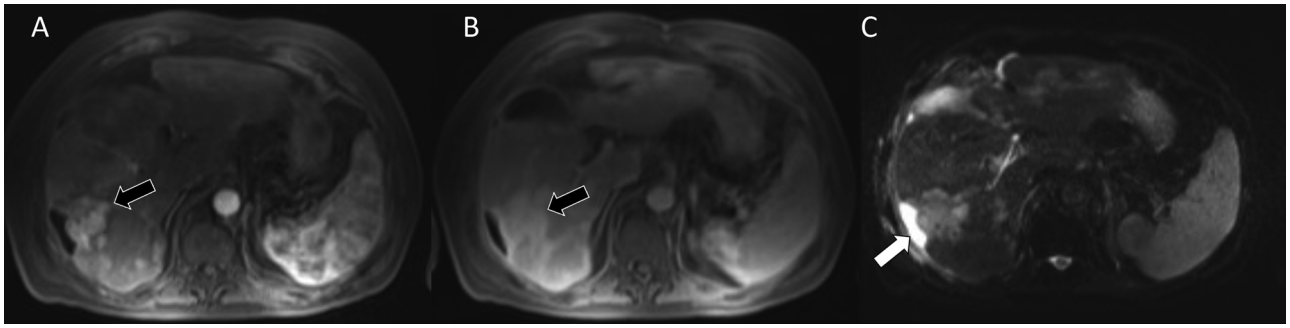

Fig. 3. Hepatic angiosarcoma (unknown history). (a) Arterial phase postcontrast T1-weighted imaging reveals multiple hypervascular lesions throughout the liver, with a dominant briskly enhancing mass (black arrow) in the right hepatic lobe. (b) This mass demonstrates persistent enhancement on later dynamic postcontrast T1-weighted imaging. (c) The mass, which is mild to moderately hyperintense on T2-weighted imaging, has caused capsular retraction (white arrow).

techniques. The reported sensitivity of MRI for hepatic metastasis ranges from 80% to 100%, with a reported specificity of up to 97% [33]. MRI demonstrates particular benefit in characterizing subcentimeter lesions that are often indeterminate on CT scans [33]. Some MRI sequences are particularly useful regardless of tumor type, including T2-weighted imaging and diffusion-weighted imaging. Most metastases demonstrate a modest level of T2-weighted hyperintensity, similar to that seen in the spleen. The degree of signal hyperintensity can be used to distinguish metastases from other lesions with more robust hyperintensity (such as hemangioma and cyst) through the use of T2-weighted images with varying echo times (TEs), including moderate (TE ˜90 ms) and heavy (TE ˜180 ms) weighting [34]. Metastases also frequently demonstrate impeded diffusion, manifesting as high signal intensity on diffusion-weighted imaging with matching low apparent diffusion coefficient value.

The enhancement pattern of metastases with traditional extracellular gadolinium contrast agents or iodinated CT contrast agents varies depending on the primary tumor. For example, hyperenhancing metastases tend to be from melanoma or from renal cell, thyroid, carcinoid, or neuroendocrine tumors, whereas hypoenhancing metastases tend to be from lung, gastrointestinal, or pancreatic primary tumors. Metastases from breast cancer have a variable enhancement pattern, with both hypovascular and hypervascular metastases reported. Mucinous cancers tend to present as small cystic lesions that can mimic simple cysts. However, a subtle difference in lesion contour/boundary between T2-weighted imaging and postcontrast imaging is often evident because of the presence of tiny enhancing marginal components [34]. When hepatobiliary contrast agents such as Gd-EOB-DTPA are used, metastases are generally well visualized. Because metastatic foci lack the appropriate transport proteins to accumulate Gd-EOB-DTPA within the cells, metastatic lesions appear hypointense versus background liver on 20-minute delayed hepatobiliary phase images [33,34].

