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Hepatoma Research

Gallo et al. Hepatoma Res. 2026;12:43 DOI:10.20517/2394-5079.2025.120

Pseudocirrhosis: pathophysiology, clinical mimics, and its role in hepatocellular carcinoma diagnosis and management

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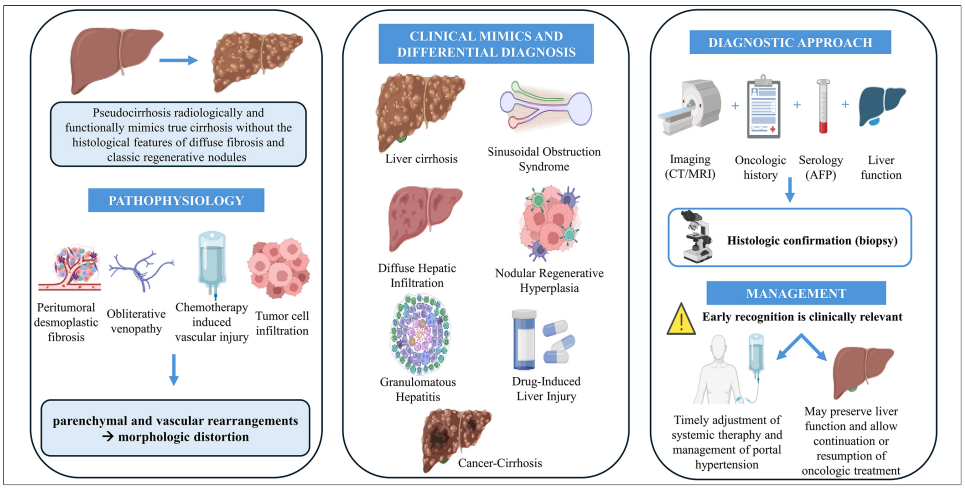
Keywords:
Pseudocirrhosis, occult metastatic disease, breast cancer, clinical mimics, hepatocellular carcinoma

Citation: Gallo P, Flagiello V, Di Pasquale G, Falcomatà A, Terracciani F, Mingiacchi A, Lerose F, Mastrostefano B, dell'Unto C, Galati G, Vespasiani-Gentilucci U, Picardi A. Pseudocirrhosis: pathophysiology, clinical mimics, and its role in hepatocellular carcinoma diagnosis and management. *Hepatoma Res.* 2026;12:43. https://dx.doi.org/10.20517/2394-5079.2025.120

Received: 31 Dec 2025 **First Decision:** 9 May 2026 **Revised:** 1 Jun 2026 **Accepted:** 25 Jun 2026 **Published:** 29 Jul 2026

Academic Editors: Huiguo Ding, Huiguo Ding **Copy Editor:** Ting-Ting Hu **Production Editor:** Ting-Ting Hu



Abstract

Pseudocirrhosis is an uncommon but clinically relevant condition that radiologically and functionally mimics true liver cirrhosis, despite the absence of the typical histological features of diffuse fibrosis and classic regenerative nodules. It most frequently occurs in patients with metastatic malignancies, particularly breast cancer, usually in the absence of known pre-existing chronic liver disease, and may progress rapidly, leading to morphologic liver distortion and clinically significant portal hypertension. Its pathophysiology is multifactorial, involving peritumoral desmoplastic fibrosis, chemotherapy-induced vascular injury with nodular regenerative hyperplasia and sinusoidal obstruction, as well as intra-sinusoidal or diffuse tumour cell infiltration with obliterative venopathy and endothelial injury. These mechanisms result in parenchymal and vascular rearrangements, often associated with portal hypertension, resembling those observed in chronic liver disease. Distinguishing pseudocirrhosis from similar conditions is critical to avoid misdiagnosis and inappropriate management. Several hepatic disorders share overlapping clinical and

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imaging findings, including vascular and granulomatous liver diseases, chronic drug-induced hepatotoxicity, and infiltrative malignancies. Accurate interpretation requires careful integration of oncologic history, treatment exposure, and specific imaging patterns, since standardized diagnostic criteria are lacking. One of the most insidious differential diagnoses is infiltrative hepatocellular carcinoma, which may exhibit diffuse parenchymal involvement, portal hypertension, and surface nodularity like pseudocirrhosis. When imaging is inconclusive and the diagnosis would influence clinical strategy, histologic confirmation is warranted, particularly when curative-intent or liver-directed therapies are being contemplated.

INTRODUCTION

Pseudocirrhosis is an uncommon but clinically relevant entity that radiologically and clinically mimics liver cirrhosis in the absence of diffuse fibrosis and classic regenerative nodules[1]. It is most frequently observed in patients with metastatic malignancies, particularly breast carcinoma, usually without known pre-existing chronic liver disease, and may evolve rapidly, leading to portal hypertension and liver failure. Historically, the term *hepar lobatum carcinomatosum* was introduced in 1924 to describe lobulated hepatic contours at autopsy in a woman with breast cancer[2]. Decades later, in 1994, Young *et al.* proposed the term *pseudocirrhosis* based on clinical and radiologic features that reproduce the cirrhosis-like liver appearance in oncologic contexts[3]. Although the exact mechanisms are not fully defined, several processes likely contribute, including sinusoidal or intra-sinusoidal neoplastic infiltration, portal venous thrombosis, perisinusoidal fibrosis, alterations of intrahepatic lymphatics, and nodular regeneration[4]. These changes can end in clinically significant portal hypertension with ascites, varices, and splenomegaly[5].

