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ABDOMINAL IMAGING

REVIEW

Diagnostic pearls and pitfalls in the radiologic evaluation of hepatic alveolar echinococcosis

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ABSTRACT

Hepatic alveolar echinococcosis is a tumor-mimicking parasitic disease characterized by infiltrative hepatic involvement and substantial radiologic overlap with malignancy. Predominantly solid morphology, pseudo-capsular margins, and deceptive enhancement patterns frequently contribute to misclassification as primary or secondary liver cancer. Intralesional calcifications on non-contrast computed tomography (CT) and the absence of true arterial phase hyperenhancement on CT and magnetic resonance imaging represent important diagnostic clues. Diffusion-weighted imaging may further aid characterization, although overlap with malignant lesions persists. Accurate interpretation requires systematic multimodality imaging assessment integrated with clinical and serologic context. Increased awareness of characteristic imaging features and common pitfalls is essential to reduce diagnostic delay and prevent inappropriate oncologic management.

KEYWORDS

Alveolar echinococcosis, *Echinococcus multilocularis*, liver, ultrasonography, computed tomography, magnetic resonance imaging

Hepatic alveolar echinococcosis (AE) is an uncommon yet severe parasitic infection caused by the larval stage of the fox tapeworm *Echinococcus multilocularis*.[1] In humans, proliferation of metacestode tissue occurs predominantly within the liver, resulting in a disease characterized by slow progression, invasive behavior, and a growth pattern that closely resembles that of malignant tumors.[2] In contrast to cystic echinococcosis, hepatic AE does not develop a distinct capsule; instead, lesions tend to spread insidiously into the surrounding hepatic parenchyma as well as biliary and vascular structures.[3]

Because of this infiltrative growth pattern, hepatic AE frequently imitates primary or secondary hepatic malignancies on radiologic imaging.[4] As the disease advances, parasitic tissue may extend into adjacent organs or, less commonly, give rise to distant involvement.[5,6] The heterogeneity and lack of specificity of its imaging manifestations further complicate diagnosis, particularly for radiologists practicing in non-endemic regions where clinical suspicion for parasitic disease is low.[3]

The prognosis is generally poor, and diagnostic delays may predispose to the development of this unfavorable clinical course.[7] In advanced cases, the emergence of inoperable lesions and chronic liver failure may lead patients to the point of requiring hepatic transplantation, thereby further complicating clinical management.[2] Errors at the initial radiologic assessment may not only postpone correct diagnosis but also lead to erroneous classification as hepatic malignancy, exposing patients to unwarranted invasive procedures and inappropriate oncologic therapies.[8] Accordingly, this review focuses on key diagnostic pearls and recurrent pitfalls in the radiologic evaluation of hepatic AE, with emphasis on imaging features that most commonly contribute to misinterpretation as malignant disease.

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Disease staging and imaging-based classification in hepatic alveolar echinococcosis

Rationale for staging in alveolar echinococcosis

AE is a chronic and potentially life-threatening parasitic infection resulting from hepatic colonization by the metacestode form of *Echinococcus multilocularis*. The disease is primarily endemic in temperate regions of the Northern Hemisphere, including selected areas of Europe, Central Asia, Western China, and North America. Human infection occurs accidentally through ingestion of parasite eggs eliminated by definitive hosts, most commonly wild canids and domestic dogs. Although its incidence in humans remains relatively low, AE carries substantial clinical relevance due to its insidious progression and destructive hepatic behavior.[9]

The natural course of AE is marked by a prolonged latent phase, often spanning years, during which parasitic proliferation occurs silently within the liver. Once symptomatic, the condition frequently mimics primary hepatic malignancies both clinically and radiologically. Pathologically, AE is distinguished from cystic echinococcosis by the absence of a unilocular cystic structure. Instead, it consists of multiple small vesicular components embedded within a dense fibroinflammatory matrix, demonstrating infiltrative expansion into adjacent parenchyma, vascular structures, and biliary ducts. This tumor-like growth pattern underlies its locally aggressive character and potential for distant spread.[1,9,10]

Main points

- Hepatic alveolar echinococcosis (AE) demonstrates infiltrative growth along biliary and vascular structures rather than forming a discrete expansile mass.
- A predominantly solid appearance and pseudo-capsular margins frequently contribute to misinterpretation as hepatic malignancy.
- Intralesional calcifications detected on non-contrast computed tomography (CT) represent a critical diagnostic anchor in infiltrative hepatic lesions.
- Hepatic AE lacks true arterial phase hyperenhancement on contrast-enhanced CT and magnetic resonance imaging; peripheral enhancement typically reflects fibroinflammatory reaction.
- Accurate diagnosis requires multimodality imaging assessment integrated with clinical and serologic context to avoid inappropriate oncologic management.

Diagnosis relies on a multimodal approach integrating cross-sectional imaging and serological analysis. Although contrast-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) delineate lesion morphology and extent, serological assays, such as enzyme-linked immunoabsorbent assay (ELISA) and confirmatory immunoblot techniques targeting specific antigens (e.g., Em2, Em18), enhance diagnostic specificity and are useful in therapeutic monitoring. Early recognition, radical surgical resection when achievable, and long-term benzimidazole therapy remain the cornerstone of management, significantly influencing prognosis.[1,10]

Hepatic AE demonstrates slow but progressive growth with an infiltrative pattern that resembles malignant disease. Rather than forming a discrete mass, the lesion spreads along biliary and vascular structures and gradually replaces normal hepatic parenchyma. This growth behavior explains the frequent involvement of critical intrahepatic structures and the potential for extrahepatic extension.[11,12]

Given this malignant-like progression, accurate assessment of disease extent is central to patient management. The degree of hepatic involvement and the presence of extrahepatic spread directly influence prognosis and therapeutic strategies, including surgical resection and liver transplantation. In this context, staging provides a standardized framework for describing disease distribution and facilitating multidisciplinary decision-making.[13,14]