3. Benign lesions

3.1. Hepatic hemangioma

Hemangiomas, the most common benign liver neoplasms, arise from proliferative vasculature and are supported by a fibrous matrix. They are incidentally discovered in 2.5% to 3.4% of imaged lesions [35,36]. Capsular retraction can be seen in association with hepatic hemangiomas in three different settings: giant hemangiomas [37], hemangiomas occurring in patients with liver cirrhosis [5,38–40], and sclerosing hemangiomas (also called thrombosed or hyalinized hemangiomas) [38–40].

Capsular retraction has been reported only rarely in the first two settings. Giant hemangiomas are variably defined as hemangiomas measuring > 6 cm and often retain the typical peripheral, nodular, and discontinuous enhancement of conventional hemangiomas; however, some of these lesions begin to lose enhancement centrally because of scar formation, which could give rise to capsular retraction [37]. In the cirrhotic liver, hemangiomas lose their characteristic imaging appearance and become smaller and more fibrotic, likely causing capsular retraction [40].

Sclerosing hemangiomas, which are uncommon, are associated with capsular retraction in up to 70% of lesions [38]. Central thrombosis leads to degeneration of a hemangioma, which can occur with or without fibrotic changes. Hemangiomas are described as “sclerosing” if there is partial obliteration of the vascular spaces or “sclerosed” if there is complete obliteration of the vascular spaces. The mechanism of capsular retraction in liver hemangioma is thought to be caused by this progressive fibrosis and scarring due to sclerosis, which may also induce atrophy of the hemangioma and surrounding parenchyma [41].

On MRI, conventional hemangiomas typically have homogeneous high signal on T2-weighted images, with high signal intensity remaining even on images with a long echo time (TE ˜180 ms). Similar to

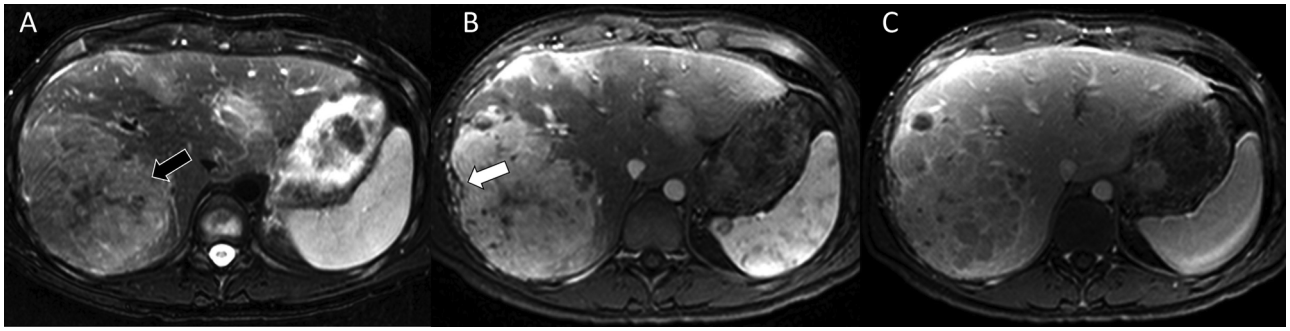

Fig. 4. 38 year-old female with fibrolamellar hepatocellular carcinoma. (a) T2-weighted imaging demonstrates a large predominantly hyperintense mass (black arrow) with a notable hypointense central scar. (b) The mass, which has caused capsular retraction (white arrow), is hypervascular on arterial phase postcontrast T1-weighted imaging, with subsequent areas of washout during the portal venous phase (c). There are no morphologic features of cirrhosis.