Pseudocirrhosis has been described primarily in metastatic breast cancer, with additional reports in pancreatic, thyroid, oesophageal, and gastroesophageal neoplasia[6-8]. Standardized diagnostic criteria are lacking, and diagnosis is predominantly imaging-based. Early recognition and close monitoring are essential to anticipate complications of portal hypertension, including variceal bleeding and hepatic encephalopathy, which are associated with poor clinical outcomes and reduced survival[9,10].

Unlike cirrhosis, pseudocirrhosis can progress over a short time window (approximately 1-3 months). Typical imaging findings include surface nodularity, capsular retraction, segmental volume loss, and caudate lobe hypertrophy[11-14]. The differential diagnosis is broad and includes, among others, vascular liver disease, granulomatous liver disease and chronic drug-induced hepatotoxicity[15].

Therefore, this narrative review aims not only to summarize the pathophysiology and imaging features of pseudocirrhosis, but also to propose a practical diagnostic framework for clinicians facing a cirrhosis-like liver morphology in oncologic patients, with particular attention to the differential diagnosis with true cirrhosis and infiltrative hepatocellular carcinoma (HCC).

PATHOPHYSIOLOGY

The pathophysiology of pseudocirrhosis is multifactorial and not yet fully elucidated. Neoplastic cells may infiltrate the liver via arterial or portal routes, forming capillary-like networks or directly obstructing hepatic sinusoids[16,17]. Although classically recognized in hematologic malignancies, diffuse intra-sinusoidal spread can also occur in solid tumours, particularly in poorly differentiated breast cancer[18].

Three non-mutually exclusive mechanisms are commonly proposed: desmoplastic retraction, fibrotic scarring, and capsular retraction occurred in response to neoplastic infiltration or as a consequence of effective cytotoxic therapy around regressed metastases; nodular regenerative hyperplasia (NRH) secondary to chemotherapy-related ischemia and sinusoidal injury; sinusoidal or venular obstruction due to microscopic neoplastic infiltration or chemotherapy-related vascular injury[14,19-22] [Figure 1].

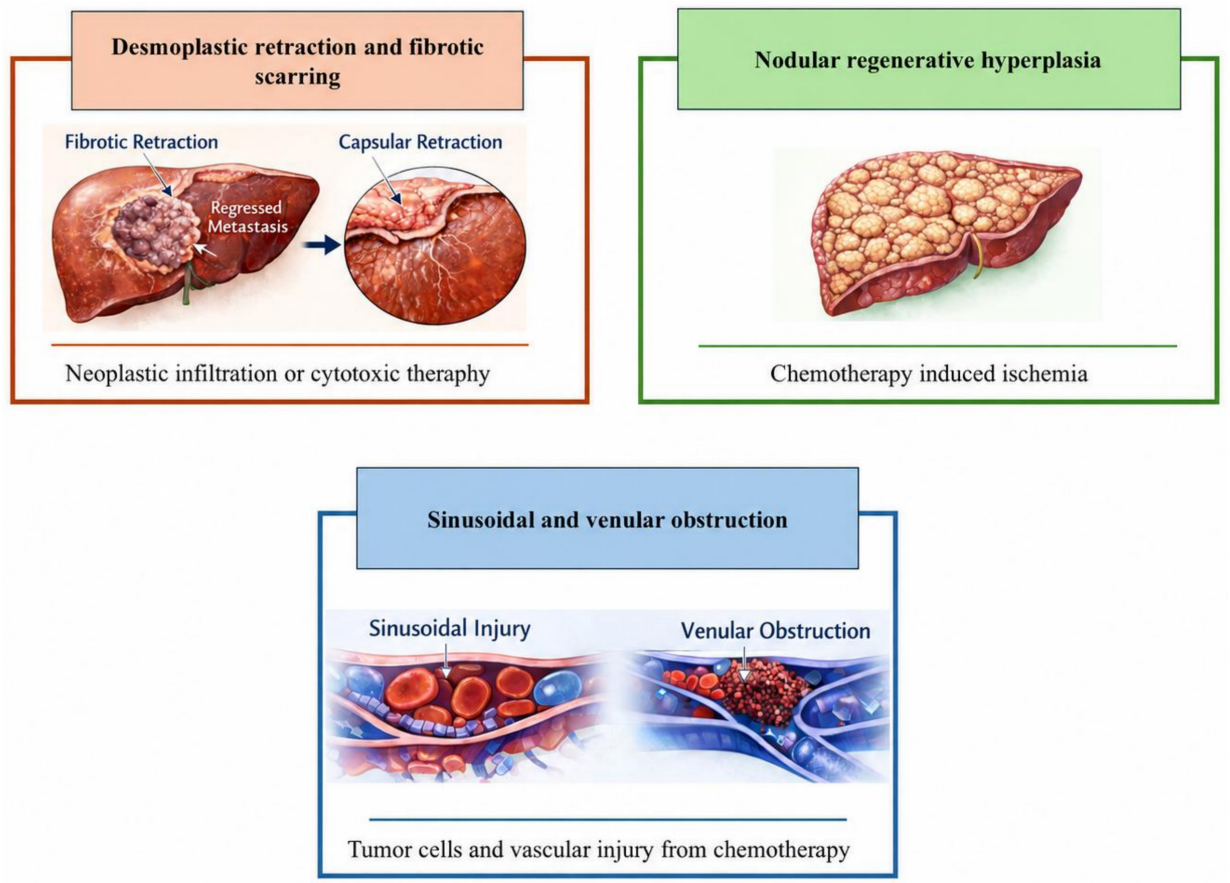

Figure 1. Schematic representation of the main mechanisms involved in pseudocirrhosis. Desmoplastic retraction and fibrotic scarring may occur after neoplastic infiltration or cytotoxic therapy, leading to capsular retraction and distortion of liver morphology. Chemotherapy-induced ischemic injury may promote nodular regenerative hyperplasia, while sinusoidal injury, venular obstruction, tumour cell infiltration, and chemotherapy-related vascular damage may contribute to intrahepatic vascular remodeling and portal hypertension. The figure was created in Biorender.com.

