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Original paper

Factors associated with diaphragmatic invasion in hepatic alveolar echinococcosis: a retrospective CT-based analysis

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Abstract

Purpose: The purpose of this study was to determine the frequency of diaphragmatic invasion and to identify topographic, clinical, and morphological factors associated with it using contrast-enhanced computed tomography (CT).

Material and methods: In this retrospective study, 201 patients with confirmed hepatic alveolar echinococcosis (AE) examined at national referral centres between 2018 and 2025 were included. All patients underwent multiphase contrast-enhanced multidetector CT of the liver. Demographic data, lesion number and localisation, Alveolar Echinococcosis Ulm Classification type, maximum lesion diameter, vascular invasion, and extrahepatic extension were recorded. Diaphragmatic invasion was defined by the presence of at least two predefined CT criteria. Associations were assessed using Cramer's V, and predictors of diaphragmatic invasion were analysed using multivariable logistic regression.

Results: Extrahepatic spread was identified in 108 patients (53.7%). The association between diaphragmatic invasion and involved liver segments did not reach statistical significance (Cramer's $V = 0.619$; $p = 0.090$). This result should be interpreted cautiously. Diaphragmatic involvement was more frequently observed in patients with lesions affecting the superior liver segments S2, S7, and S8. No significant positive associations were found between diaphragmatic invasion and vascular involvement (Cramer's $V \leq 0.484$; all $p > 0.65$). In multivariable analysis, portal vein trunk invasion was inversely associated with diaphragmatic involvement (OR = 0.234; 95% CI: 0.062-0.883; $p = 0.032$).

Conclusions: Diaphragmatic invasion is a relatively frequent feature of hepatic AE. Associations with liver segment involvement and vascular invasion require cautious interpretation. Recognition of characteristic CT findings remains important for pre-operative assessment and surgical planning.

Key words: risk factors, diaphragm, multidetector computed tomography, hepatic alveolar echinococcosis, surgical planning, organ invasion.

Introduction

Alveolar echinococcosis (AE) of the liver is a chronic parasitic disease caused by the larval stage of *Echinococcus multilocularis* [1]. Hepatic lesions are characterised by infiltrative growth and multiple small cystic structures, and they can closely mimic malignant tumours by invading vascular and biliary structures and adjacent organs [2]. Because of this behaviour, AE is often described as "parasitic liver cancer".

For many years AE was considered endemic to selected regions of the Northern Hemisphere. However, over the past two decades its geographical distribution has expanded substantially [3]. The disease holds particular epidemiological importance in Central Asian countries, where a marked increase in incidence has been documented [4]. In the Kyrgyz Republic, for example, the number of reported AE cases rose from 2 in 2002 to 92 in 2024, with the incidence increasing from 0.05 to 1.7 per100,000 population [5]. A wave-like temporal pattern has been observed,

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Authors' contribution:
A Study design · **B** Data collection · **C** Statistical analysis · **D** Data interpretation · **E** Manuscript preparation · **F** Literature search · **G** Funds collection

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with a peak incidence of 3.9 per 100,000 in 2015, emphasising the need for continued epidemiological surveillance and improved diagnostic strategies [6].

The clinical course of AE is characterised by a long asymptomatic period, and by the time of diagnosis the disease frequently extends beyond the liver [7]. To improve the assessment of vascular invasion in hepatic AE, imaging criteria based on multislice computed tomography (CT) with intravenous bolus contrast enhancement have been proposed [8]. Invasion of the diaphragm, stomach, adrenal glands, kidneys, and other adjacent structures significantly complicates treatment, contributes to more advanced disease staging, and determines the extent and complexity of surgical intervention [9].

In this context, early and accurate evaluation of the local spread of the parasitic process is critical [10]. Multiphase contrast-enhanced multidetector CT is widely available and allows not only detection of primary hepatic lesions but also assessment of their relationship with vascular structures and adjacent organs, providing key information for staging and surgical decision-making.