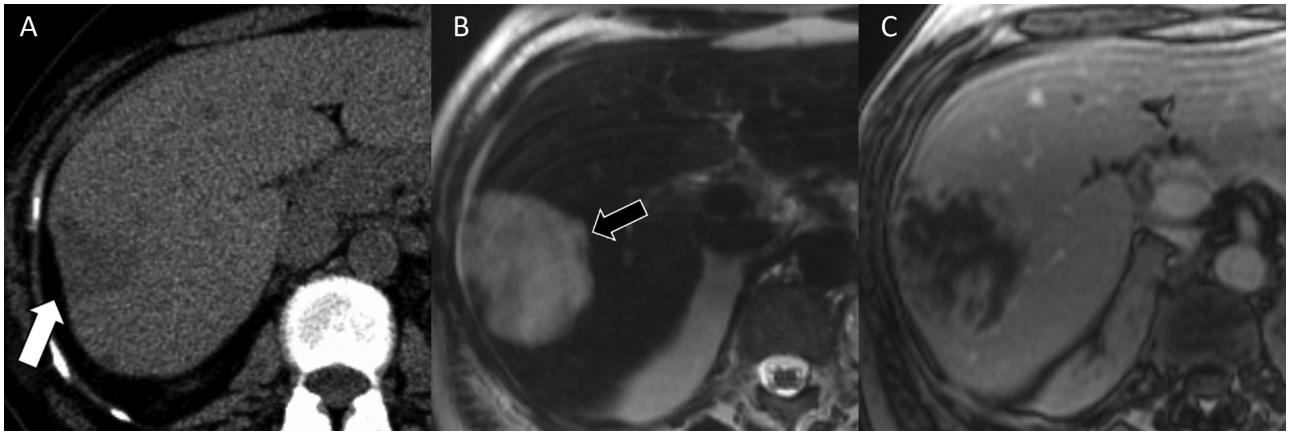

Fig. 5. 65 year-old female with incidentally discovered sclerosing hemangioma. (a) Non-contrast CT scan demonstrates a hypoattenuating lesion in the right hepatic lobe with capsular retraction (white arrow). (b) T2-weighted imaging performed 10 years prior demonstrates a corresponding hyperintense lesion in the right hepatic lobe. (c) On venous phase postcontrast T1-weighted imaging, this lesion demonstrates peripheral, nodular, and discontinuous enhancement, consistent with conventional hemangioma. Without the availability of the previous MRI images, this lesion would have been considered indeterminate, and biopsy would have been necessary. However, because the current lesion corresponds exactly with the previously noted hemangioma, a diagnosis of sclerosing hemangioma can be rendered confidently.

CT, these lesions demonstrate peripheral nodular discontinuous enhancement with centripetal fill and delayed hyperintensity similar to that seen in the blood pool. Sclerosing hemangiomas generally demonstrate more heterogeneous, patchy high signal on T2-weighted images and peripheral enhancement that may show loss of the nodular puddling enhancement pattern, as well as heterogeneous delayed enhancement (Fig. 5). Progressive sclerosis results in a loss of the typical radiologic features of a hemangioma; therefore, diagnosis requires biopsy unless a conventional hemangioma was demonstrated at the site on a previous study.

3.2. Confluent hepatic fibrosis

Confluent hepatic fibrosis refers to the excess synthesis of extracellular matrix tissues within the liver. It is reflective of the sequelae of chronic inflammatory changes and is suggestive of advanced disease, most commonly in the context of long-standing alcoholic cirrhosis. Confluent hepatic fibrosis generally appears as triangular, wedge-shaped, hypoattenuating areas that radiate peripherally from the porta hepatis. When subcapsular involvement occurs, overlying capsular retraction predominantly involves the anterior right lobe or medial left lobe of the liver (Fig. 6) [42–44]. These fibrotic regions can result in a delayed enhancement pattern and are frequently hyperintense on T2-weighted images. Importantly, confluent hepatic fibrosis is typically non-space-occupying with wedge-shape and bile duct dilation is not observed; these features help to distinguish this condition from the mass-like intrahepatic cholangiocarcinoma which also demonstrates progressive contrast enhancement.