Peritumoral desmoplastic fibrosis is considered a major driver, representing a fibrotic stromal response to tumour infiltration that leads to capsular retraction, marked lobulation, and lobar atrophy, often predominant in the right lobe[11,23]. Hypoxia, tissue injury, and fibroblast activation, mediated by cytokines and growth factors from tumour cells or injured hepatocytes, promote collagen deposition in peritumoral regions[24,25].

NRH represents a compensatory response to subacute or chronic vascular injury and has been associated with systemic chemotherapy, chronic immunosuppressants (including thiopurines), or antiretroviral agents[26]. It is characterized histologically by diffuse small regenerative nodules in the absence of bridging fibrosis and can cause clinically relevant non-cirrhotic portal hypertension[5,15,20,27]. Finally, intrahepatic venous outflow impairment may arise from external compression or intra sinusoidal obstruction by metastases, mimicking aspects of Budd-Chiari pathophysiology[28]. For instance, in metastatic breast carcinoma, several reports have documented extensive sinusoidal dissemination of tumour cells with associated stromal desmoplasia[29], a pattern that is typically associated with rapid clinical deterioration and poor prognosis [Table 1]. Lastly, certain chemotherapeutic agents (e.g. oxaliplatin, taxanes) directly injure sinusoidal endothelium and contribute to the morphologic and functional alterations observed in pseudocirrhosis[19,40].

Table 1. Literature review of pseudocirrhosis cases in metastatic breast carcinoma with sinusoidal tumour cell infiltration and stromal desmoplasia: diagnostic approach and prognosis

First author	Diagnosis	Outcome from the admission
Borja *et al.* (1975)[30]	Histology: autopsy	Death in 10 days
Nascimento *et al.* (2001) - case 1[29]	Histology: transjugular liver biopsy	Death in 28 days
Nascimento *et al.* (2001) - case 2[29]	Histology: percutaneous liver biopsy	Death in 28 days
Mitchell *et al.* (2001)[31]	Histology: transjugular liver biopsy	Death in 21 days
Nakajima *et al.* (2005)[32]	Histology: laparoscopic liver biopsy	Death in few days
Fournier *et al.* (2010)[33]	Histology: transjugular liver biopsy	Unknown
Graber *et al.* (2010)[34]	Histology: transjugular liver biopsy	Unknown
Jüngst *et al.* (2013)[35]	Histology: transjugular liver biopsy	Death in 30 days
Hidalgo-Blanco *et al.* (2018)[36]	Histology: percutaneous liver biopsy	Death in 30 days
Knouse *et al.* (2019)[37]	Histology: transjugular liver biopsy	Death in a few days
Millard *et al.* (2019)[38]	Histology: percutaneous liver biopsy	Unknown
Hoshina *et al.* (2021)[39]	Histology: laparoscopic liver biopsy	Death in 30 days
Takata *et al.* (2022)[24]	Histology: percutaneous liver biopsy	Death in a few days

Diagnostic modality and short-term outcome from hospital admission are reported. "Unknown" indicates that the outcome was not available in the original publication.

CLINICAL MIMICS OF PSEUDOCIRRHOSIS

From a clinical standpoint, pseudocirrhosis should be approached as part of a stepwise differential diagnosis rather than as an isolated radiologic label. The first diagnostic question is whether the cirrhosis-like morphology reflects pre-existing or newly recognized chronic liver disease. In patients with established risk factors for HCC, infiltrative tumour must be actively excluded, particularly when alpha-fetoprotein (AFP) is markedly elevated. Conversely, pseudocirrhosis becomes more likely when a rapidly evolving nodular liver contour, capsular retraction, segmental volume loss, and portal hypertension develop in a patient with metastatic disease, especially after systemic therapy, in the absence of prior chronic liver disease and without typical HCC imaging features.

Several other clinical conditions may mimic pseudocirrhosis, both radiologically and functionally, despite having distinct aetiologies and pathophysiologic mechanisms. A proper differential diagnosis is essential to avoid misclassification and inappropriate management, particularly in oncologic patients [Figure 2 and Table 2].

Liver cirrhosis

Among all mimics, cirrhosis remains the most challenging to distinguish, as it most closely resembles pseudocirrhosis on imaging, showing lobulated contours, retractive changes, and signs of portal hypertension[4,11,13]. Moreover, liver dysfunction and clinically significant portal hypertension may occur in both entities, but in pseudocirrhosis they are often linked to tumour burden or therapeutic toxicity rather than to progressive hepatic architectural collapse[41]. Crucially, although cirrhosis can occasionally be undiagnosed, it is typically a known pre-existing condition, diagnosed based on clinical history and previous investigations, whereas pseudocirrhosis is usually identified at the time of diagnosis, often as a new and unexpected finding during oncologic follow-up.

