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Echinococcus multilocularis infection acquired in central Europe in a migrant from an *E. granulosus*-endemic country — A case report

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ABSTRACT

Background: Echinococcosis is a zoonotic infection caused by tapeworms of the genus *Echinococcus*. Geographic provenance is often used to guide species attribution, but may be misleading in patients who have migrated into endemic areas.
Case presentation: A cystic hepatic lesion was discovered in a migrant from Eritrea, residing in Germany for 8 years. Imaging studies (Ultrasound, CT, MRI, PET-CT) demonstrated a polycystic structure in the right lobe. Serology was positive for *Echinococcus* (without species differentiation). Based on the patient's origin and imaging morphology, cystic echinococcosis caused by *E. granulosus* was initially assumed. After initiating albendazole therapy, hemihepatectomy was performed. Unexpectedly, molecular analysis demonstrated >99% concordance with *E. multilocularis,* confirming a diagnosis of alveolar echinococcosis (AE) acquired in Germany.
Conclusion: Migrants may acquire endemic zoonoses including AE in Central Europe, assumptions based on geographic origin may thus be misleading. Molecular species confirmation is helpful when imaging and serology do not unequivocally differentiate *Echinococcus* species.

1. Introduction

Echinococcosis is caused by larval stages of cestodes of the genus *Echinococcus*. The two principal forms in humans are cystic echinococcosis (CE), caused by *E. granulosus sensu lato*, and alveolar echinococcosis (AE), caused by *E. multilocularis*. These entities differ in geographic distribution, clinical presentation, prognosis, and management. CE is globally distributed and endemic in the Mediterranean basin, the Middle East, Central Asia, and Sub-Saharan Africa, including the Horn of Africa. AE occurs mainly in the northern hemisphere including southern Germany as an endemic region [1].

E. multilocularis maintains a sylvatic lifecycle between foxes and small rodents. Humans become accidental intermediate hosts through accidental ingestion of parasite eggs shed by infected foxes or dogs via contaminated soil, berries or leafy vegetables, water, or direct and indirect contact with infected definitive hosts [2]. However, the relative importance of these transmission routes remains unclear, and the source of infection is generally impossible to determine because of the prolonged incubation period.

AE carries a substantially worse prognosis than CE. Untreated disease may show infiltrative hepatic growth with metastatic spread and historically exceeded 90% ten-year mortality [3,4]. Consequently, species identification directly influences surgical management, antiparasitic treatment duration, prognosis, and follow-up strategies [3,5].

In clinical routine, geographic origin of the host is frequently used to infer the causative *Echinococcus* species. Patients from *E. granulosus*-endemic regions are often assumed to have CE, while AE may not be actively considered. This assumption can be misleading after prolonged residence in Central Europe, where AE is endemic. We report a case of molecularly confirmed autochthonous AE in a patient from Eritrea who was initially treated for presumed CE, highlighting a diagnostic pitfall in migrant medicine and the importance of molecular species confirmation.

Abbreviations: AE, alveolar echinococcosis; CE, cystic echinococcosis; PCR, polymerase chain reaction; PET-CT, positron emission tomography-computed tomography; ABZ, albendazole; CEUS, contrast enhanced ultrasound.

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2. Case presentation

2.1. Patient history and clinical presentation

A 33-year-old male patient from Eritrea originally presented to a regional hospital with an acute abdomen. The patient retrospectively reported fatigue without other preceding symptoms. Exploratory laparotomy was performed to exclude relevant bowel ischemia after a diagnosis of Dunbar syndrome via CT imaging. As an incidental finding, abdominal ultrasound showed a cystic lesion with a calcified rim in hepatic segment VIII (approximately 4 × 4 cm). The finding raised initial suspicion of echinococcosis. A supplementary PET-CT showed mild peripheral uptake, interpreted as consistent with a moderately active echinococcal cyst. Contrast enhanced ultrasound (CEUS) showed no hyperenhancement. Liver enzymes were within normal limits; eosinophil count was borderline elevated. Serology for *Echinococcus* (ELISA) was initially negative. Sonographic follow-up was recommended.

Since his arrival in Germany, the patient had not traveled outside Bavaria and reported no visits to friends or relatives abroad. He had been working as a kitchen assistant and denied any history of gardening, animal contact, or significant outdoor activities. After arriving in Bavaria, he initially lived in a rural area and regularly went for walks.

2.2. Progression and re-evaluation

At a follow-up assessment in the following year, mild lesion growth was noted. Repeat serology was now positive for *Echinococcus* IgG (Western blot) without species differentiation. After referral to an infectious disease specialist, antiparasitic therapy with albendazole [dose: 400 mg twice daily] was initiated. Based on the patient's country of origin, infection with *E. granulosus* was presumed.