3.3. Inflammatory pseudotumor

Inflammatory pseudotumors are focal lesions composed predominantly of inflammatory cells within a fibrous stroma [45]. Though the etiology remains to be elucidated, proposed mechanisms include autoimmune causes, trauma, and infection [46]. The imaging appearance of inflammatory pseudotumor may often be nonspecific, which gives rise to its moniker as “the great mimicker” [47,48]. On MRI, they typically have low signal on T1-weighted images and increased signal on T2-weighted images with variable enhancement patterns which reflects the variable cellular composition (Fig. 7) [49]. Final diagnosis is often one of exclusion, typically after biopsy with pathologic evaluation excludes malignancy, infection, and other processes.

4. Iatrogenic and post-treatment changes

4.1. “Pseudocirrhosis” after treatment for hepatic metastases

Pseudocirrhosis in the setting of treated metastatic disease can be the result of reactive changes in both the lesion itself and in the uninvolved liver parenchyma after nonoperative therapy (Fig. 8). Post-treatment changes contributing to capsular retraction include necrosis, fibrosis, and atrophy of the lesion [1,2,34,50,51], as well as nodular

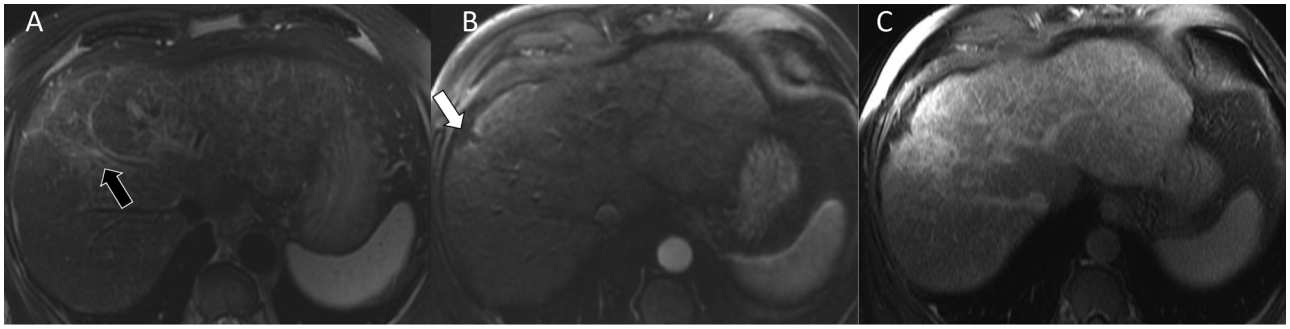

Fig. 6. Confluent hepatic fibrosis in a patient with cirrhosis. (a) Fat-suppressed T2-weighted imaging demonstrates heterogeneous mildly hyperintense geographic lesion (black arrow) involving the anterior right lobe. (b) Arterial phase postcontrast T1-weighted imaging demonstrates vague, low-level enhancement with adjacent capsular retraction (b, white arrow). (c) Delayed postcontrast T1-weighted imaging demonstrates corresponding avid enhancement. Note the absence of biliary ductal dilation. Capsular retraction, delayed enhancement and mild hyperintensity on T2-weighted imaging can mimic cholangiocarcinoma in a cirrhotic patient.