Sinusoidal obstruction syndrome

Another important condition in the differential diagnosis of pseudocirrhosis is sinusoidal obstruction syndrome (SOS), also known as veno-occlusive disease. This entity most commonly arises because of chemotherapy-induced endothelial injury, particularly following oxaliplatin-based regimens in colorectal

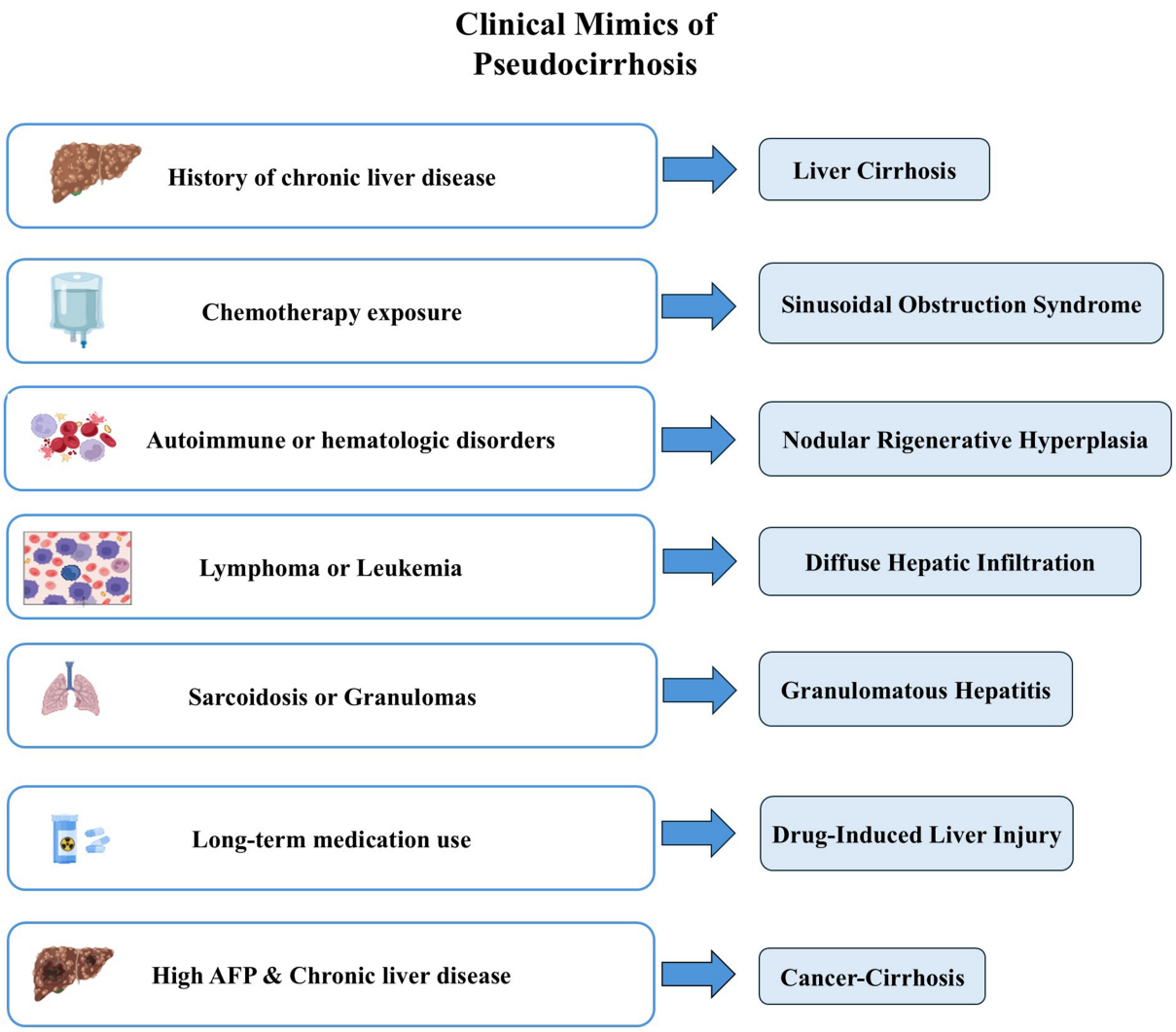

Figure 2. Clinical conditions that may mimic pseudocirrhosis according to the main clinical context or predisposing factor. Chronic liver disease may indicate true liver cirrhosis, chemotherapy exposure may suggest sinusoidal obstruction syndrome, autoimmune or hematologic disorders may be associated with nodular regenerative hyperplasia, and lymphoma or leukaemia may cause diffuse hepatic infiltration. Granulomatous disease, chronic drug exposure, and elevated AFP in patients with chronic liver disease should prompt consideration of granulomatous hepatitis, drug-induced liver injury, and Cancer-Cirrhosis, respectively. The figure was created in Biorender.com. AFP: Alpha-fetoprotein.

cancer or conditioning regimens in hematologic malignancies such as leukaemia and after hematopoietic stem cell transplantation[35,42-44]. Clinically, SOS may manifest with ascites, splenomegaly, and portal hypertension, features also frequently observed in pseudocirrhosis. However, SOS is not caused by metastatic infiltration, and it typically lacks imaging findings such as retractive changes or segmental volume loss, which are more characteristic of pseudocirrhosis. On imaging, the liver may appear enlarged, with heterogeneous enhancement and periportal oedema, rather than lobulated or atrophic[18]. Contrast-enhanced ultrasound (CEUS) has been explored in the evaluation of SOS. Some studies have reported that CEUS can detect diffuse or geographic enhancement patterns in the hepatic parenchyma, with scattered hypoechoic areas[45]. However, these findings are not specific, and CEUS remains of limited diagnostic value in differentiating SOS from pseudocirrhosis, as both conditions may lack distinctive enhancement patterns or washout[45].