From a radiologic standpoint, staging supports systematic evaluation of anatomic extent but does not fully reflect the variability of imaging appearances encountered in practice. Lesions with similar extent may exhibit different morphologic features, some of which closely mimic hepatic malignancy and contribute to diagnostic uncertainty.[15]

The "parasitic mass in the liver, neighboring organ involvement, and metastasis" classification system

The "parasitic mass in the liver, neighboring organ involvement, and metastasis" (PNM) classification system is the most commonly used staging framework for hepatic AE. Adapted from oncologic staging models, it categorizes disease based on hepatic involvement (P), invasion of neighboring structures (N), and the presence of distant metastases (M).[16]

In clinical practice, the PNM system provides a standardized approach for describing disease extent and guiding management decisions. Cross-sectional imaging is essential for assigning PNM stage by enabling the evaluation of lesion distribution, involvement of vascular and biliary structures, and detection of extrahepatic disease.[17]

Despite its clinical utility, PNM staging primarily reflects anatomic extent and does not account for morphologic heterogeneity or lesion biology. Consequently, lesions within the same PNM stage may show diverse imaging appearances, limiting the ability of staging alone to explain radiologic variability or prevent misinterpretation as hepatic malignancy.[18,19]

Imaging-based morphologic classifications and their limitations

Multiple imaging-based classification systems have been introduced to describe the wide morphologic variability of hepatic AE. Unlike staging systems that primarily address the anatomic extent of disease, these classifications focus on lesion morphology and internal architecture as depicted on cross-sectional imaging, including solid components, cystic areas, fibrosis, necrosis, and calcifications.[20,21]

A commonly applied morphologic framework divides hepatic AE into five imaging-based types. Type 1 lesions are characterized by clusters of small cysts without a solid component. Type 2 lesions show a combination of solid tissue and multiple small cysts. Type 3 lesions, representing the most prevalent pattern, consist of solid tissue associated with large, irregular cysts. Type 4 lesions appear as purely solid masses without cystic elements, and type 5 lesions present as a single large cyst without accompanying solid tissue.[20]

MRI-oriented classifications tend to emphasize multilocular or honeycomb-like internal patterns, whereas CT–based systems primarily focus on the extent of calcification and infiltrative lesion margins. These morphologic descriptions support standardized reporting and improve communication of imaging findings among clinicians.[18]

However, morphology-based classifications have inherent limitations. Considerable overlap exists between categories, and lesions with similar imaging appearances may demonstrate different biological behavior. Moreover, several morphologic patterns closely resemble primary or secondary he-

patic malignancies, particularly when lesions are predominantly solid or poorly marginated. Consequently, reliance on morphologic classification alone may be insufficient for confident diagnosis in routine radiologic practice.[15,19]

Overall, staging systems and morphologic classifications provide a valuable conceptual framework for hepatic AE. Nevertheless, accurate diagnosis and assessment ultimately depend on a comprehensive evaluation using multimodality imaging. The following section summarizes the characteristic ultrasonographic, computed tomographic, and MRI features of this entity (Table 1).

Multimodality imaging features of hepatic alveolar echinococcosis

Ultrasonography

Ultrasonography is frequently the first imaging modality used in patients presenting with non-specific hepatic symptoms or incidentally detected liver lesions, and it also represents the initial imaging step in the diagnostic evaluation of hepatic AE.[22,23] In hepatic AE, ultrasonographic findings are variable and largely non-specific, often reflecting the complex histopathologic composition of the lesion. Lesions typically appear as heterogeneous masses with irregular or ill-defined margins, corresponding to the coexistence of solid, necrotic, and microcystic components. Hypoechoic areas usually represent necrosis or microcystic structures, whereas hyperechoic foci with posterior acoustic shadowing are indicative of intralesional calcifications.[8,24]

On color Doppler ultrasonography, internal vascularity is generally absent or minimal; however, peripheral vascular signals may occasionally be detected, potentially contributing to misinterpretation as malignant hepatic lesions.[25] The infiltrative growth pattern and absence of a well-defined capsule further limit the diagnostic specificity of ultrasonography. Consequently, hepatic AE is rarely diagnosed with confidence based on ultrasonography alone, and further cross-sectional imaging is usually required for accurate lesion characterization.[1,26]

Computed tomography

CT is one of the main imaging modalities in the evaluation of hepatic AE and plays an important role in assessing lesion morphology and disease extent. On non-contrast CT, the disease typically appears as an infiltrative mass with irregular margins and heterogeneous attenuation. Intralesional calcifications are a distinguishing feature and may present as punctate, linear, or amorphous patterns, which are reliably detected on CT.[8]

On contrast-enhanced CT, lesions typically demonstrate mild, heterogeneous, or peripheral enhancement, reflecting the coexistence of fibrotic tissue, necrosis, and parasitic components. The absence of a true capsule and a propensity for infiltrative spread into the adjacent hepatic parenchyma, bile ducts, as well as the portal and hepatic veins, are frequently observed features. These imaging characteristics may closely mimic hepatic malignancies, particularly cholangiocarcinoma and metastatic disease. Furthermore, vascular invasion may occur and can lead to the development of Budd–Chiari syndrome.[25,27]

In suspected cases, additional non-contrast or multiphasic CT acquisitions may be required to evaluate calcifications and enhancement patterns better, potentially increasing radiation exposure. However, the use of dual-energy CT (DECT) allows the generation of virtual non-enhanced (VNE) images, which can reduce the need for additional non-contrast acquisitions. VNE images have been shown to provide diagnostic confidence comparable with true non-contrast CT, thereby contributing to a reduction in overall radiation dose.[28,29]

CT also enables detailed assessment of lesion number, size, and anatomical location, supporting surgical and interventional treatment planning. Accurate evaluation of vascular, biliary, and extrahepatic involvement facilitates safe planning of interventional procedures.[30-32] Nevertheless, despite its high sensitivity, CT findings alone may not be definitive, as the solid appearance and invasive growth pattern of the lesions can closely mimic malignant hepatic tumors. Therefore, although CT remains superior for the assessment of calcifications, a multimodality imaging approach is often required for definitive diagnosis and accurate lesion characterization.[15]