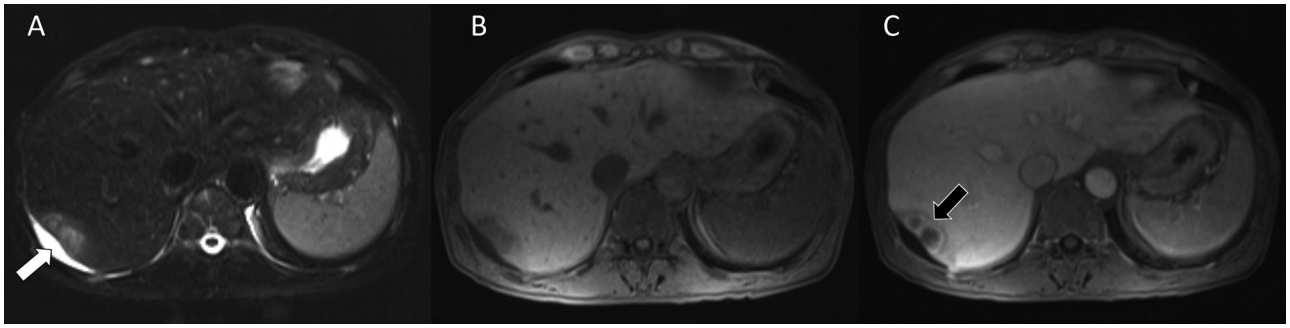

Fig. 7. 59 year-old woman with inflammatory pseudotumor. (a) Fat-suppressed T2-weighted imaging demonstrates a mass-like area of hyperintensity in the lateral right hepatic lobe with associated capsular retraction (white arrow). The mass is hypointense on T1-weighted imaging (b) and enhances peripherally on post-contrast images (c, black arrow). Further patient workup found no abnormalities and the mass subsequently resolved with prednisone.

regenerative hyperplasia in the liver parenchyma adjacent to the treated tumor [51–53]. Capsular retraction is most frequently described after treatment of large breast cancer metastases [50,52–54]. One series observed that capsular retraction in this setting is independent of the number of metastatic lesions, type of chemotherapy, and histopathologic diagnosis of the primary tumor [54]. The authors also found that capsular retraction was present in 50% of cases of breast cancer metastasized to the liver.

4.2. Changes after locoregional therapy

Numerous locoregional therapeutic interventions are used to treat, palliate, or downstage liver tumors, most frequently HCC. Endovascular techniques include bland embolization, chemoembolization, and yttrium-90 radioembolization. Ablative techniques include radiofrequency, microwave, and thermal ablation. Capsular retraction may be observed adjacent to the targeted lesion after any of these interventions due to the induction of tumor necrosis [55,56].

Treatment with chemoembolization can result in the appearance of a heterogeneous lesion with varying degrees of hyperintensity on T2-weighted images and partial enhancement depending on the degree of tumor necrosis (Fig. 9). Capsular retraction can occur after chemoembolization due to intratumoral necrosis and the resulting inflammatory response in the perilesional tissues. Capsular retraction has been reported to occur after as many as 89% of radioembolization procedures performed for the treatment of HCC [55]; this is thought to be due to necrosis of the tumor and fibrosis and scarring of the treated parenchyma [57]. Reduced tumor size, tumor necrosis, and lack of enhancement are typically seen after successful radioembolization treatment.

4.3. Hepatic infarction

Hepatic infarction is generally rare because of the presence of a dual blood supply, with the portal vein accounting for ˜70% and the hepatic artery accounting for ˜30%. However, in patients with cirrhosis, portal blood flow is decreased and the demand for blood supply from the hepatic artery is subsequently increased [58,59]. Effective portal blood flow is decreased even further in a patient with cirrhosis who has undergone a TIPS placement. While the exact mechanism by which TIPS infarct occurs is unknown, proposed mechanisms include direct injury to an adjacent artery or vein, vascular compression by the stent, or congestion due to outflow obstruction; these effects are likely further exacerbated by the altered vascular and biochemical pathophysiology caused by the underlying disease process [60–63]. Infarctions can also occur in the setting of liver transplant due to perfusional changes occurring intraoperatively or during graft harvesting.

On cross-sectional imaging, infarcts appear as triangular wedge-shaped lesions typically located at the periphery of the liver. These infarcts can be associated with capsular retraction as they become chronic. The infarcts show variable signal intensity on T2-weighted images, low signal on T1-weighted images, and diminished enhancement or nonenhancement on postcontrast imaging (Fig. 9). While focal fat deposition may be a potential confounder of acute infarction on other imaging modalities, it is typically well recognized on MRI as signal dropout on opposed-phase T1-weighted images relative to in-phase gradient echo T1-weighted images in addition to other features such as typical location along the hepatic fissures, geographic shape, absence of mass effect, and enhancement pattern similar to adjacent hepatic parenchyma [64].