Table 2. Differential diagnosis between pseudocirrhosis and its major clinical and radiologic mimics

Condition	Typical clinical setting	Key imaging findings	Portal hypertension	Laboratory clues	Distinguishing features
Pseudocirrhosis[1,11-14,19-22,41]	Metastatic malignancy with liver involvement, with or without systemic treatment	Capsular retraction, lobulated contour, segmental volume loss, caudate hypertrophy, rapid morphologic evolution	Frequent and often rapidly progressive	Variable liver dysfunction, thrombocytopenia, and tumour-specific biomarker abnormalities	Cirrhosis-like morphology in the absence of diffuse bridging fibrosis and classic regenerative nodules, possible association with desmoplastic fibrosis, sinusoidal tumour infiltration, and chemotherapy-related vascular injury
Liver cirrhosis[4,11,13]	Known chronic liver disease, recognized liver disease risk factors, or newly diagnosed chronic liver disease	Diffuse nodular contour, regenerative nodules, volume redistribution with caudate hypertrophy, slowly progressive morphologic remodeling	Typical and slowly progressive	Variable liver dysfunction and thrombocytopenia	Established cirrhotic architecture with bridging fibrosis and regenerative nodules
Sinusoidal obstruction syndrome[18,28,42-45]	Oxaliplatin-based chemotherapy, hematopoietic stem cell transplantation, or hematologic malignancies	Hepatomegaly, heterogeneous enhancement, periportal oedema, usually without capsular retraction or segmental volume loss	Frequent and potentially acute	Cholestatic liver test abnormalities, thrombocytopenia, and variable liver dysfunction	Sinusoidal endothelial injury with congestion and venular obliteration in the absence of metastatic infiltration
Nodular regenerative hyperplasia[26,46-51]	Autoimmune disease, hematologic disorders, immunosuppressive or cytotoxic therapy, and chemotherapy exposure	Mild surface nodularity, usually without marked lobar distortion or capsular retraction	Possible and usually non-cirrhotic	Usually preserved liver synthetic function with possible thrombocytopenia	Diffuse micronodular regenerative transformation in the absence of bridging fibrosis or fibrous septa
Infiltrative haematological malignancies[32,34,38,52]	Lymphoma, leukaemia, myeloproliferative disorders	Diffuse hepatic infiltration, hepatomegaly, miliary nodules, usually without segmental atrophy or capsular retraction	Possible	Elevated LDH, cytopenias, systemic inflammatory markers	Diffuse sinusoidal or portal infiltration by malignant haematologic cells
Granulomatous liver disease[53-56]	Sarcoidosis and other granulomatous disorders	Coarse nodular hepatic echotexture, parenchymal heterogeneity, without marked retractive changes, lobar collapse, or segmental volume loss	Possible in advanced disease	Elevated ACE, cholestatic liver test abnormalities	Non-caseating granulomas or granulomatous inflammation
Drug-induced liver injury[11,21,41,57]	Long-term exposure to methotrexate, tamoxifen, antiretroviral agents, or other hepatotoxic drugs	Diffuse steatosis, periportal changes, or chronic fibrotic remodeling, usually without segmental remodeling, capsular retraction, or lobar volume loss	Possible in advanced disease	Variable liver dysfunction according to injury pattern	Steatosis, steatohepatitis, pericellular fibrosis, vascular injury, depending on the causative agent

Infiltrative hepatocellular carcinoma[58-65]	Usually advanced chronic liver disease or cirrhosis	Diffuse or permeative infiltration, subtle or patchy arterial enhancement, heterogeneous washout, hepatobiliary phase hypointensity, and portal vein tumour thrombosis	Frequent and often progressive	AFP often markedly elevated with variable liver dysfunction	Diffuse malignant hepatocellular infiltration with vascular invasion

Major clinical and radiologic mimics of pseudocirrhosis and their distinguishing features. Although several conditions may share imaging findings such as nodular liver contour, portal hypertension, or parenchymal heterogeneity, differences in clinical setting, enhancement patterns, laboratory findings, and histopathology may support the differential diagnosis. ACE: Angiotensin-converting enzyme; AFP: alpha-fetoprotein; LDH: lactate dehydrogenase.

Nodular regenerative hyperplasia

Although NRH may also be encountered in the setting of pseudocirrhosis, it is a distinct clinicopathologic entity that can arise across a broad spectrum of other systemic disorders and itself mimic pseudocirrhosis, thereby entering the differential diagnosis.

NRH has been associated with a range of systemic conditions, including hematologic disorders[46], autoimmune diseases[47], long-term exposure to immunosuppressive and cytotoxic agents (such as azathioprine or 6-thioguanine)[41] and to chemotherapy for solid tumours and hematologic malignancies[41,48]. Histologically, NRH is defined by diffuse transformation into small regenerative nodules without fibrous septa, clearly distinguishing it from both cirrhosis and pseudocirrhosis[49]. Clinically, it may remain silent or manifest with non-cirrhotic portal hypertension, including splenomegaly or variceal bleeding[49]. When present, imaging abnormalities are typically mild and lack distinguishing features (such as marked surface irregularity or lobar volume loss), thus making NRH difficult to identify radiologically without clinical suspicion[50]. Surface nodularity and signs of portal hypertension may be observed, but there is no segmental volume loss, caudate hypertrophy, or lobular contour retraction, which are more typical of pseudocirrhosis. On contrast-enhanced studies, NRH usually demonstrates homogeneous or mildly heterogeneous parenchymal enhancement, without nodular collapse or retractive changes[49,50]. CEUS has been reported to show non-specific findings in NRH, and nodules are often not visualized or not differentiable from background liver tissue[51]. To date, no characteristic enhancement pattern has been validated, and CEUS remains of limited utility in diagnosing NRH[51].

Infiltrative haematological malignancies

Infiltrative hematologic malignancies, such as lymphoma and leukaemia, can diffusely involve the liver parenchyma without forming discrete masses, leading to hepatomegaly, irregular hepatic contours, and portal hypertension[38]. These may present radiologically as diffuse hepatic infiltration, homogeneous enlargement, or a miliary pattern of multiple small nodules, particularly in lymphoma[38]. However, these infiltrative patterns generally lack the lobar volume loss, contour retraction, or post-treatment fibrosis that characterize pseudocirrhosis[52]. Hepatic morphology is often preserved in early stages, and diagnosis relies on clinical and laboratory findings and evidence of underlying hematologic disease[32,34].