Magnetic resonance imaging

MRI complements CT in the evaluation of hepatic AE by providing superior soft-tissue contrast and improved lesion characterization. It is particularly useful for assessing internal lesion architecture and biliary and vascular involvement and for differentiating viable parasitic tissue from fibrotic or necrotic components.[1,15]

On T1-weighted images, lesions typically appear hypointense relative to the surrounding liver parenchyma. T2-weighted sequences demonstrate heterogeneous signal intensity with hypointense fibrotic areas and hyperintense microcystic components, producing a multilocular or "honeycomb-like" appearance, which represents a characteristic MRI feature of the disease.[21,33]

Diffusion-weighted imaging (DWI) may show restricted diffusion in solid components containing active parasitic tissue; however, diffusion findings are variable and may overlap with those of malignant hepatic tumors, constituting a potential diagnostic pitfall. Accordingly, apparent diffusion coefficient (ADC) maps often reveal intralesional heterogeneity reflecting mixed tissue composition.[34,35]

Dynamic contrast-enhanced MRI usually demonstrates subtle, heterogeneous, or delayed peripheral enhancement related mainly to fibrotic tissue. The absence of a true capsule and infiltrative extension into adjacent hepatic parenchyma, bile ducts, and vascular

Table 1. Summary of key ultrasound, computed tomography, and magnetic resonance imaging features of hepatic alveolar echinococcosis

Imaging modality	Key features	Diagnostic pitfalls
Ultrasonography	Heterogeneous echotexture, hyperechoic foci (calcifications), absent/minimal vascularity	Pseudo-capsule, infiltrative margins
CT	Infiltrative mass, irregular margins, punctate/linear calcifications, mild peripheral enhancement	May mimic cholangiocarcinoma or metastasis
MRI	Hypointense on T1, heterogeneous T2 signal with honeycomb-like microcysts, peripheral diffusion restriction	Solid appearance, pseudo-enhancement, variable diffusion signal

CT, computed tomography; MRI, magnetic resonance imaging.

structures are common findings and may closely resemble cholangiocarcinoma or infiltrative hepatocellular carcinoma.[36]

MRI also provides important information on biliary involvement, vascular encasement, and extrahepatic extension, contributing to surgical and interventional planning.[15] Nevertheless, despite its high diagnostic value, MRI alone may not always allow confident differentiation from malignant liver tumors. Therefore, accurate diagnosis requires integration of MRI findings with CT, clinical data, and serologic results within a multimodality imaging approach.[15,37]

Diagnostic pitfalls

Solid appearance

Hepatic AE frequently presents as a predominantly solid lesion on radiologic evaluation, representing a major diagnostic pitfall.[19] The absence of a well-defined cystic morphology and the tumor-like appearance of the lesion may prompt a reflex interpretation in favor of malignancy at the initial assessment. This tendency is particularly evident in non-endemic regions, where awareness of parasitic diseases is limited and clinical suspicion for AE is generally low.[3]

When the solid morphology is accompanied by an infiltrative growth pattern, the lesion may convey an impression of aggressive biological behavior, further complicating differentiation from primary or secondary hepatic malignancies.[35]

Accordingly, AE may be mistaken for a malignant process rather than a parasitic infection during the initial diagnostic evaluation. Such misinterpretation can lead to delayed diagnosis, unnecessary invasive interventions, and inappropriate therapeutic management. Importantly, a predominantly solid appearance of the lesion should not be considered sufficient to exclude this entity. Failure to recognize this diagnostic pitfall may further complicate lesion interpretation in subsequent assessments.[29,32,33] These potential challenges are discussed in detail in the following sections.

Capsule pitfall

Assessment of lesion margins represents an important diagnostic pitfall in the radiologic evaluation of hepatic AE. Unlike cystic echinococcosis, AE does not develop a true epithelial capsule. Nevertheless, dense perilesional fibro-inflammatory tissue and compression of the adjacent hepatic parenchyma may create a capsule-like appearance on imaging, which can be misleading.[38]

On ultrasonography, hepatic AE typically appears as a large, irregularly marginated mass with a heterogeneous internal echotexture, composed of intermixed hyperechoic and hypoechoic areas. Intralesional calcifications and hypoechoic regions corresponding to central necrosis may be present, often accompanied by an irregular peripheral hyperechoic zone reflecting fibrotic tissue and creating a pseudo-capsular impression.[8,39]

Hepatic AE lesions exhibit a multivesicular architecture with numerous microcystic components embedded within extensive solid fibro-inflammatory tissue. This configuration may mimic a well-circumscribed lesion and lead to consideration of alternative diagnoses, including benign cystic lesions or encapsulated hepatic tumors.[40]

In some cases, a relatively sharp interface between the lesion and the surrounding liver parenchyma may reinforce the false impression of a well-circumscribed mass, despite the underlying infiltrative growth pattern. Misinterpretation of this capsule-like appearance can obscure the infiltrative nature of hepatic AE and delay recognition of subtle extension into adjacent hepatic parenchyma, biliary, or vascular structures, particularly in early or intermediate stages of disease. Careful assessment of lesion margins across multiple imaging planes is therefore essential to avoid underestimation of disease extent (Figure 1).[11,18]

Contrast enhancement pitfall

Contrast-enhanced imaging can pose a diagnostic challenge in hepatic AE when areas of enhancement are incorrectly attributed to viable neoplastic tissue. Although AE lacks true tumoral enhancement, peripheral or irregular contrast uptake may be observed, typically during early or delayed phases, secondary to fibroinflammatory host reaction rather than active parasitic proliferation. Alteration of normal vascular anatomy and reactive changes in the surrounding liver parenchyma may further contribute to this deceptive appearance.[1,41]