4.4. Changes after radiation therapy

Although the delivery of external beam radiation therapy is

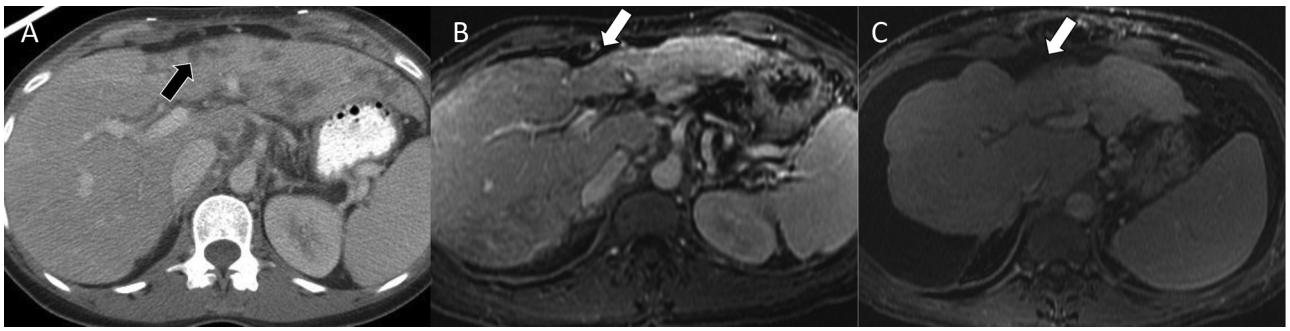

Fig. 8. 42 year-old female with breast cancer metastases to the liver causing pseudocirrhosis. (a) Initial contrast-enhanced CT scan demonstrates multiple hypoattenuating lesions along the anterior liver (black arrow), causing irregularity of capsule. Subsequent postcontrast T1-weighted MR images in venous phase obtained 1 (b) and 2 (c) years later show progressive capsular retraction (white arrows).

