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REVIEW

# Uncommon imaging evolutions of focal liver lesions in cirrhosis

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## Abstract

**Objective** The purpose of this article is to describe and illustrate uncommon imaging evolutions of benign (i.e., cyst, hemangioma, focal nodular hyperplasia-like nodules, and hepatic angiomyolipoma) and malignant (i.e., HCC and non HCC malignancies) lesions in a cirrhotic liver. The content highlights relevant pathogenesis and imaging clues for proper differential diagnosis. Revision of prior imaging and knowledge of these scenarios may help the abdominal radiologist to reach a noninvasive diagnosis and direct the patient to the most appropriate clinical management.

**Conclusion** Uncommon imaging evolutions of focal liver lesions in cirrhosis may represent a challenge for the abdominal radiologist, with atypical changes in size, and internal vascularization changes that may lead to misdiagnoses.

**Keywords** Hepatocellular carcinoma · Magnetic resonance imaging · Computed tomography · Liver cirrhosis · Liver neoplasms

## Introduction

The detection and characterization of focal liver lesions in cirrhotic patients is a daily, challenging task for the abdominal radiologist. One of the most important clinical scenarios is the differentiation of hepatocellular carcinoma (HCC)—which is the most common malignant lesion arising in a cirrhotic liver—from other focal lesions that may be encountered in cirrhosis including cysts, hemangiomas, focal confluent fibrosis, regenerative nodules, dysplastic nodules, and non-HCC malignancies. When the typical imaging features are visualized (i.e., arterial phase hyperenhancement, portal-venous phase, or delayed phase washout and enhancing capsule), it is possible to make a definitive diagnosis of HCC without the need of a pathological confirmation [1]. However, in a cirrhotic liver, both benign and malignant lesions may lack typical imaging features. The definitive diagnosis may therefore require comparison with prior studies, imaging follow-up, or tissue sampling.

Prior and serial follow-up imaging examinations allow for the evaluation of lesion stability or size change, with lesion growth suggesting a diagnosis of malignancy [1], while size stability or reduction suggesting benignity [2].

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However, the increase in size of a benign or non-HCC lesion, although uncommon, may occur [3, 4], resulting in a false positive rate of 17% when using threshold growth as major imaging feature for the diagnosis of HCC in cirrhosis [2]. Conversely, hepatic malignancies may uncommonly show spontaneous shrinkage or disappearance without treatment [5]. These atypical evolutions add several clinical challenges for the radiologists and hepatologists and may induce pitfalls in imaging interpretation with significant implications for patients' management.

In this review article, we report our experience with uncommon or very rare cases of imaging evolution of benign lesions (i.e., hepatic cyst, hemangioma, angiomyolipoma) as well as malignant lesions (i.e., HCC, cholangiocarcinoma, metastases) in cirrhotic patients. Uncommon evolution of these lesions in cirrhosis include atypical changes in size, imaging evolution of the enhancement pattern, as well as, unexpected very long-term recurrence of malignancy.

## Benign liver lesions

### Atypical size changes

Lesion stability or shrinkage in cirrhotic liver has a reported specificity of 99% for the diagnosis of benignity in untreated lesions [2]. This high specificity allows for a confident diagnosis of benign lesions even when typical imaging features are lacking. As an example, hepatic cyst may demonstrate spontaneous increased attenuation which may limit the evaluation of contrast enhancement on CT and MRI. In these cases, the signal intensity on T2-weighted images, the evaluation of subtracted images, as well as the stability or decrease in size in the long term generally permits a confident diagnosis (Fig. 1). Pseudolesions in a cirrhotic liver may appear as enhancing nodules on hepatic arterial phase, thus mimicking malignancy (Fig. 2) [6, 7]. However, the lack of visibility on MR images acquired during the hepatobiliary phase and the absence of diffusion restriction [7], stability, or decrease in size at imaging follow-up are usually sufficient to suggest a diagnosis of benignity. In cirrhosis,