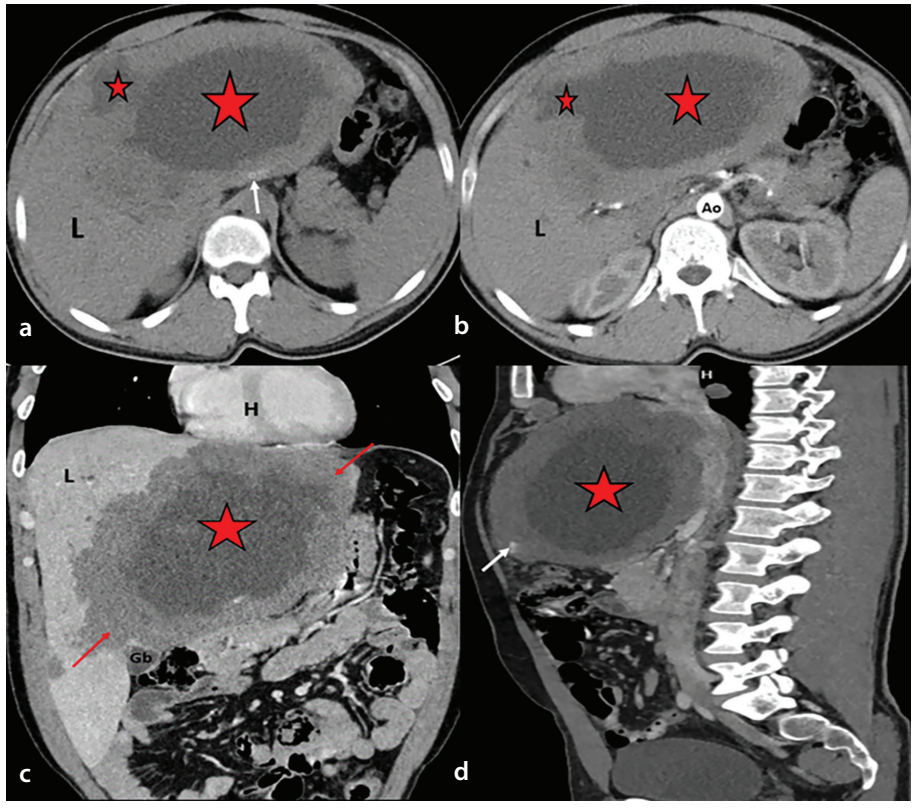

Figure 1. Non-contrast computed tomography (CT) and contrast-enhanced CT images of a 48-year-old man demonstrate a bilobulated hepatic lesion measuring approximately 8 × 6 cm, infiltrating segments II, IV, and VIII, and lacking a well-defined capsule. (**a**) Pre-contrast axial images show a centrally cystic-necrotic component (red stars) and a linear calcification along the inferomedial wall (white arrow). (**b**) The lesion shows no significant enhancement on the arterial phase. (**c**) Coronal delayed venous phase images demonstrate heterogeneous perilesional enhancement (red arrows). (**d**) On sagittal portal venous phase images, the lesion margins are partially more clearly distinguished from the surrounding hepatic parenchyma. These imaging findings are consistent with hepatic alveolar echinococcosis. (Ao, abdominal aorta; H, heart; L, liver).

Such enhancement patterns may falsely suggest hepatic malignancy, including hypervascular primary tumors or metastatic disease. When contrast findings are interpreted in isolation, they may bias lesion characterization toward an oncologic diagnosis and obscure the underlying parasitic nature of the disease.[4,42,43]

For this reason, enhancement characteristics should be assessed in conjunction with morphologic features and evaluated across all contrast phases, rather than being relied upon as a primary indicator of lesion viability.

Overlooked calcifications

Overlooked intralesional calcifications represent a common diagnostic pitfall in hepatic AE. Although calcifications are a characteristic imaging feature of AE, they may be missed when non-contrast CT is not performed or when image interpretation relies predominantly on MRI.[20,28]

In contrast-enhanced CT, enhancement of surrounding fibro-inflammatory tissue may further reduce the conspicuity of small or scattered calcifications. Failure to recognize calcifications may lower suspicion for AE and lead to misinterpretation of the lesion as a malignant hepatic mass. Therefore, careful evaluation of non-contrast CT images or the use of DECT with VNE reconstructions is essential to improve diagnostic confidence.[28]

Neglecting the clinical context

Hepatic AE remains a challenging diagnosis because of its tumor-like appearance on imaging studies.[44] When radiologic findings are interpreted without sufficient clinical and epidemiological context, the likelihood of diagnostic error increases. This resemblance to malignant hepatic disease is especially problematic in patients with an unclear exposure history or in non-endemic regions.[3] Current literature indicates that imaging alone is often insufficient for reliable diagnosis. Accurate identification of AE requires integration of radiologic features with clinical evaluation and serological findings to prevent misclassification as hepatic malignancy.[16]

Diagnostic pearls

Infiltrative growth over mass effect

Hepatic AE primarily demonstrates infiltrative growth rather than a true mass effect.[1,15] Lesions extend along biliary ducts and vascular structures. Adjacent liver parenchyma is often invaded rather than displaced (Figure 2A, B).[39] This growth pattern reflects the biological behavior of the parasite.[16] It also explains the frequent radiologic misinterpretation as hepatic malignancy. Awareness of infiltrative spread in the absence of a well-defined mass should prompt consideration of AE in appropriate clinical or epidemiologic settings.[19]

Calcification as a diagnostic anchor

Intralesional calcifications are a key imaging feature of hepatic AE.[8,45] They are most reliably detected on non-contrast CT. Calcifications may appear punctate, linear, or amorphous (Figure 3). Their presence strongly supports a parasitic etiology in infiltrative hepatic lesions.[46] Failure to recognize calcifications may lead to misclassification as malignant disease. Careful and deliberate assessment for calcification, therefore, provides an important diagnostic anchor.[28,47]