Despite the recognised importance of extrahepatic extension, data specifically addressing the patterns and predictors of diaphragmatic invasion in hepatic AE remain limited. Given the close anatomical relationship between the superior hepatic segments and the diaphragmatic dome, diaphragmatic involvement may be expected, but its frequency, risk factors, and relationship with vascular invasion are not well characterised.

The aim of this study was therefore to evaluate the frequency of diaphragmatic invasion in hepatic AE and to identify topographic, clinical, and morphological factors associated with diaphragmatic involvement based on multislice CT findings.

Material and methods

Study design and population

We conducted a retrospective observational study based on the analysis of 201 CT examinations of patients with hepatic AE. All patients were managed and examined at national referral centres in the Kyrgyz Republic between 2018 and 2025. For each patient, a standardised electronic record was generated, including demographic data, clinical history, imaging protocols, and, where available, operative reports and histological findings.

Inclusion criteria

Patients were eligible for inclusion if they fulfilled all the following criteria:

1. Confirmed diagnosis of hepatic AE, either histologically or based on typical clinical and radiological features in accordance with international recommendations World Health Organization Informal Working Group on Echinococcosis [11]. These features included infiltrative hepatic lesions with irregular margins, a heterogeneous structure with multiple small cystic components ("alveolar" pattern), presence of calcifications, absence of a well-defined capsule, and progressive contrast enhancement patterns on delayed CT phases, in combination with compatible clinical presentation and epidemiological history [1].
2. Availability of a complete CT examination of the liver with intravenous bolus contrast enhancement.
3. Availability of basic clinical and anamnestic data, and, if surgical treatment was performed, corresponding operative information.

Exclusion criteria

Exclusion criteria were as follows:

1. Incomplete, technically inadequate, or low-quality CT datasets precluding reliable assessment of hepatic and extrahepatic disease.
2. Incomplete medical documentation that did not allow full evaluation (e.g. missing imaging archives or reports).
3. Follow-up CT examinations in patients already included in the study, when only the most recent examination was available and serial changes could not be assessed consistently.

CT technique and image analysis

All patients underwent multiphase contrast-enhanced multidetector CT of the liver according to institutional protocols. Examinations included at least a contrast-enhanced phase suitable for the evaluation of parenchymal lesions and vascular structures. Delayed phase imaging was available and used to help confirm organ invasion where required.

Images were analysed on dedicated workstations using axial and multiplanar reconstructions. Liver segments were defined according to the Couinaud classification (S1-S8). For each patient, the number, size, and segmental localisation of hepatic lesions were recorded. Maximum lesion diameter was measured in the axial plane.

The morphological type of the largest lesion was classified according to Alveolar Echinococcosis Ulm Classification (AEUC), a stage-oriented CT-based system derived from the *Echinococcus multilocularis* Ulm classification for CT [12]. The AEUC system defines five primary morphological types (AEUC I-V), reflecting progressive structural changes of hepatic AE.

AEUC I corresponds to small cystoid or metastasis-like lesions with a microcystic structure. AEUC II represents diffuse infiltrative lesions without a clearly defined growth centre, often with irregular margins and longitudinal spread. AEUC III includes circumscribed tumour-like lesions with a defined central component and convex contours. AEUC IV is characterised by predominantly cystoid lesions with a large component of liquid necrosis. AEUC V

corresponds to predominantly calcified lesions, usually representing more advanced or regressive stages. In addition, the classification incorporates patterns of calcification (e.g. feathery, focal, or diffuse), which further characterise lesion morphology and may reflect different stages of disease evolution. This classification allows structured assessment of lesion morphology and provides insight into the progression of hepatic AE from early infiltrative to advanced and partially regressive stages.