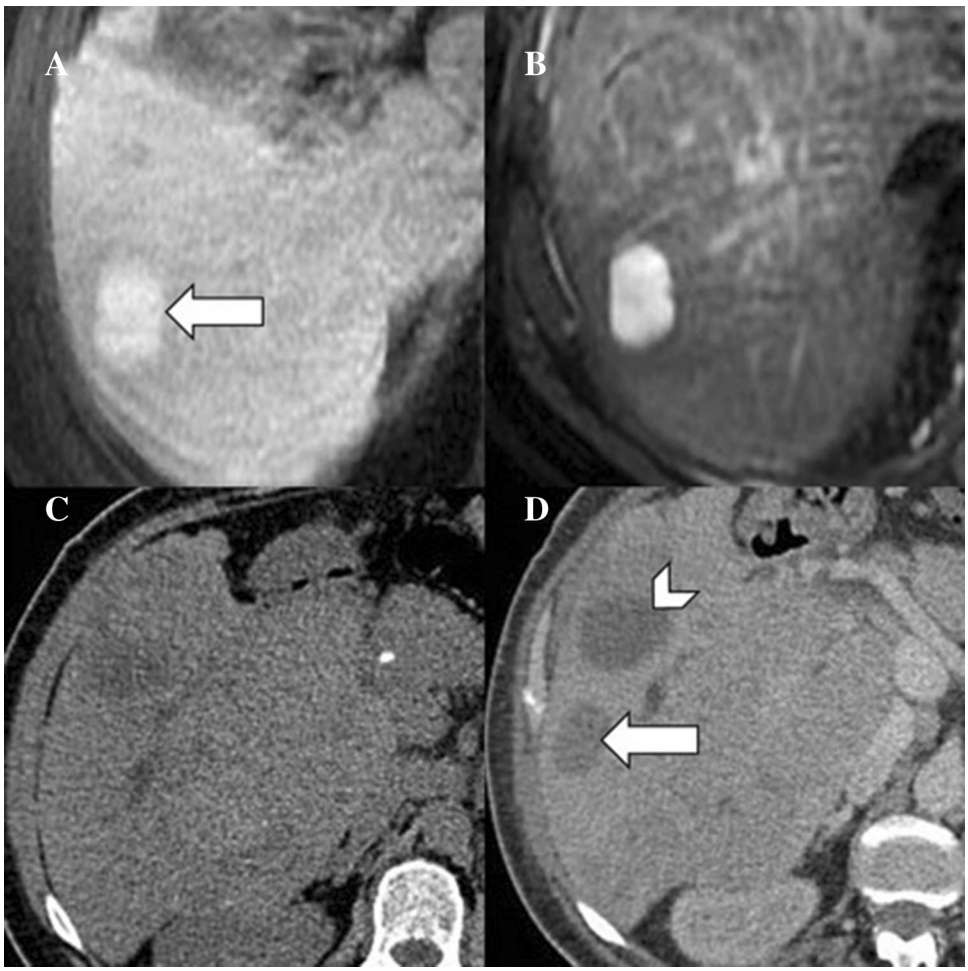


**Fig. 1** 63-year-old man with nonalcoholic cirrhosis. Axial baseline MRI scan shows a 3.6 cm cyst (arrow) hyperintense on T1-weighted image (**a**) and hyperintense on T2-weighted image (**b**), consistent with hemorrhagic cyst. At 5-year CT follow-up (C and D), the cyst (arrow) slightly reduces in size (3.0 cm), and it is iso-attenuating to the liver on unenhanced (**c**) and hypoattenuating (arrow) on portal-venous phase (**d**) CT images. Please note an adjacent observation (arrowhead) representing a treated HCC

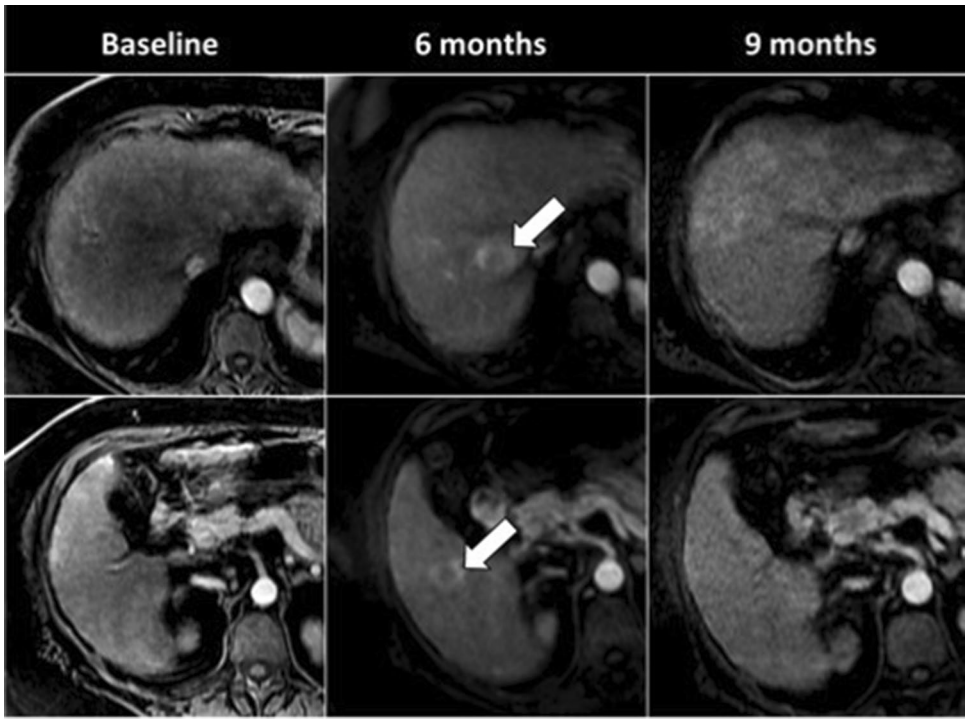


**Fig. 2** 72-year-old woman with NASH cirrhosis. On baseline MRI examination, no focal liver lesion is noted on the images obtained during the hepatic arterial phase. At six-month follow-up MRI, two lesions (arrows) with rim arterial phase hyperenhancement in segments 8 and 6 are categorized as LR-M observations (i.e., high likelihood of being malignant but with imaging presentation not typical for HCC). These lesions disappeared on the follow-up MRI performed at 9 months with no interval treatment. Ultrasound target liver biopsy obtained after the six-month follow-up MRI showed active steatohepatitis without evidence of tumor

pseudolesions are likely to represent perfusion alterations, hypertrophic or inflammatory pseudomasses, regenerative nodules, or artifacts after contrast injection [6, 7].