Granulomatous disease

Hepatic granulomatous diseases, including sarcoidosis, may also mimic pseudocirrhosis through chronic lobular inflammation, periportal granuloma formation, and secondary fibrosis[53,54]. These conditions can present with portal hypertension, capsular surface irregularities, and distortion of liver morphology[53,55]. However, they typically do not exhibit segmental volume loss, caudate lobe hypertrophy, or parenchymal collapse, which are more typical of pseudocirrhosis secondary to metastatic disease or chemotherapy[3,11,12].

Imaging may show coarse nodular hepatic echotexture and signs of portal hypertension but lacks the retractive features or treated-lesion sequelae of pseudocirrhosis[11,13]. Final diagnosis often requires correlation with clinical features, laboratory markers and, in selected cases, histological confirmation through liver biopsy[36,56].

Drug-induced liver injury

In this context, drug-induced liver injury (DILI) should enter the differential diagnosis of pseudocirrhosis only when it results in insidious, chronic architectural damage, effectively representing an evolving cirrhotic process that may be clinically unexpected. This scenario deeply differs from the predominantly vascular, reactive mechanism of pseudocirrhosis, in which chemotherapy-related sinusoidal injury and NRH underlie the cirrhosis-like hepatic remodelling. DILI may occur in patients exposed to long-term treatment. Agents most frequently implicated include methotrexate, tamoxifen, and certain antiretrovirals, which can induce diffuse architectural remodelling or periportal fibrosis, simulating some features of pseudocirrhosis[41]. Methotrexate, widely used in autoimmune diseases and malignancies, has been associated with hepatic steatosis, fibrosis, and, in some cases, cirrhosis[57]. Histologically, it may cause macrovesicular steatosis, hepatocyte ballooning, and pericellular fibrosis[11,57]. Tamoxifen, a selective estrogen receptor modulator used in breast cancer, has been associated with the development of metabolic-associated steatotic liver disease (MASLD) and, in some cases, with progression to steatohepatitis and fibrosis. Imaging typically shows diffuse hepatic steatosis without lobular distortion, surface nodularity, or lobar atrophy, and therefore lacks the morphologic features that would raise suspicion for pseudocirrhosis; however, if drug-induced liver disease silently progresses to steatohepatitis and advanced fibrosis, it may eventually enter the differential diagnosis as an insidious, evolving cirrhotic process[21]. Antiretroviral agents, particularly didanosine and stavudine, have been linked to mitochondrial toxicity, hepatic steatosis, and rare cases of fibrosis[49]. Imaging findings may include diffuse steatosis or periportal changes, but without the hallmark features of pseudocirrhosis such as retractive changes, surface irregularity, or segmental volume loss[11].

In all these scenarios of DILI, the parenchymal changes tend to be diffuse and evenly distributed, without the segmental remodelling and volume distortion typical of pseudocirrhosis. These features, together with a suggestive pharmacologic history, aid in distinguishing drug-induced hepatotoxicity from pseudocirrhosis[11].

PSEUDOCIRRHOSIS AND HCC

HCC, the most common primary malignancy of the liver, typically arises in the context of chronic liver disease and cirrhosis. The nodular and massive growth patterns of HCC are usually radiologically remarkable and often fulfill classic imaging criteria on dynamic contrast-enhanced computed tomography or magnetic resonance imaging (MRI). Conversely, the infiltrative subtype of HCC, accounting for 7%-20% of all cases, is notoriously difficult to detect and may present as a diffuse alteration of the hepatic parenchyma without a dominant lesion[58]. This growth pattern can involve entire lobes or even the whole liver and is often accompanied by portal vein tumour thrombosis and aggressive biological behaviour[15,59,60].

Thus, the presence of HCC risk factors changes the diagnostic hierarchy. In a patient with chronic liver disease or cirrhosis, a new diffuse hepatic distortion should not be attributed to pseudocirrhosis until infiltrative HCC has been reasonably excluded. The diagnostic intersection between these two entities represents a critical clinical scenario where imaging features, oncologic history, and liver function all converge to guide decision-making. From a radiologic point of view, infiltrative HCC tends to show subtle or patchy arterial enhancement, often with heterogeneous washout and hypo-intensity in the hepatobiliary phase, particularly when using hepatobiliary contrast agents[61,62]. Diffusion-weighted MRI may increase lesion detectability in these cases[63]. In particular, hepatobiliary contrast-enhanced MRI may improve lesion conspicuity in infiltrative HCC, especially in patients with diffuse or heterogeneous parenchymal distortion.

These features, however, are not pathognomonic, and similar findings can be seen in cases of pseudocirrhosis with underlying diffuse metastatic disease. Importantly, while infiltrative HCC and pseudocirrhosis may both present with nodular liver contour, capsular retraction, segmental atrophy, and signs of portal hypertension, they arise from deeply different processes[3,14,35].

Serologic markers, especially AFP, have limited diagnostic accuracy and are no longer recommended as stand-alone tests for the diagnosis of HCC in major international guidelines. When AFP is used as a diagnostic marker, low cut-offs (e.g. 20 ng/mL) provide only moderate sensitivity but poor specificity, whereas higher thresholds of 200-400 ng/mL markedly increase specificity at the expense of very low sensitivity. In most series, AFP values ≥ 200-400 ng/mL have shown specificities close to or above 95%-99% for HCC in patients with chronic liver disease[59,64,65]. Despite these limitations, very high AFP concentrations often carry important diagnostic information. Markedly elevated AFP values, in the hundreds or thousands of ng/mL, are strongly associated with advanced, biologically aggressive HCC and with features such as large tumour burden and portal vein invasion[66,67]. In particular, infiltrative HCC frequently presents with elevated AFP levels, and published series report median values in the several-hundred ng/mL range, with a substantial proportion of patients exceeding conventional diagnostic thresholds[59]. By contrast, in pseudocirrhosis related to metastatic breast cancer or other non-AFP-producing primaries, AFP levels are typically normal or only mildly elevated. Thus, while normal AFP does not exclude infiltrative HCC, a strikingly elevated AFP level in a cirrhosis-like liver should strongly favour the diagnosis of HCC over pseudocirrhosis in the appropriate clinical context[58].