Definition and assessment of invasion into adjacent structures

Invasion into adjacent anatomical structures was assessed for the diaphragm, gallbladder, right adrenal gland, kidneys, stomach, pancreas, and intestinal loops. Invasion was defined by a combination of the following CT signs:

1. Loss of the fat plane and absence of a clear interface between the lesion and the adjacent organ.
2. Deformation and/or displacement of the anatomical contours of the adjacent organ at the contact site.
3. Altered tissue density in the contact zone (e.g. stranding, soft-tissue thickening).
4. Presence of tissue with imaging characteristics consistent with AE within the adjacent organ, confirmed in delayed enhancement phases.
5. Morphological changes of the affected organ (e.g. wall thickening, focal defects, necrosis, or calcifications).
6. Diaphragmatic invasion was considered present when at least two of these criteria were fulfilled. The approximate area of invasion was measured in cm^2 using axial and multiplanar reconstructions.

Evaluated variables

To identify factors associated with extrahepatic spread and specifically with diaphragmatic invasion, the following parameters were collected:

- sex and age;
- number of hepatic lesions;
- AEUC type of the largest lesion;
- maximum diameter of the largest lesion;
- segmental localisation (S1-S8);
- number of involved liver segments and sectors;
- presence of biliary hypertension;
- vascular invasion (portal vein trunk, right and left portal branches, right, middle, and left hepatic veins, inferior vena cava, and hepatic arterial branches);
- involvement of adjacent organs (diaphragm, gallbladder, right adrenal gland, kidneys, stomach, pancreas, small and large intestine, duodenum).

Statistical analysis

Categorical variables were summarised using absolute and relative frequencies. Comparisons between groups were performed using contingency tables (CROSSTABS) with calculation of the φ coefficient and Cramer's V as measures of association. The χ^2 test was applied to assess statistical significance at a two-sided significance level of $p < 0.05$.

To further investigate predictors of diaphragmatic invasion, a binary logistic regression model was constructed with diaphragmatic invasion (yes/no) as the dependent variable and vascular invasion variables as predictors. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated.

All statistical analyses were carried out using IBM SPSS Statistics, version 23.0. Data visualisation (histograms, distribution charts, and heat maps) was performed with SPSS and Microsoft Excel.

Ethical considerations

All patient data were anonymised before analysis. Linkage keys were stored separately and securely by the project supervisor. The study was conducted in accordance with local ethical standards and the principles of the Declaration of Helsinki. Formal approval was obtained from the Bioethics Committee of the National Surgical Center named after Academician M.M. Mamakeev, Ministry of Health of the Kyrgyz Republic (Protocol No. 15, dated 16 May 2022).

Results

General characteristics of the study population

A total of 201 patients with hepatic AE were included: 113 women (56.2%) and 88 men (43.8%). The mean age was 37.5 ± 14 years (median 35 years; range 10-77 years). More than half of the cohort (over 55%) were between 20 and 40 years of age, consistent with previous reports describing a peak incidence in the working-age population.

With regard to lesion number, a single hepatic lesion was present in 129 patients (64.2%), two lesions in 37 patients (18.4%), three lesions in 9 patients (4.5%), four lesions in 6 patients (3.0%), and five or more lesions in 15 patients (7.5%). Extrahepatic recurrence after previous surgical intervention was observed in 5 patients (2.5%), predominantly in the region of the hepatoduodenal ligament. Five patients had no detectable intrahepatic lesions at the time of CT examination because they had previously undergone surgical treatment for hepatic alveolar echinococcosis and presented with extrahepatic disease.

Segmental involvement

Segmental distribution of lesions is illustrated in Figure 1. Segments S1, S4, S7, and S8 were affected in more than 50% of cases, indicating a typical localisation pattern in the hilar region and right hepatic lobe. Segment S3 was affected less frequently (approximately 30%).