Hepatic cysts—which are the most common nonhepatocellular focal lesions in cirrhosis [8]—and hemangiomas—which occur in 0.6% of cirrhotic patients at CT [3]—may also increase in size, but the prevalence of this atypical evolution is unknown in the literature. Although lesion growth has a high specificity for malignancy (83–91%) [2], growth of benign lesions in cirrhotic patients may occur (Fig. 2). While it is known that benign hepatic cysts may enlarge in the general population without chronic liver disease [4], there are no studies on the natural history of cysts in cirrhosis. In our experience, hepatic cysts in cirrhosis are usually stable or decrease in size over time (Fig. 3). When enlargement occurs (Fig. 4), it usually does not reach threshold growth according to the definition of LI-RADS v. 2018 [1]. In these cases, the differential diagnosis with malignancies—i.e., HCC with cystic degeneration due to internal necrosis or cystic metastases—may be challenging,

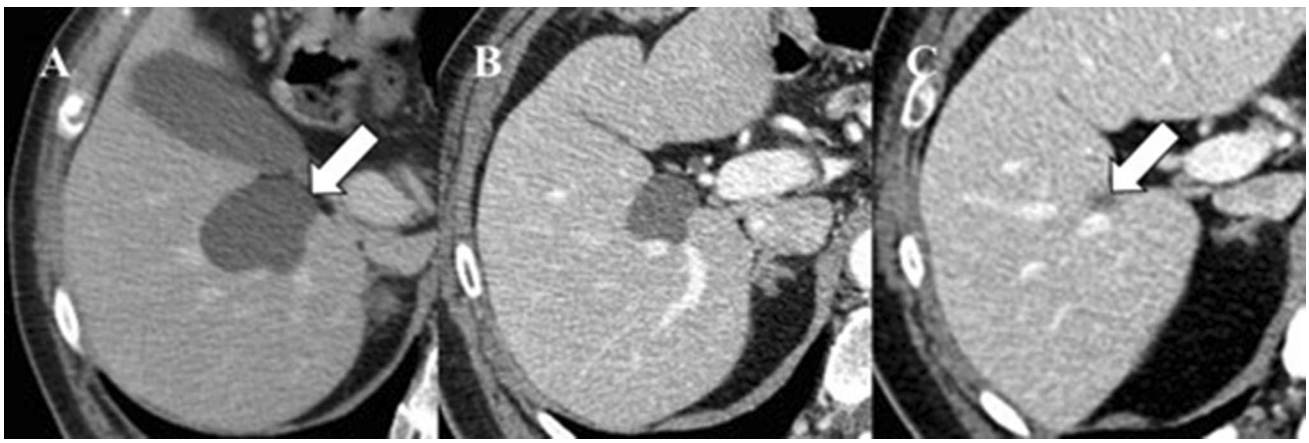


**Fig. 3** 74-year-old man with cirrhosis. Baseline contrast-enhanced CT on portal-venous phase **a** shows a 4.5 cm hepatic cyst (arrow) in segment 6. Follow-up CT imaging demonstrates shrinkage of the cyst (arrow) measuring 2.3 cm and 1.0 cm after 2 years (**b**) and 4 years, respectively (**c**). At subsequent 1-year follow-up CT (not shown), the lesion completely disappeared

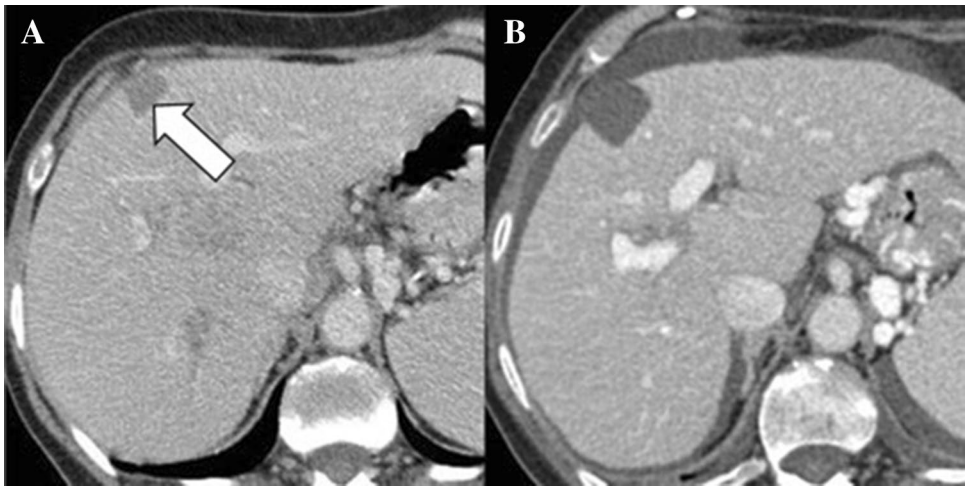


**Fig. 4** 93-year-old woman with cirrhosis and history of breast cancer. Baseline contrast-enhanced CT on portal-venous phase **a** shows a 2.0 cm hepatic cyst (arrow) in segment 8. Seven-year CT follow-up **b** demonstrates enlargement of the cyst, measuring 2.7 cm

and other imaging features, including homogeneity of lesion attenuation and lack of any contrast enhancement, should be considered to narrow the differential diagnosis [1]. To our knowledge, enlargement of hepatic hemangiomas in cirrhosis has never been reported in the literature. In our practice, we have encountered a hepatic hemangioma in cirrhosis enlarging over time, although we do not have a convincing hypothesis to explain this phenomenon (Fig. 5).