PROPOSED DIAGNOSTIC APPROACH TO PSEUDOCIRRHOSIS

Clinicians should suspect pseudocirrhosis when a patient with metastatic cancer, particularly with liver involvement, develops a rapid change in hepatic morphology characterized by surface nodularity, capsular retraction, segmental volume loss, caudate lobe hypertrophy, and signs of portal hypertension. The suspicion is strengthened by the absence of previous chronic liver disease, a close temporal relationship with systemic therapy, and discordance between improving or stable tumour burden and worsening portal hypertensive features. Serial comparison with prior imaging is essential, because rapid evolution over weeks to a few months is one of the most useful clues distinguishing pseudocirrhosis from conventional cirrhosis.

Distinguishing between infiltrative HCC and pseudocirrhosis can be challenging; a structured diagnostic algorithm can help guide the stepwise evaluation and multidisciplinary decision-making process in this context [Figure 3].

Obviously, the key starting point is the patient's medical history. If a patient with known metastatic disease develops a liver morphology resembling cirrhosis, especially after systemic therapy, the diagnosis of pseudocirrhosis should be favored if the radiological changes are rapid, AFP is normal or only slightly elevated, and no dominant lesions, tumor-in-vein, or typical enhancement patterns are identified. Conversely, infiltrative HCC should be strongly considered when parenchymal distortion is associated with markedly elevated or progressively rising AFP levels, hyperenhancement in the arterial phase, hypointensity in the hepatobiliary phase, diffusion restriction, tumor thrombosis of the portal vein, but especially a history of chronic liver disease.

In this context, a multimodal approach is essential. Histologic confirmation via biopsy should be considered when imaging is inconclusive, when tumour markers and imaging are discordant, when occult sinusoidal metastasis is suspected despite apparently negative imaging, or when the result would change oncologic management. Infiltrative HCC, due to its aggressive nature and poor prognosis, in addition to the frequently observed associated chronic liver disease, is typically not amenable to curative treatment. Pseudocirrhosis,

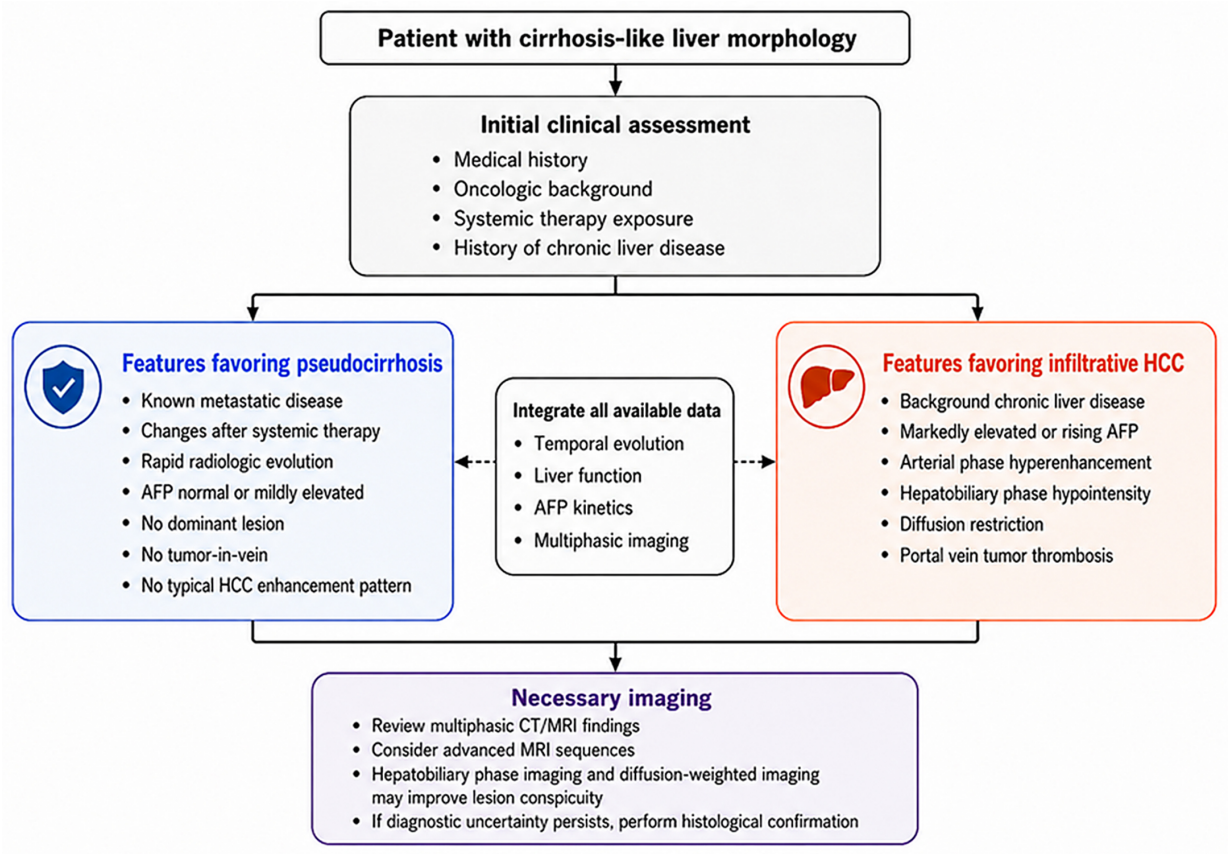

Figure 3. Diagnostic approach to pseudocirrhosis *vs.* infiltrative HCC. The figure was created in Biorender.com. AFP: Alpha-fetoprotein; CT: computed tomography; HCC: hepatocellular carcinoma; MRI: magnetic resonance imaging.