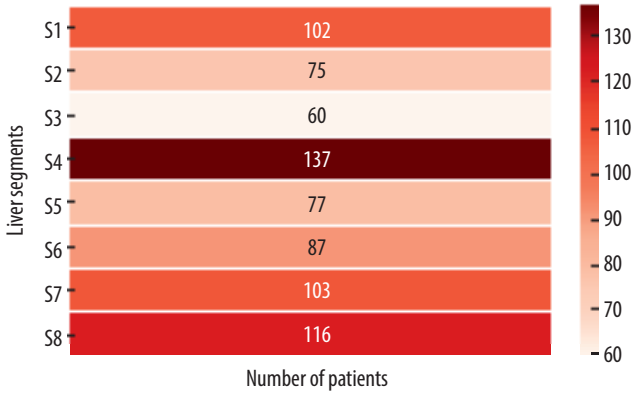


Figure 1. Heat map of liver segment involvement in hepatic alveolar echinococcosis

Extrahepatic spread and organ invasion

Analysis of CT data from 201 patients demonstrated that the disease frequently extended beyond the hepatic parenchyma, involving adjacent anatomical structures (Table 1). Invasion of surrounding organs was documented in 108 cases (53.7%), with substantial variability in the degree and spatial extent of involvement depending on lesion localisation and morphological type.

The diaphragm, gallbladder, and right adrenal gland were the most commonly affected extrahepatic structures. These organs lie in direct anatomical proximity to the liver and are separated only by thin fascial planes or loose connective tissue, which favours infiltrative spread of parasitic tissue. In the following, we focus on factors associated with diaphragmatic invasion. In addition to the range of invasion area, mean values demonstrated substantial variability in the extent of organ involvement. The largest mean invasion area was observed in diaphragmatic involvement, reflecting the broad contact surface between the hepatic dome and the diaphragm. In contrast, invasion into smaller structures such as the adrenal gland, duodenum, or gallbladder was characterised by more limited areas of involvement. These differences likely reflect both the anatomical proximity and structural resistance of adjacent tissues to parasitic infiltration.

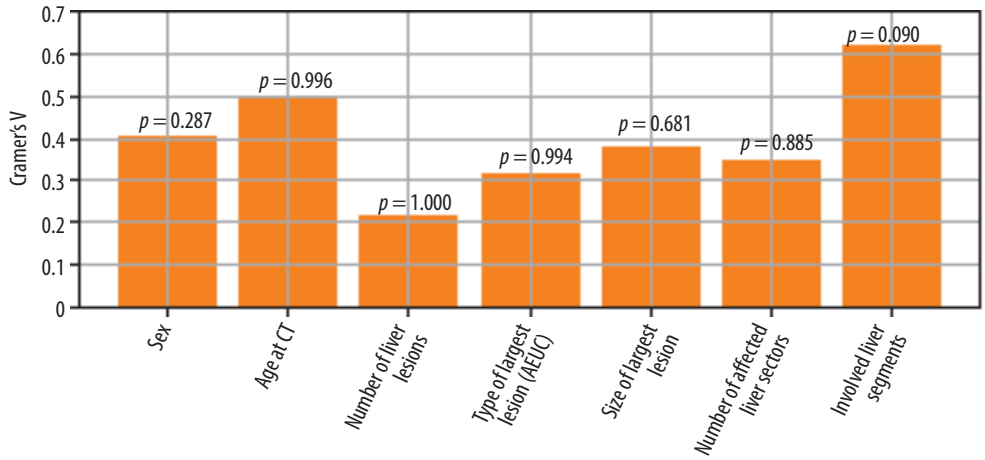
Diaphragmatic invasion

Diaphragmatic invasion was identified in 38 cases (18.9%), while 163 patients (81.1%) showed no CT evidence of diaphragmatic involvement.

Table 1. Invasion into adjacent organs in hepatic alveolar echinococcosis

Organ	Number of patients (*n*)	Frequency (%)	Range of invaded area (cm^2)	Mean ± SD (cm^2)
Diaphragm	38	18.9	11-400	85.0 ±90.0
Gallbladder	20	10.0	5-33	14.5 ±8.0
Right adrenal gland	28	13.9	4-30	11.0 ±7.5
Right kidney	5	2.5	6-35	18.0 ±11.0
Pancreas	6	3.0	11-20	15.5 ±4.0
Stomach	9	4.5	15-100	42.0 ±28.0
Duodenum	3	1.5	6-24	13.0 ±9.0
Large intestine	1	0.5	6	single case
Small intestine	1	0.5	45	single case