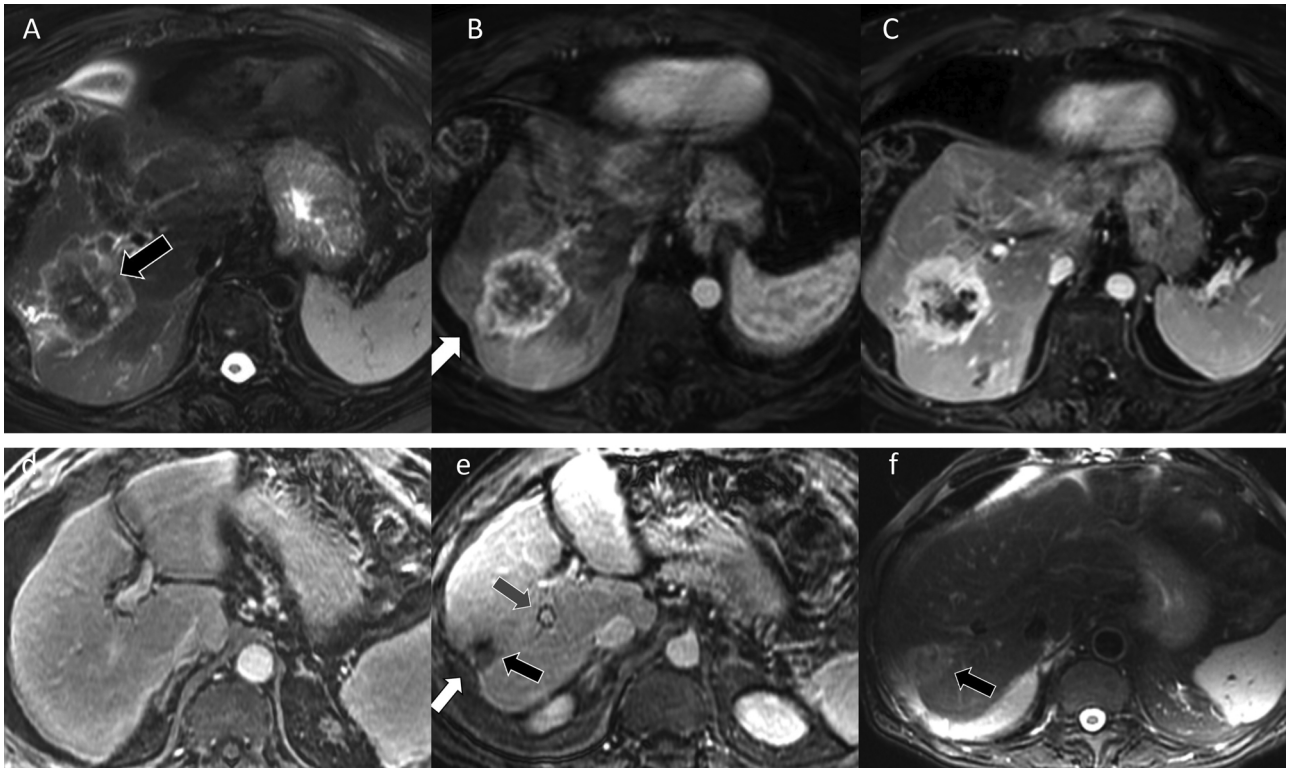

Fig. 9. Parenchymal changes after transarterial chemoembolization of hepatocellular carcinoma (top row). (a) A large mass (black arrow) in the right hepatic lobe demonstrates heterogeneous signal on T2-weighted imaging. (b) On arterial phase postcontrast T1-weighted imaging, the mass demonstrates heterogeneous, predominantly peripheral enhancement with adjacent capsular retraction (white arrow). (c) Delayed phase postcontrast T1-weighted imaging shows progressive peripheral enhancement, suggesting a central core of necrosis.
Infarction occurring after transjugular intrahepatic portosystemic shunt (TIPS) placement (bottom row). (d) Initial postcontrast MR image obtained before therapy demonstrates lobular hepatic contour. (e) Follow-up postcontrast MR image obtained after TIPS placement (gray arrow) demonstrates a new area of capsular retraction (white arrow) with a wedge-shaped area of low signal (black arrow) without enhancement on postcontrast T1-weighted imaging, corresponding to mildly increased signal on T2-weighted imaging (f, black arrow) that is consistent with infarct.

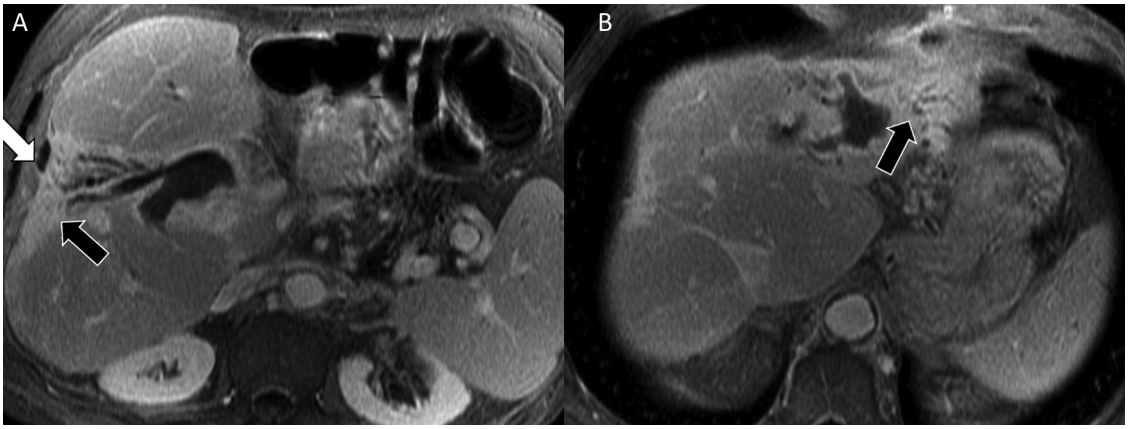

Fig. 10. Ischemic cholangiopathy (unknown history). (a and b) Axial delayed phase postcontrast T1-weighted imaging demonstrates wedge-shaped areas of enhancement (black arrows) with adjacent capsular retraction (white arrow). There is diffuse biliary dilation involving the intrahepatic ducts with luminal irregularity.