Other less-common benign lesions in cirrhotic patients include focal nodular hyperplasia-like nodules [8] and

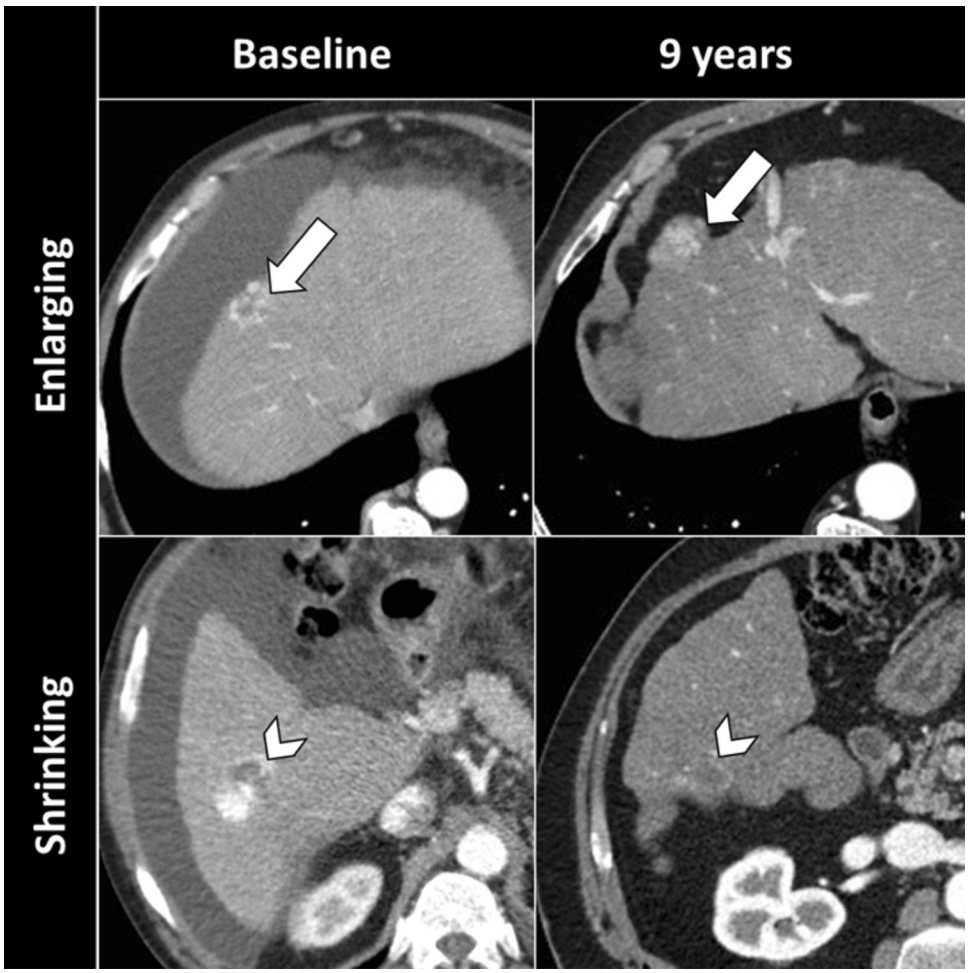


**Fig. 5** 63-year-old man with alcoholic cirrhosis. Baseline axial CT scan in the arterial phase shows a 2.0 cm hemangioma in segment 8 (*upper row*) and a 3.7 cm hemangioma in segment 6 (*bottom row*). At 9-year follow-up, the segment 8 hemangioma enlarges (from 2.0 to 3.0 cm, arrows), while the segment 6 hemangioma shrinks (from 3.7 cm to 2.0 cm, arrowheads) and shows new arterial phase hyperenhancement. The patient also had another hepatic hemangioma on the baseline CT scan that disappeared at 9-year follow-up (not shown). Note also the evolution of hepatic morphologic changes of cirrhosis on the follow-up CT scan

hepatic angiomyolipoma [9], but, to our knowledge, only scant data exist in the literature about their natural history [10, 11]. In our experience, we observed an uncommon case of growth of a lesion mimicking an HCC according to LI-RADS [1] which unexpectedly turned out to be an angiomyolipoma in cirrhosis at explant (Fig. 6).