Lack of true arterial hyperenhancement

Hepatic AE does not demonstrate true arterial hyperenhancement on CT (Figure 4A) or MRI (Figure 4B). Any apparent early enhancement usually reflects peripheral fibroinflammatory tissue.[41,45] This finding may mimic hypervascular malignancy on arterial phase imaging. Overinterpretation of arterial phase findings can therefore be misleading. Evaluation of enhancement patterns across all contrast phases is essential for accurate interpretation.[8,45]

Diffusion helps exclude malignancy

In hepatic AE, diffusion restriction is generally not pronounced; however, limited diffusion restriction may be observed in peripheral regions containing active parasitic tissue. This restriction is typically milder and more localized than primary hepatic malignancies, reflecting the low cellularity and heterogeneous tissue composition of the lesion.[35]

DWI has emerged as a valuable functional modality in the assessment of hepatic AE. Importantly, diffusion restriction is predominantly observed at the peripheral infiltrative margins (Figure 5), corresponding to metabolically active and viable parasitic tissue. In contrast, centrally necrotic or inactive components generally exhibit higher ADC values and lack significant diffusion restriction.[35,48]

Therefore, peripheral diffusion restriction represents active parasitic tissue, and the overall ADC profile remains higher than that of primary hepatic malignancies; this re-

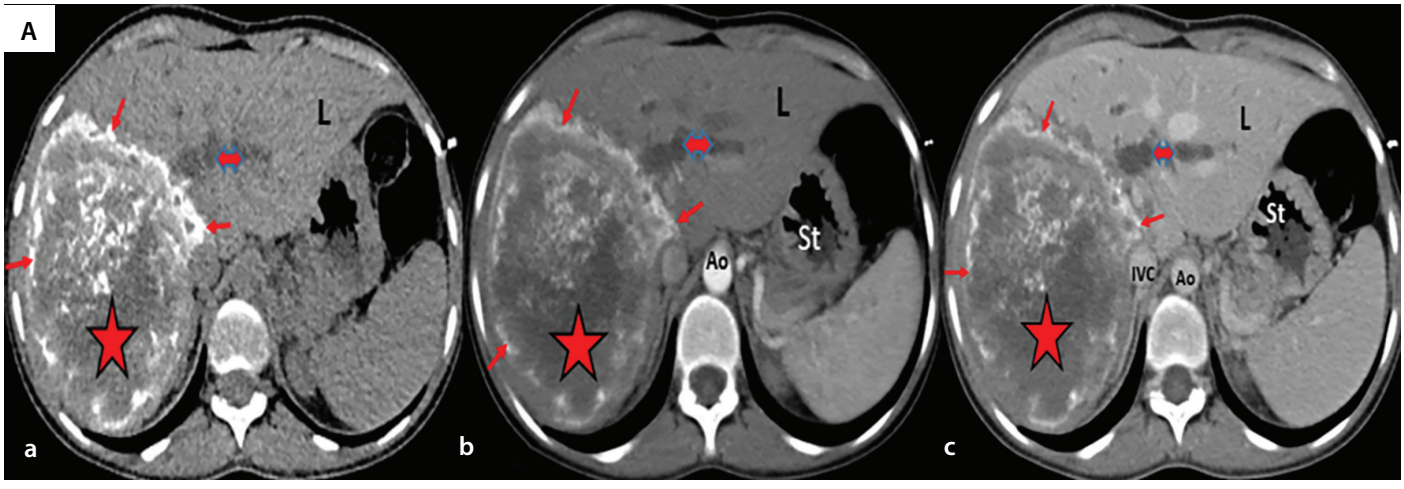

Figure 2A. Non-contrast and contrast-enhanced computed tomography images of a 39-year-old man demonstrate a 10 × 9 cm hepatic lesion involving segments V, VII, and VIII. (**a–c**) On pre-contrast, arterial, and delayed venous phase images, respectively, the lesion exhibits a centrally cystic-necrotic component (red star) with dense amorphous and linear calcifications located both centrally and peripherally (red arrows). In addition, compression–invasion of the adjacent medial intrahepatic bile ducts results in marked biliary dilatation (double-headed red arrow). The imaging findings are consistent with hepatic alveolar echinococcosis. (Ao, abdominal aorta; IVC, inferior vena cava; L, liver; St, stomach).