becoming more precise with advances in technology, some damage to adjacent organs can still occur, as is often true with most procedures used to deliver targeted hepatic radiation. Changes in the irradiated liver can lead to hepatic capsular retraction in the affected area. Irradiated liver parenchyma undergoes a spectrum of changes, characterized first by edema and then by progressive fibrosis and atrophy associated with capsular retraction [65]. On imaging, the edema manifests as low attenuation on CT images or as heterogeneous increased signal intensity on T2-weighted images. Fibrosis is better delineated on dynamic postcontrast imaging, manifesting as slow accumulation of contrast material within the fibrous tissue (best appreciated on delayed postcontrast imaging). The relationship between dose and extent of imaging findings is not linear.

5. Chronic biliary obstruction

Chronic biliary obstruction is often associated with hepatic capsular retraction and may result from various causes including traumatic, vascular, inflammatory, infectious, and neoplastic processes. The exact mechanism of capsular retraction varies by specific process; two

representative processes are discussed in further detail below.

5.1. Ischemic cholangiopathy

The biliary tree is reliant on arterial perfusion; therefore, ischemic cholangiopathy occurs in the setting of hepatic arterial insufficiency. Diminished arterial flow occurs in patients with arterial thrombus or vasculitis; patients who have undergone liver transplant; and those who have undergone interventions, including radiation [66]. Ischemia of the biliary tree and subsequent healing may result in stricture and/or necrosis with bile lake formation (Fig. 10) [66]. Atrophy of the liver segments drained by the affected bile ducts may occur, subsequently leading to capsular retraction [66].

5.2. Recurrent pyogenic cholangiohepatitis

Recurrent pyogenic cholangiohepatitis, an uncommon entity in the United States, is associated with infection with *Clonorchis sinensis* or *Ascaris lumbricoides*. Patients with this condition present with recurrent pain, fever, and jaundice due to effects on the biliary system. Large intraductal calculi arise from the nidus of infection and cause biliary obstruction and diffuse dilation of the ducts [50,67]. With progressive obstruction and recurrent infection, stricture and parenchymal destruction occur, leading to capsular retraction [50,67,68]. These patients are at increased risk for developing cholangiocarcinoma.

6. Mimickers of capsular retraction

Since the identification of capsular retraction can potentially lead to further imaging or surgical intervention, it is important to distinguish true capsular retraction from pseudo capsular retraction. One commonly seen cause of pseudo capsular retraction is extrinsic compression of the hepatic capsule by the adjacent ribs or diaphragm; this finding can be easily confirmed with review of multiplanar reformats. Similarly, penetrating or blunt trauma may distort the hepatic parenchyma to give an appearance of capsular retraction. However, patient history and review of prior imaging will often provide the salient information needed to make the correct diagnosis. Pseudomyxoma peritonei is a rare condition whereby mucinous peritoneal implants, most often arising from the appendix, adhere to the liver capsule in a scalloped pattern that can mimic capsular retraction [69]. If such a pattern is recognized, dedicated imaging of the lower abdomen or pelvis will often assist in the identification of the source to steer the radiologist toward the correct diagnosis.

7. Conclusion

Hepatic capsular retraction is associated with a variety of conditions and is a useful feature in liver imaging. It may be indicative of an underlying fibrotic process (either neoplastic or non-neoplastic in origin) or of focal parenchymal atrophy in response to vascular insult or chronic biliary obstruction. By itself, capsular retraction provides a useful list of potential differential diagnosis considerations. However, when combined with other imaging findings and clinical data, the presence of capsular retraction can substantially narrow a differential diagnosis, particularly when modern imaging techniques such as dynamic post-contrast body MRI are utilized.

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Conflicts of Interest

The authors have no conflicts of interest to declare.

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