## Internal and vascularization changes

In a cirrhotic liver, hemangiomas may demonstrate a fibrotic involution—also known as "sclerosed hemangiomas" or "hyalinized hemangiomas" [12]. This fibrotic involution may root in the altered blood flow due to obliteration of vascular channels and in the modified intrahepatic environment, and may lead to fibrotic degeneration or hemorrhage and thrombosis [12–14]. The process of sclerosis generally starts in the center and then extends to the entire lesion. These changes may result in size reduction of the hemangiomas (Fig. 7) and might explain the significantly lower size of hemangiomas in cirrhosis compared to normal liver [3, 15]. Furthermore, the fibrotic degeneration may lead to peripheral capsular retraction or concavity over the lesion (Fig. 7), and loss of the typical imaging features of hemangiomas, including T2 hyperintensity, nodular peripheral enhancement with centripetal filling and the enhancement parallel to blood vessels [3, 16, 17]. Central fibrotic degeneration may result in central hypointensity on T2-weighted images and lack of T2-shine-through effect compared to lesions

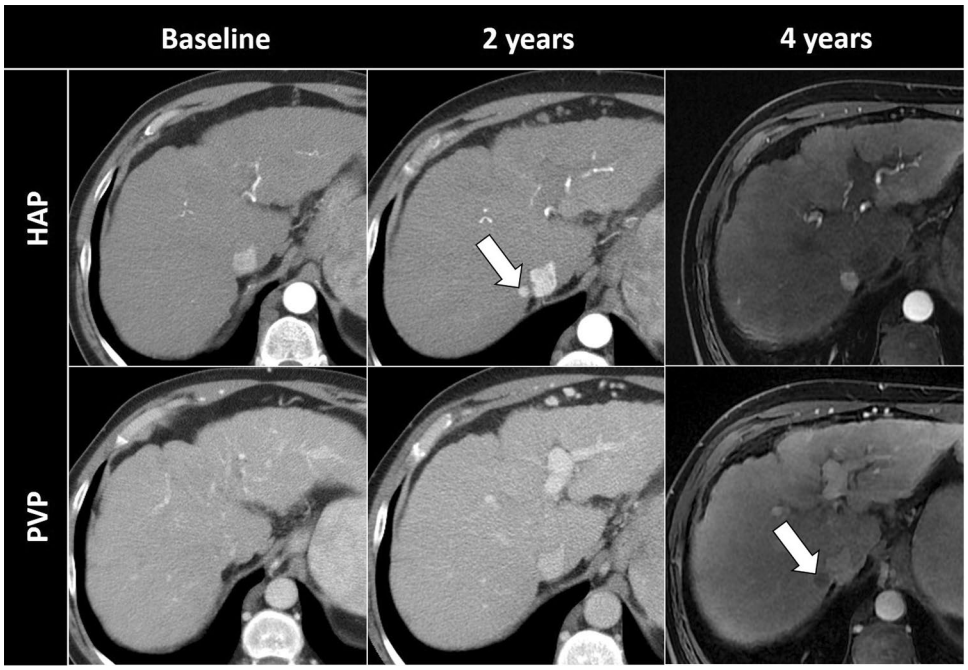


**Fig. 6** 58-year-old man with HCV and alcoholic cirrhosis. Contrast-enhanced CT and MR images obtained during the hepatic arterial (upper row) and portal-venous phase (bottom row) show a progressively slow-growing lesion during 4-year follow-up. A new arterial phase hyperenhancing lesion is clearly appreciated at 2-year follow-up (arrow) and a washout (arrow) of this lesion appeared at 4-year follow-up. The lesion was isointense on all other MR sequences (not shown). A dominant nodule (arrow) is easily visible at macroscopy specimen after orthotopic liver transplantation. At immunohistochemistry (not shown), the lesion was strongly HMB-45 positive and diagnosed as angiomyolipoma

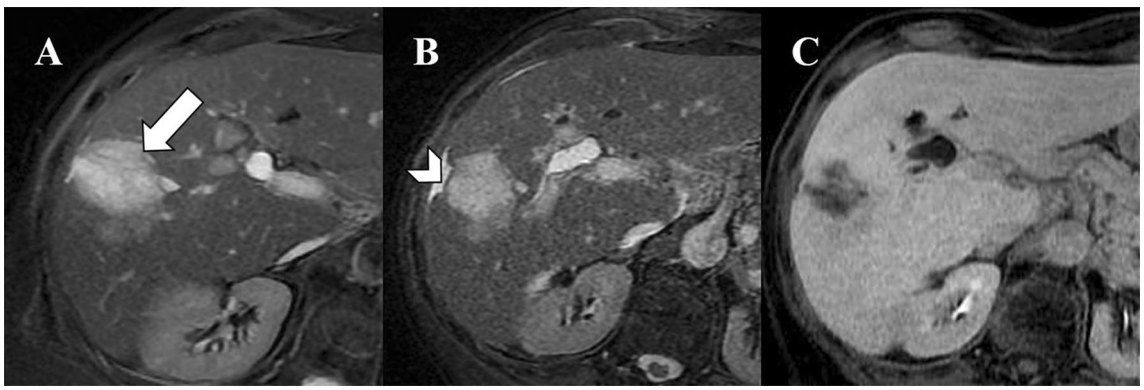


**Fig. 7** 74-year-old woman with cirrhosis. Axial T2-weighted MR images shows a hemangioma (**a**, arrow) in segment V. Spontaneous shrinkage of the lesion with progressive capsular retraction (arrowhead) **b** with incomplete globular peripheral enhancement in the delayed phase **c** is noticed at 1-year MRI follow-up

occurring in normal or mildly fibrotic liver [4, 15]. Sclerosed hemangioma may appear as a hypoenhancing lesion or may show rim arterial phase hyperenhancement (Fig. 5). The decreased enhancement in the dynamic study correlates with the histological degree of sclerosis [12, 13, 17]. These imaging changes may lead to a false positive diagnosis of LR-M (i.e., probably or definitely malignant but not HCC specific). Indeed, almost 6% of LR-M include benign lesions, and the analysis of prior imaging may be considered as a problem solving tool with lesion stability or decrease in size for at least 24 months favoring benignity, and unequivocal increase in lesion size (e.g., at least 50% before or at 6 months, 100% or greater increase in size after 6 months) favoring malignancy [1, 2].