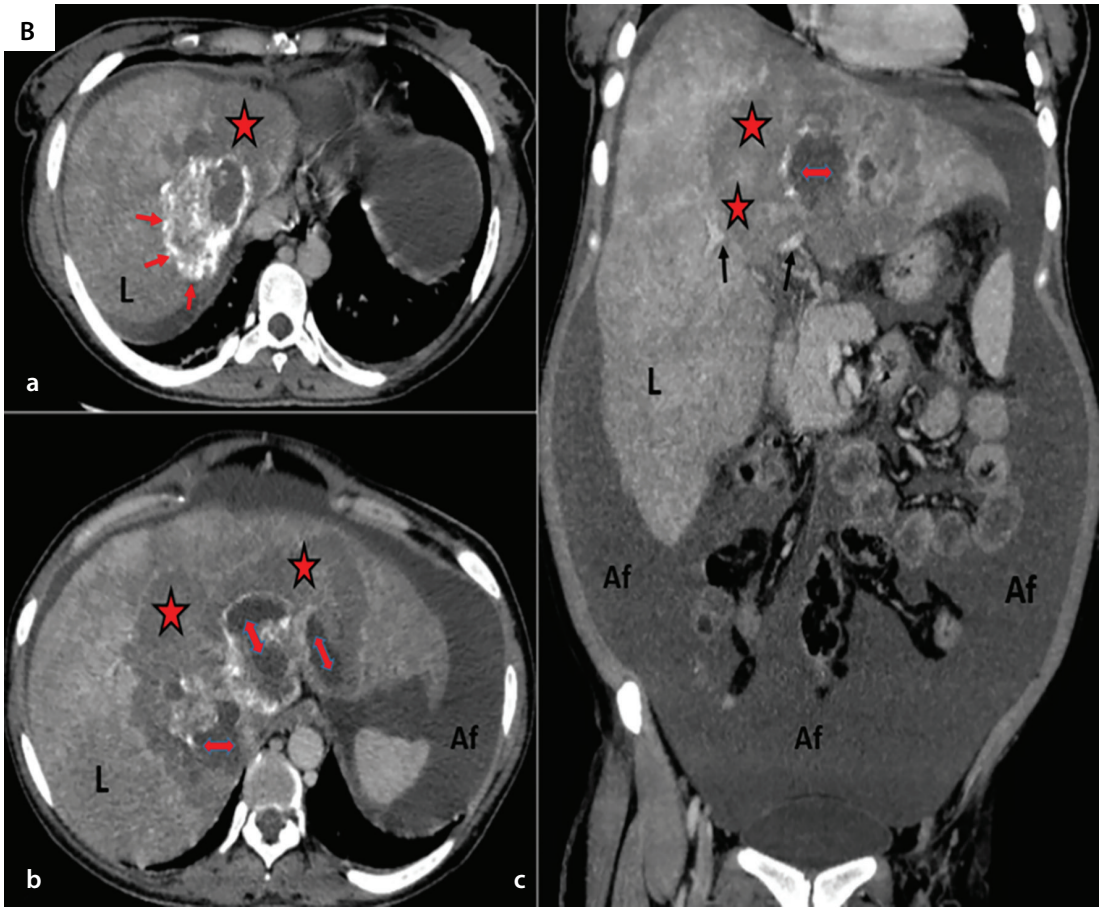

Figure 2B. A 41-year-old female patient presenting with symptoms of liver failure was diagnosed with alveolar echinococcosis. Axial (**a, b**) and coronal (**c**) contrast-enhanced computed tomography images show a 15 cm heterogeneous hepatic mass involving the left, right, and caudate lobes, with cystic areas (red stars) and calcifications (red arrows). The portal vein branches adjacent to the lesion are indicated (black arrows). The hepatic veins are not visualized, likely due to invasion by the mass. Cystic components within the lesion are indicated (double-headed arrow). No biliary dilatation is observed. The patient developed acute liver failure secondary to Budd–Chiari syndrome. Diffuse abdominal ascites is also present (Af, ascitic fluid).

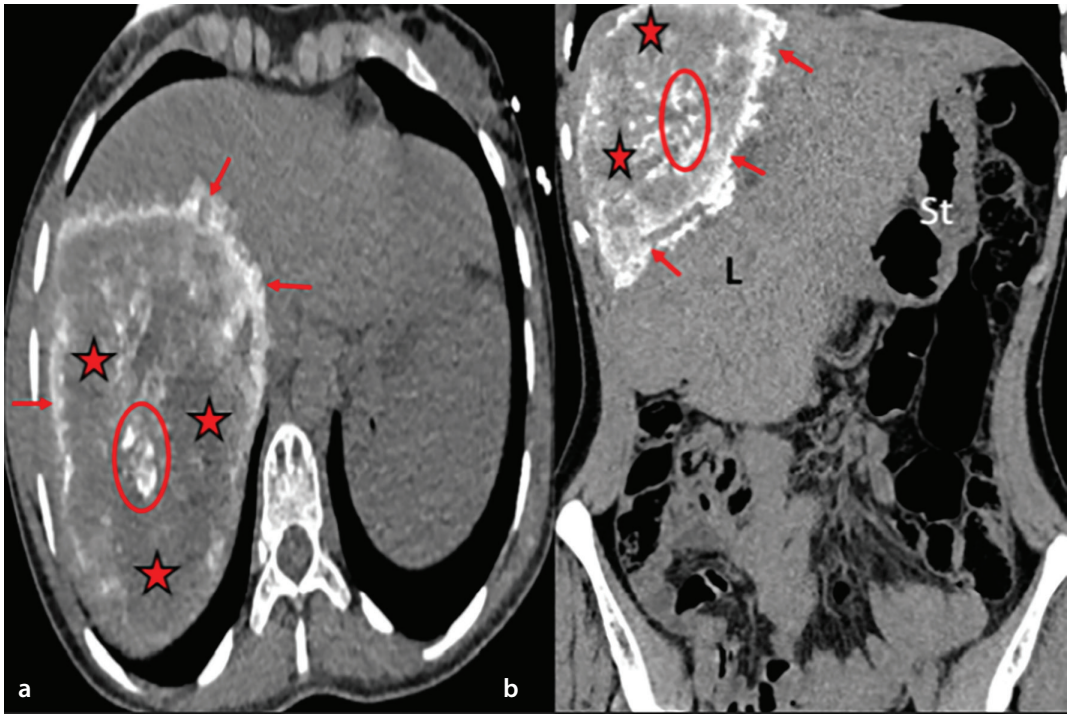

Figure 3. Pre-contrast computed tomography images of a 45-year-old woman demonstrate a large hepatic lesion measuring approximately 13 × 10 cm at the level of segments IV, V, VII, and VIII. (**a, b**) Axial and coronal images reveal dense linear, punctate, and amorphous calcifications (red arrows and ellipse) associated with a centrally cystic-necrotic component (red stars). These findings are consistent with hepatic alveolar echinococcosis. (L, liver; St, stomach).