although potentially associated with signs of portal hypertension and hepatic failure, does not always carry the same oncologic implications[3,68]. In patients with ascites, portal hypertension, coagulopathy, or thrombocytopenia, the transjugular route should generally be preferred. Thus, multidisciplinary discussion involving hepatologists, oncologists, radiologists, and pathologists is recommended before discontinuing potentially effective antitumour therapy or before assigning the patient to best supportive care.

MANAGEMENT AND TREATMENT OF PSEUDOCIRRHOSIS

There is no disease-specific treatment for pseudocirrhosis, and management should be individualized according to the dominant mechanism, severity of portal hypertension, liver function, and oncologic status. When chemotherapy-related sinusoidal injury, NRH, or treatment-induced desmoplastic retraction is suspected, temporary interruption, dose reduction, or modification of the implicated systemic therapy should be discussed. Supportive treatment should follow the principles used for portal hypertension and be individualized according to liver function, tumour burden, and life expectancy.

In highly selected patients with symptomatic malignant pseudocirrhosis and refractory portal hypertension, transjugular intrahepatic portosystemic shunt (TIPS) has been reported as a feasible palliative option, although evidence remains limited and patient selection should be multidisciplinary[69].

The prognosis becomes particularly poor when pseudocirrhosis is complicated by clinically significant portal hypertension. In a systematic review and meta-analysis including 389 patients[70], portal hypertension was observed in most patients reported in case reports and case series, with ascites and oesophageal varices being

the most frequent manifestations; the median time from pseudocirrhosis diagnosis to death was only 2 months in case reports and case series, whereas observational studies reported median times to death ranging from 3.6 to 8.5 months. Contemporary breast cancer cohorts confirm this adverse prognosis, with median overall survival after pseudocirrhosis diagnosis of approximately 7.6-7.9 months[19]. The presence of clinically significant portal hypertension may further identify a subgroup with particularly poor outcomes: in one cohort, patients with endoscopically evident oesophageal or gastric varices had a median overall survival of 5 months, compared with 13 months in those without varices, and median survival after documentation of varices was only 2 months[71]. However, these estimates should be interpreted cautiously because survival is strongly influenced by tumour subtype, tumour burden, liver function, availability of further oncologic therapy, and heterogeneous definitions of portal hypertension across studies.

Although pseudocirrhosis is often associated with poor prognosis, partial or complete regression has been reported in selected cases, especially when the process is driven by treatment-related injury or scarring around regressed metastases rather than uncontrolled diffuse tumour infiltration[6]. Therefore, early recognition is clinically relevant because timely adjustment of systemic therapy and management of portal hypertension may preserve liver function and allow continuation or resumption of oncologic treatment.

CONCLUSIONS

Pseudocirrhosis is often a rapidly progressive condition with a poor prognosis, reflecting both the underlying malignancy and treatment-related liver injury. Early recognition allows for closer monitoring of hepatic function, timely adjustment or withdrawal of potentially hepatotoxic systemic therapies, and earlier institution of supportive measures before overt hepatic failure precludes further oncologic options. Supportive care includes management of ascites, endoscopic treatment of varices, and management of hepatic encephalopathy, while in severe cases advanced interventions such as TIPS may be considered for refractory portal hypertension. In selected patients, partial regression of the pseudo-cirrhotic morphology after modification or discontinuation of systemic therapy has been reported, but this appears to be the exception rather than the rule.

At the same time, in patients with a cirrhosis-like liver and equivocal imaging findings, early histologic confirmation plays a pivotal role. Indeed, only a definite diagnosis reached by liver histology can safely justify active tumour-directed treatment, including rescue chemotherapy regimens, before progressive liver failure irreversibly limits these options.

Finally, current evidence on pseudocirrhosis remains weak and largely based on case reports, small retrospective cohorts, and heterogeneous systematic reviews. Diagnostic definitions vary across studies, histologic confirmation is available only in a minority of cases, and the relative contribution of different pathophysiological mechanisms is often difficult to identify. These limitations currently prevent the development of validated diagnostic criteria or evidence-based management algorithms. Future studies should aim to establish prospective multicentre registries with standardized imaging criteria, systematic comparison with previous imaging, predefined definitions of portal hypertension, integrated radiology-pathology correlation whenever biopsy is feasible and particular attention to modern systemic therapies.

DECLARATIONS

Acknowledgments

The graphical abstract was created in Biorender.com.

Authors' contributions

Conceptualized the study and drafted the manuscript: Gallo P, Flagiello V, Di Pasquale G, Falcomatà A
Contributed to drafting the manuscript: Terracciani F, Mingiacchi A, Lerose F, Mastrostefano B, dell'Unto C, Galati G

Supervised the study and critically revised the manuscript for important intellectual content: Picardi A, Vespasiani-Gentilucci U
All authors read and approved the final manuscript.

Availability of data and materials

Not applicable.

AI and AI-assisted tools statement

During the preparation of this manuscript, the AI tool ChatGPT (version GPT-5.5 Thinking, released 2026-04-23) was used solely for figure editing. The tool did not influence the study design, data collection, analysis, interpretation, or the scientific content of the work. All authors take full responsibility for the accuracy, integrity, and final content of the manuscript.

Financial support and sponsorship

None.

Conflicts of interest

All authors declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

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