SD – standard deviation



AEUC – Alveolar Echinococcosis Ulm Classification

Figure 2. Strength of association (Cramer's V) between selected factors and diaphragmatic invasion

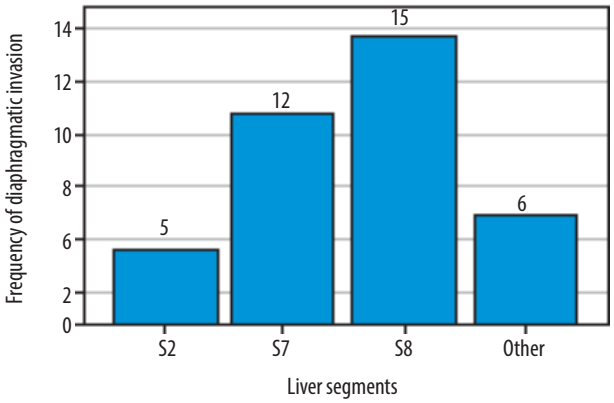


Figure 3. Frequency of diaphragmatic invasion according to affected liver segments (absolute values)

Association analysis (Figure 2) yielded Cramer's V = 0.619 for involved liver segments, but the association did not reach statistical significance (p = 0.090). The result should be interpreted cautiously because of sparse data in the contingency table.

When lesion topography was analysed in more detail, a clear trend towards a higher frequency of diaphragmatic invasion was found in cases involving upper liver segments S2, S7, and S8 (Figure 3). In contrast, isolated involvement of lower segments was rarely associated with diaphragmatic invasion. These findings support the key role of localisation in segments S7-S8 and, to a lesser extent, S2 as a topographic risk factor for diaphragmatic involvement.

No clinically meaningful associations with diaphragmatic invasion were observed for sex, age, number of lesions, AEUC type, size of the largest lesion, or number of involved hepatic sectors (all $p > 0.28$), highlighting the predominant importance of anatomical localisation rather than overall lesion burden or morphology.

Relationship between diaphragmatic invasion and vascular involvement

To explore potential links between diaphragmatic invasion and vascular involvement, an analysis was performed using hepatic vascular territories (Table 2).

None of the evaluated vascular predictors showed a statistically significant positive association with diaphragmatic invasion.

In the binary logistic regression analysis (Table 3), which included CT indicators of invasion of the main hepatic vas-

Table 2. Correlation between diaphragmatic invasion and vascular involvement

No.	Predictor	Cramer's V	*p*-value	Interpretation
1	Portal vein trunk invasion	0.299	1.000	No significant correlation
2	Right portal vein branch invasion	0.236	1.000	No significant correlation
3	Left portal vein branch invasion	0.389	0.661	No significant correlation
4	Right hepatic vein invasion	0.269	1.000	No significant correlation
5	Middle hepatic vein invasion	0.253	1.000	No significant correlation
6	Left hepatic vein invasion	0.377	0.830	No significant correlation
7	Inferior vena cava invasion	0.484	1.000	No significant correlation

Table 3. Multivariable logistic regression analysis of vascular invasion as a predictor of diaphragmatic invasion

Predictor	B	S.E.	Wald	*p*-value	OR (Exp(B))	95% CI (lower)	95% CI (upper)
Common hepatic artery	1.386	1.165	1.416	0.234	4.000	0.408	39.216
Proper hepatic artery	–1.693	1.456	1.352	0.245	0.184	0.011	3.191
Right hepatic artery	–0.596	0.737	0.653	0.419	0.551	0.130	2.338
Left hepatic artery	0.107	0.675	0.025	0.874	1.113	0.297	4.175
Portal vein trunk	–1.454	0.679	4.591	0.032	0.234	0.062	0.883
Right portal vein branch	–0.184	0.546	0.113	0.737	0.832	0.286	2.426
Left portal vein branch	0.226	0.578	0.152	0.696	1.253	0.403	3.895
Right hepatic vein	0.204	0.469	0.189	0.664	1.226	0.489	3.071
Middle hepatic vein	–0.658	0.497	1.749	0.186	0.518	0.195	1.373
Left hepatic vein	0.846	0.501	2.853	0.091	2.331	0.873	6.224
Inferior vena cava	0.420	0.434	0.934	0.334	1.521	0.650	3.563
Intercept	–1.564	0.310	25.476	<0.001	0.209	–	–