## Malignant liver lesions

### Atypical size changes

Lesion growth remains one of the most concerning feature for malignancy. Threshold growth is currently regarded as one of the major imaging features for the definitive diagnosis of HCC according to LI-RADS, and American Association for the Study of Liver Diseases (AASLD) guidelines [1, 18, 19]. The LI-RADS definition of growth is based on an established "threshold" for the definitive diagnosis of HCC which has been modified and simplified over time. The latest LI-RADS v2018 definition—which includes a size increase of at least 50% in less than 6 months—is based on a median tumor volume doubling time of 178 days [1, 20]. However, threshold growth is not specific for HCC as it may also occur in 7.1% of non-HCC malignancies arising in cirrhosis [21].

Despite size increase is the most common scenario, spontaneous tumor regression has been described [22] (Fig. 8). The two major underlying mechanisms that may explain untreated HCC shrinkage include tumor hypoxia—caused by spontaneous hepatic artery or portal vein thrombosis, rapid tumor growth, hemodialysis, or massive gastrointestinal hemorrhage—and systemic immunological reactions which may inhibit tumor growth [5, 22].

### Internal and vascularization changes

The typical imaging features of HCC on contrast-enhanced CT and MRI are nonrim arterial phase hyperenhancement

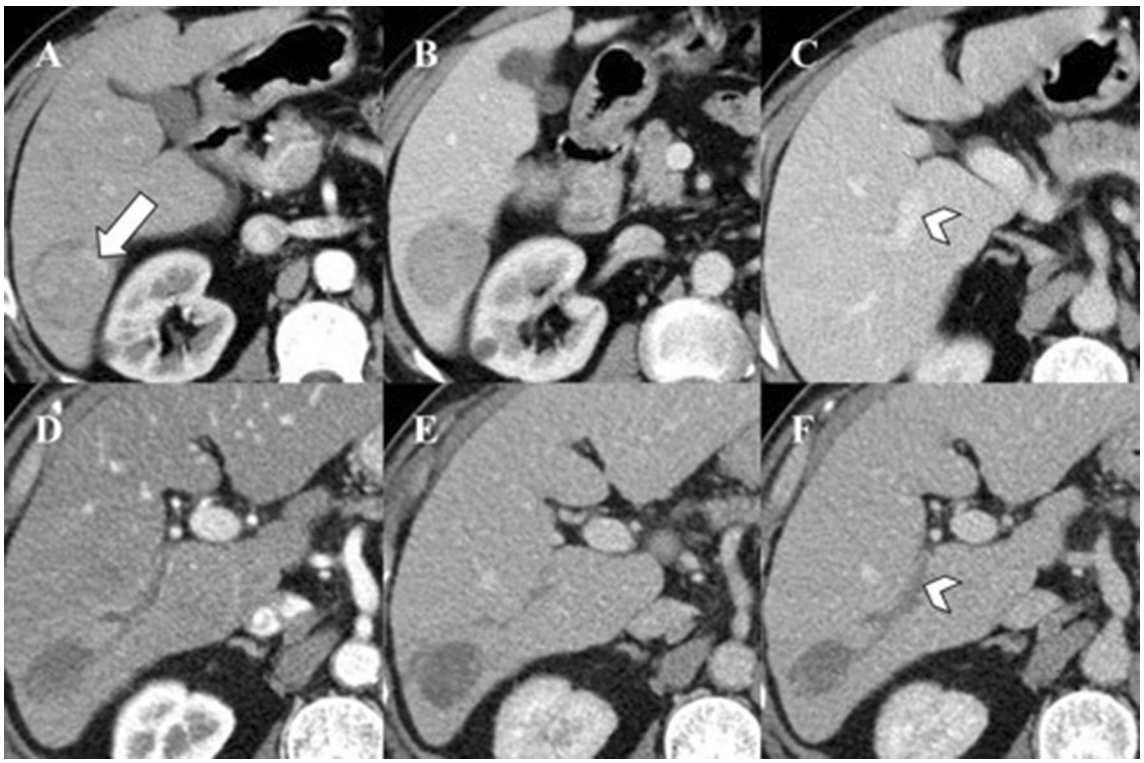


**Fig. 8** 56-year-old man with HCV cirrhosis. Baseline contrast-enhanced CT (upper row) shows a 4.3 cm lesion with arterial phase hyperenhancement (**a**, arrow) and washout on portal-venous phase (**b**). Note a patent right portal vein (**c**, arrowhead). At six-month CT follow-up (bottom row), without interval treatment, demonstrates spontaneous decrease in size of the lesion, with loss of arterial phase hyperenhancement (**d**) and hypoattenuation on portal-venous phase **e** with newly developed portal vein thrombosis (arrowhead) (**f**). Percutaneous biopsy of the lesion showed no evidence of viable HCC