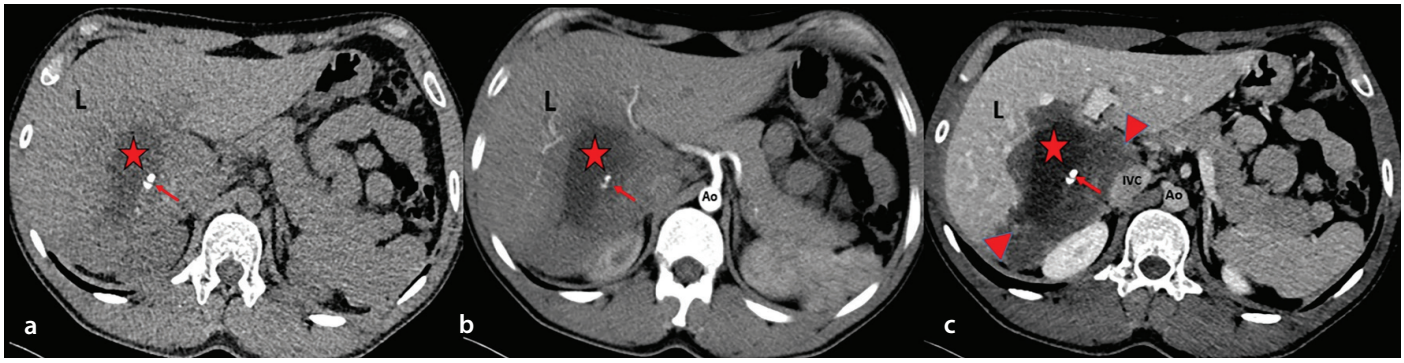

Figure 4A. Non-contrast and contrast-enhanced computed tomography images of a 35-year-old man demonstrate a hepatic lesion involving segments I, V, and VI. (**a**) Pre-contrast axial images show a centrally cystic-necrotic component (red star) with millimetric amorphous calcifications along the medial wall (red arrow). (**b**) The lesion shows no significant enhancement on the arterial phase. (**c**) On delayed venous phase images, heterogeneous mild peripheral enhancement is observed (red arrowheads). These findings are consistent with hepatic alveolar echinococcosis. (Ao, abdominal aorta; IVC, inferior vena cava; L, liver).

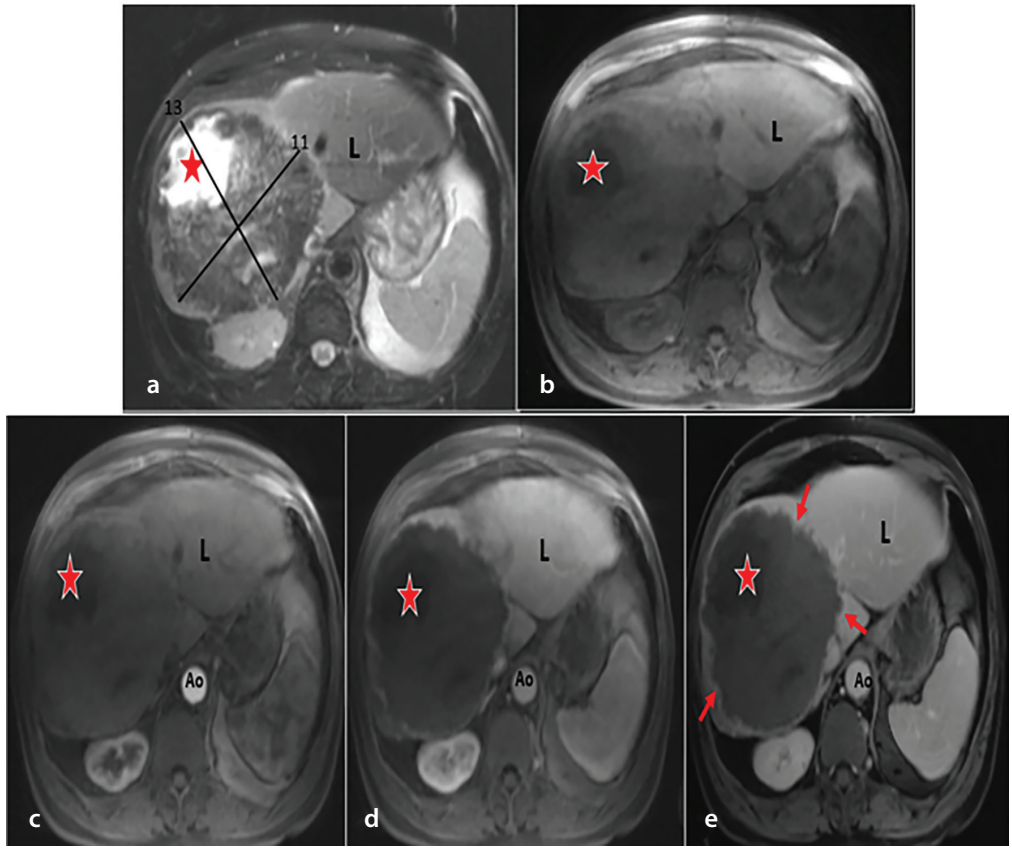

Figure 4B. Alveolar echinococcosis in a 68-year-old male patient presenting with right upper quadrant pain and fever. Axia T2-weighted image (**a**) and axial pre-contrast T1-weighted image (**b**) show a 13 × 11 cm heterogeneous mass located in the right lobe, extending into segment IV and segment I. The lesion contains large cystic areas (red star). Axial arterial phase (**c**), portal venous phase (**d**), and delayed phase (**e**) contrast-enhanced images demonstrate peripheral fibroinflammatory enhancement (red arrows) on the delayed phase. (Ao, abdominal aorta; L, liver).

solves the apparent contradiction between peripheral diffusion restriction and the exclusion of malignancy.

Accordingly, DWI may provide indirect information regarding lesion viability and biological activity beyond conventional morphologic imaging. Furthermore, reduction or disappearance of peripheral diffusion restriction on follow-up imaging may serve as a potential indicator of therapeutic response.[48]

Proposed diagnostic approach algorithm for hepatic alveolar echinococcosis

In clinical practice, a structured and stepwise diagnostic approach may improve recognition of hepatic AE and reduce misinterpretation as hepatic malignancy. Based on the characteristic imaging features and common diagnostic pitfalls discussed in this review, the following simplified algorithm is proposed:

Step 1: Identify lesion morphology

In the presence of an infiltrative hepatic lesion with ill-defined margins and absence of a true capsule, AE should be considered in the differential diagnosis.