B – regression coefficient, CI – confidence interval, OR – odds ratio, S.E. – standard error

cular structures as predictors and diaphragmatic invasion as the dependent variable, only portal vein trunk invasion was identified as a statistically significant factor ($p = 0.032$). Interestingly, the OR was < 1, indicating an inverse relationship between portal trunk invasion and diaphragmatic involvement.

The OR for portal vein trunk invasion was 0.234 (95% CI: 0.062-0.883), indicating that the presence of portal trunk invasion was associated with a roughly 77% lower odds of diaphragmatic invasion. For the remaining vascular structures, no statistically significant associations with diaphragmatic invasion were found (all $p > 0.05$). There was a non-significant trend towards an increased risk associated with left hepatic vein invasion ($p = 0.091$; OR = 2.331; 95% CI: 0.873-6.224), which may reflect the anatomical proximity of segment S2 lesions to the left diaphragmatic dome.

Taken together, the logistic regression results are consistent with the correlation analysis and support the notion that the key factor in the development of diaphragmatic invasion is not vascular involvement, but rather the topographic localisation of lesions in the upper liver segments, corresponding to a predominantly peripheral growth pattern.

Discussion

This study demonstrates that invasion of hepatic AE into surrounding organs is a common finding, occurring in more than half of the patients (53.7%) in our cohort. The most frequent sites of extrahepatic spread were the diaphragm, right adrenal gland, and gallbladder. This pattern reflects the anatomical arrangement of these structures, which are closely apposed to the liver and separated only by thin fascial layers or loose connective tissue.

Topographic risk factors for diaphragmatic invasion

Diaphragmatic invasion was observed in 18.9% of patients and showed a clear association with lesion localisation in the superior hepatic segments S7-S8 and S2. Although the association between the number of involved segments and diaphragmatic invasion did not meet the conventional threshold for statistical significance ($p = 0.090$), it had the largest effect size (Cramer's $V = 0.619$) among the evaluated factors and showed a consistent pattern in the segment-based analysis. These findings highlight the key role of topography, with lesions located beneath the diaphragmatic dome showing the highest propensity to invade the diaphragm.

Previous reports have also suggested that lesions in the upper hepatic segments are more likely to invade the diaphragm in both malignant tumours and parasitic disease [10-12]. Our results are in line with these observations and extend them by quantifying the frequency of diaphragmatic involvement and systematically analysing its associations with vascular invasion and other clinical variables.

Relationship between vascular and diaphragmatic invasion

In contrast to the clear influence of lesion localisation, we did not observe a positive association between diaphragmatic invasion and vascular involvement. None of the evaluated vascular territories showed a statistically significant positive correlation with diaphragmatic invasion in the bivariate analysis. Surprisingly, portal vein trunk invasion emerged as a negative predictor of diaphragmatic involvement in the multivariable model, with an OR of 0.234. One possible explanation is that portal trunk invasion reflects a predominantly central growth pattern of AE within the hepatic hilum, whereas diaphragmatic invasion is more characteristic of lesions with peripheral growth towards the hepatic dome.

The observed non-significant trend towards increased risk associated with left hepatic vein invasion may be related to the anatomical proximity of segment S2 lesions to the left diaphragmatic dome. However, given the limited number of cases and the lack of statistical significance, this finding should be interpreted cautiously and confirmed in larger series.