and nonperipheral "washout" on portal-venous or delayed phases. The nonrim arterial phase hyperenhancement is due to the neoangiogenesis and formation of nontriadal or unpaired arteries in progressed HCC [23, 24]. The nonperipheral "washout" on portal-venous or delayed phases is likely the result of a combination of phenomena, including diminished portal-venous blood supply, high tumor cellularity with associated small extracellular volume, and expanded extracellular space of the surrounding cirrhotic parenchyma [20, 23–25]. These typical imaging features have to be differentiated from the rim arterial phase hyperenhancement and the peripheral "washout" more commonly encountered in non-HCC malignancies, including metastases, intrahepatic mass-forming cholangiocarcinoma, and combined hepatocellular-cholangiocarcinoma. Rim APHE and peripheral washout probably reflect the peripheral tumoral cellularity and central fibrous stroma or necrosis [26]. However, emerging data reported that up to 15% of overall non-HCC malignancies, and up to 50% of non-HCC malignancies smaller than 2 cm may show nonrim arterial phase hyperenhancement [21, 27, 28]. In our practice, we have occasionally noted liver observations initially showing a nonrim arterial phase hyperenhancement progressing to a rim arterial phase hyperenhancement at imaging follow-up (Fig. 9) and vice versa (Fig. 10). Although Tanabe et al [29] showed that 2%-5% of LR-4 may progress into LR-M—which includes rim arterial phase hyperenhancement lesions—no data exist in the literature on the evolution of nonrim arterial phase hyperenhancement into rim arterial phase hyperenhancement. We speculate that these changes in imaging presentation at contrast-enhanced CT/MR might be due to histologic changes within the lesion—i.e., necrosis, fibrosis—and/or to variable CT/MR acquisition protocol or timing.

## Unusual tumor recurrence

Intrahepatic recurrence of HCC occurs in 8–54% of cirrhotic patients after surgical resection with a median time of 22-32 months [30, 31], and in 10–45% following

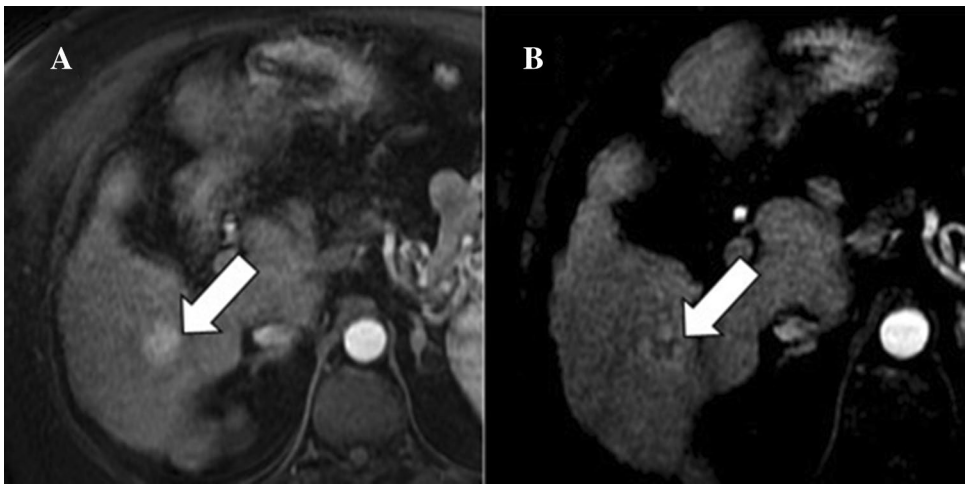


**Fig. 9** 58-year-old woman with HCV cirrhosis. **a** Baseline MRI on hepatic arterial phase shows a 2.0 cm lesion in segment 6 (arrow) with nonrim arterial phase hyperenhancement. At 3-month MRI follow-up **b** without interval treatment, the lesion (arrow) demonstrates rim arterial phase hyperenhancement and was classified as LR-M, highly concerning for malignancy but not typical for HCC

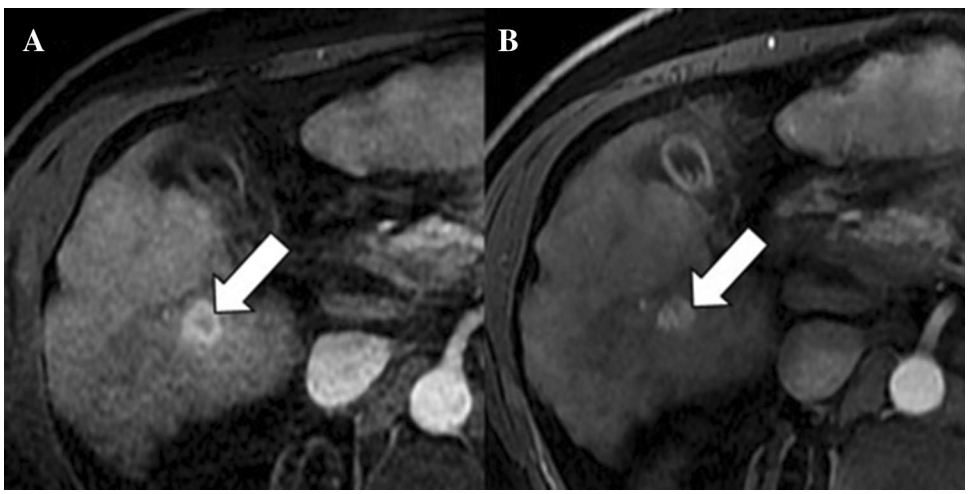


**Fig. 10** 62-year-old man with NASH cirrhosis. **a** Baseline MRI on hepatic arterial phase shows a 1.6 cm lesion (arrow) in segment 6 with rim arterial phase hyperenhancement. At 3-month MRI follow-up (**b**) without interval treatment, the lesion demonstrates nonrim arterial phase hyperenhancement (arrow). The lesion was a pathologically proven HCC