Step 2: Assess for intralesional calcifications

Evaluate carefully, preferably on non-contrast CT or VNE images. Presence of punctate, linear, or amorphous calcifications strongly favors AE.

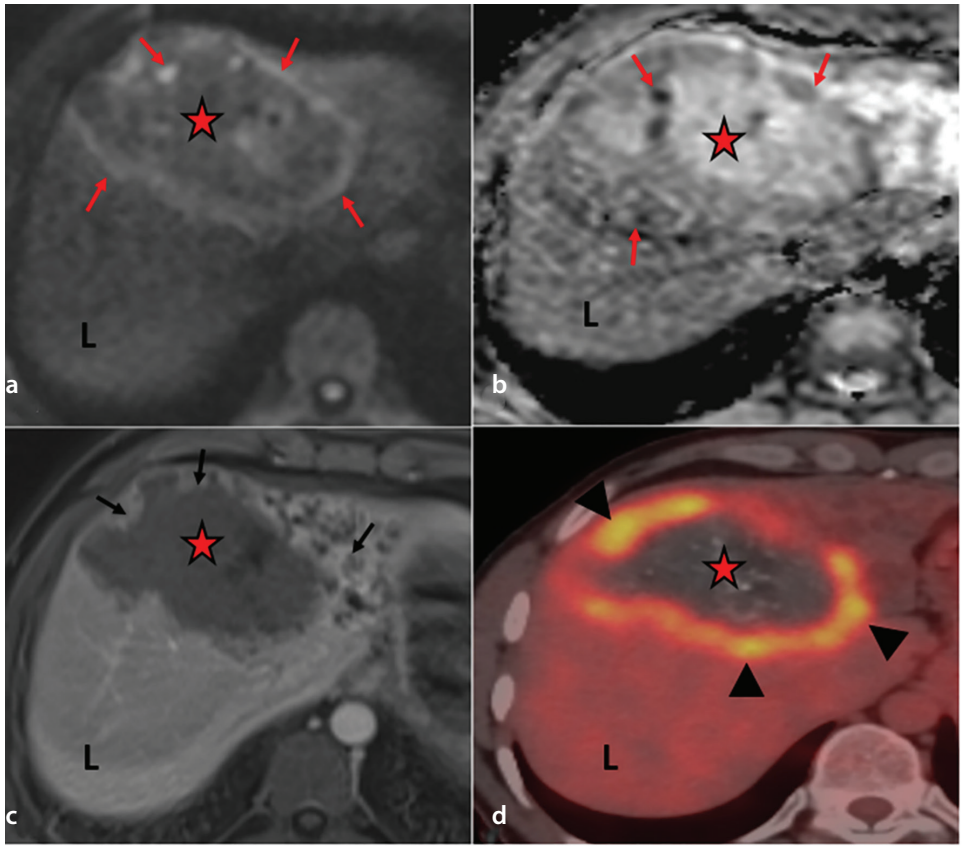

Figure 5. Alveolar echinococcosis (AE) in a 37-year-old male patient who presented with abdominal pain. Axial diffusion-weighted imaging (b: 800 s/mm^2) (**a**) and the corresponding apparent diffusion coefficient map (**b**) demonstrate peripheral diffusion restriction (red arrows) in an approximately 12 × 8 cm lesion involving liver segments 2, 4, and 8. In the venous phase, (**c**) the lesion shows heterogeneous peripheral fibroinflammatory mild enhancement (black arrows), with a centrally cystic component (red asterisk). Additionally, fluorodeoxyglucose (FDG) positron emission tomography/computed tomography imaging (**d**) reveals increased FDG uptake at the periphery of the mass (black arrowheads). These findings are consistent with AE. (L, liver).

Step 3: Evaluate contrast enhancement pattern

Determine whether there is true arterial hyperenhancement. A lack of true arterial hyperenhancement and the presence of mild, irregular, or delayed peripheral enhancement support AE rather than hypervascular malignancy.

Step 4: Analyze diffusion-weighted imaging

Look for peripheral, limited diffusion restriction corresponding to active parasitic tissue.

Higher overall ADC values than those of malignant lesions favor AE.

Step 5: Evaluate growth pattern and associated findings

Assess for infiltrative extension along biliary and vascular structures rather than mass effect. Identify multivesicular/microcystic components or "honeycomb" appearance on MRI.

Step 6: Integrate clinical and serologic data

Consider epidemiologic background (endemic regions, exposure history). Confirm with serologic tests (e.g., ELISA, *Echinococcus multilocularis* immunoblot) when imaging findings are suggestive.

Step 7: Exclude malignancy and guide management

If imaging and clinical findings are concordant, establish a diagnosis and proceed with staging (e.g., PNM classification). In indeterminate cases, multidisciplinary evaluation is recommended to avoid unnecessary invasive procedures.

Hepatic AE remains a challenging entity in abdominal imaging because of its infiltrative growth pattern and close radiologic resemblance to hepatic malignancies. A predominantly solid appearance, pseudo-capsular margins, and misleading enhancement patterns frequently lead to misinterpretation, especially in non-endemic regions. Careful evaluation for infiltrative extension along biliary and vascular structures is essential. Deliberate assessment for intralesional calcifications provides an important diagnostic clue. Staging systems and morphologic classifications are useful but should be considered supportive tools rather than definitive criteria. Accurate diagnosis relies on a multimodality imaging approach combined with clinical, epidemiological, and serologic information. Increased awareness of characteristic imaging features and common pitfalls may reduce diagnostic delay. This approach also helps prevent inappropriate oncologic management and supports improved clinical outcomes in this potentially life-threatening disease.

Footnotes

Conflict of interest disclosure

The authors declared no conflicts of interest.

Consent for publication

Not required, as all radiological images included in this review are fully anonymized and contain no identifiable patient information.

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