Clinical implications for surgical planning

The presence of diaphragmatic invasion has direct implications for surgical planning. Involvement of the diaphragm increases the likelihood of combined hepatic and diaphragmatic resections and adds complexity to liver mobilisation, especially in extensive right lobe disease. Early and accurate recognition of diaphragmatic invasion on CT enables surgeons to anticipate the need for *en bloc* resection of the involved diaphragm, to plan the extent of resection and potential reconstruction, and to assess operative risks.

Our findings underscore the importance of systematic evaluation of the hepatic dome and adjacent diaphragmatic region when interpreting CT examinations in patients with hepatic AE. Radiologists should pay particular attention to the presence or absence of the fat interface, deformation of the diaphragmatic contour, and alterations in tissue density at the liver–diaphragm contact zone. Multiphase contrast-enhanced imaging and delayed phases can improve the differentiation between simple compression and true invasion.

Epidemiological context and radiological practice

The increasing incidence of AE in the Kyrgyz Republic and other endemic regions [5,6] places additional demands on radiology services. Radiologists in these areas regularly encounter advanced cases with complex patterns of intrahepatic and extrahepatic spread. Standardisation of CT protocols and reporting templates for hepatic AE, including explicit assessment of organ invasion and segmental disease distribution, may help improve diagnostic consistency and interdisciplinary communication.

Our data highlight the diaphragm as a common and clinically relevant site of invasion, together with the right adrenal gland and gallbladder. Awareness of these patterns can prompt targeted evaluation and facilitate early detection of invasion, potentially improving resectability assessment and long-term outcomes.

Limitations

Several limitations of this study should be acknowledged. First, the retrospective design is susceptible to selection bias; patients referred to national centres are likely to have more advanced disease than the general AE population. Secondly, CT protocols were not fully standardised across all examinations, which may have influenced the sensitivity for detecting organ invasion, particularly in the absence of uniform delayed-phase imaging. Thirdly, histopathological confirmation of diaphragmatic invasion was not available in all cases; in some patients, assessment was based solely on imaging criteria, albeit stringent ones (≥ 2 CT signs). Fourthly, the number of cases with diaphragmatic invasion (n = 38) is relatively modest compared with the number of predictors included in the logistic regression model, raising the possibility of overfitting. The identified inverse association between portal vein trunk invasion and diaphragmatic involvement should therefore be interpreted as hypothesis-generating and requires validation in independent cohorts.

Finally, our study was conducted in a single country with a high endemic burden of AE; extrapolation of these results to other geographic regions and healthcare settings should be performed with caution.

Conclusions

1. Extrahepatic spread of hepatic AE was identified in more than half of the patients in this cohort, most frequently involving the diaphragm, gallbladder, and right adrenal gland.
2. Diaphragmatic invasion occurred in 18.9% of cases. Although superior hepatic segments (S7-S8 and S2) were frequently involved, the segment-based association did not reach statistical significance (p = 0.090).
3. Vascular invasion did not show a positive association with diaphragmatic involvement; portal vein trunk invasion was inversely related to diaphragmatic invasion, suggesting different patterns of central versus peripheral spread.
4. CT markers such as loss of the fat interface, deformation of the diaphragmatic dome, and changes in tissue density at the liver–diaphragm interface are important indicators of diaphragmatic invasion and should be actively sought in reporting.
5. Accurate assessment of diaphragmatic involvement is crucial for pre-operative planning, influencing the extent of resection, the need for combined hepato-diaphragmatic procedures, and the anticipated complexity of liver mobilisation.
6. Routine use of multiphase contrast-enhanced multidetector CT and structured assessment of extrahepatic spread can improve staging accuracy and contribute to better clinical outcomes in patients with hepatic AE.
7. Standardisation of radiological criteria for invasion and targeted training of radiologists in endemic regions represent important steps towards optimising the diagnostic and surgical management of AE.

Disclosures

1. Institutional review board statement: The study was approved by the Bioethics Committee of the National Surgical Center named after Academician M. M. Mamakeev, Ministry of Health of the Kyrgyz Republic (Protocol No. 15, dated 16 May 2022).
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

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