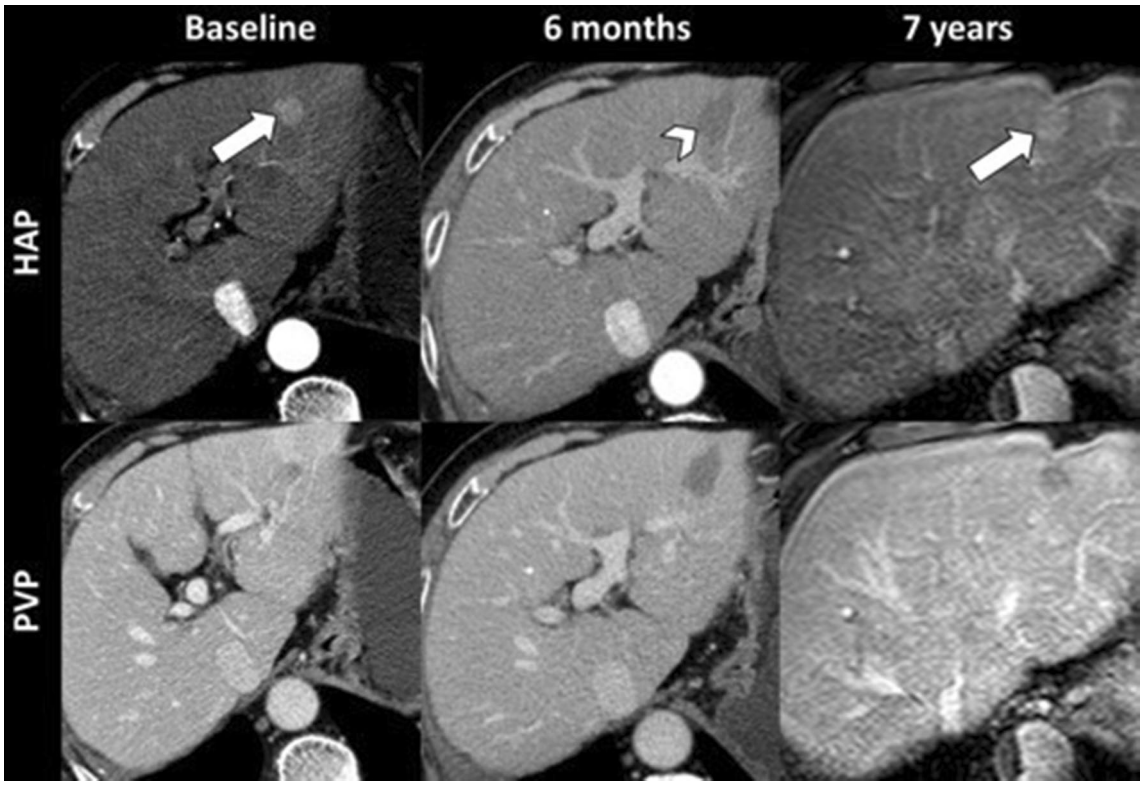


**Fig. 11** 83-year-old man with HCV cirrhosis. Baseline, 6-months and 7-years follow-up CT and MR images obtained on hepatic arterial (HAP, upper row) and portal-venous (PVP, bottom row) phase. Baseline CT demonstrated a nonrim arterial phase hyperenhancing lesion (arrow) with washout on portal-venous phase. The lesion underwent radiofrequency ablation with no evidence of local recurrence (arrowhead) at 6-month imaging follow-up. The 7-year MRI follow-up demonstrates nodular arterial phase hyperenhancement (arrow) at the ablation site compatible with tumor recurrence

locoregional treatments. The median tumor volume doubling time of recurrent HCC is 82 days [32–34]. Although HCC recurrence after radiofrequency thermal ablation usually develops within the first 3 years after treatment [32], we encountered local tumor recurrence more than 5 years following locoregional treatment (Fig. 11).

## Summary

In conclusion, we described benign and malignant lesions in cirrhosis with an uncommon evolution. Lesions showing unusual size change, loss of typical imaging features, and late tumor recurrence represent a diagnostic challenge. Knowledge of these scenarios may help the abdominal radiologist to reach a noninvasive diagnosis and direct the patient to the most appropriate clinical management.

### Compliance with ethical standards

**Disclosures** Federica Vernuccio, Roberto Cannella, Giorgia Porrello, Alberto Calandra, Massimo Midiri, Alessandro Furlan, and Giuseppe Brancatelli have no conflicts of interest to disclose for this study. Alessandro Furlan: Research Grant with General Electric; Elsevier/Amirsys author.

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