At first presentation to our infectious disease outpatient clinic at the University Hospital Regensburg, MRI of the abdomen demonstrated a polycyclically demarcated cystoid structure in the right hepatic lobe, measuring 6 × 4.5 × 4 cm, morphologically consistent with an echinococcal lesion see Fig. 1. Repeat serology was still unable to differentiate between species. The clinical working diagnosis remained *E. granulosus* CE (PNM-Staging P1 N0 M0), and in a multidisciplinary board surgical resection was chosen as definitive treatment.

2.3. Surgery and molecular diagnosis

Right hemihepatectomy was performed, and histopathology demonstrated the characteristic acellular laminated membrane of echinococcosis.

Molecular genetic analysis of the resected tissue was performed using PCR targeting the mitochondrial 12 S rDNA gene as previously described [6], followed by sequencing and alignment against the NCBI database, yielding a 99% sequence match with *Echinococcus multilocularis*(see Fig. 2). Retrospectively, the diagnosis was revised to autochthonous alveolar echinococcosis acquired in Germany. The patient is currently in good general condition and remains under postoperative follow-up. Albendazole therapy is planned to be continued for at least two years after resection in accordance with current guidelines [7] for AE.

2.4. Timeline

Date	Event/Intervention	Diagnostic/Clinical detail
July 2015	Relocation from Eritrea to Germany (No previous travel history outside the country.)	no return travel until date of diagnosis
2021		Onset of fatigue symptoms
July 2023	Acute abdomen → regional hospital	CT: Dunbar syndrome; exploratory laparotomy; incidental hepatic cyst segment VIII (~4 × 4 cm), calcified rim
July 2023	PET-CT + CEUS	Mild peripheral uptake; no hyperenhancement on CEUS; serology negative; Aminotransferases normal; borderline eosinophilia
Oct 2024	Lesion growth on follow-up US	Serology positive (Western blot) — no species differentiation
Dec 2024	ID consultation (external)	Albendazole initiated; *E. granulosus* assumed based on patient's origin
Aug 2025	first presentation to our clinic	MRI: polycystic lesion 6 × 4.5 × 4 cm right lobe; repeat serology: no species differentiation; indication for surgery
Dec 2025	Right hemihepatectomy	Histology: laminated membrane; PCR + sequencing → 99% *E. multilocularis*

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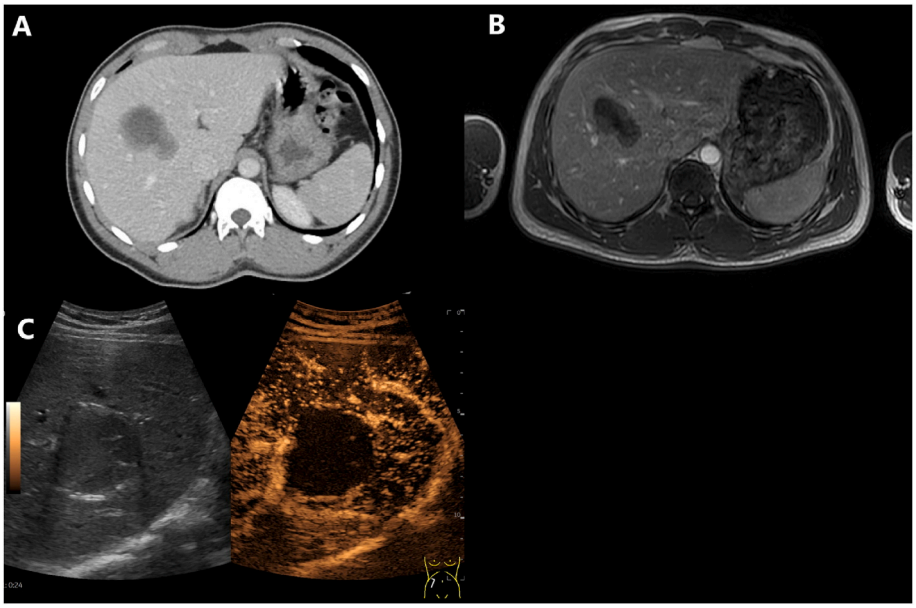

Fig. 1. 1 A - CT Abdomen: incidental hepatic cyst segment VIII (~4 × 4 cm), calcified rim
1B - MRI: polycystic lesion 6 × 4.5 × 4 cm in the right lobe
1C - CEUS showed no hyperenhancement.

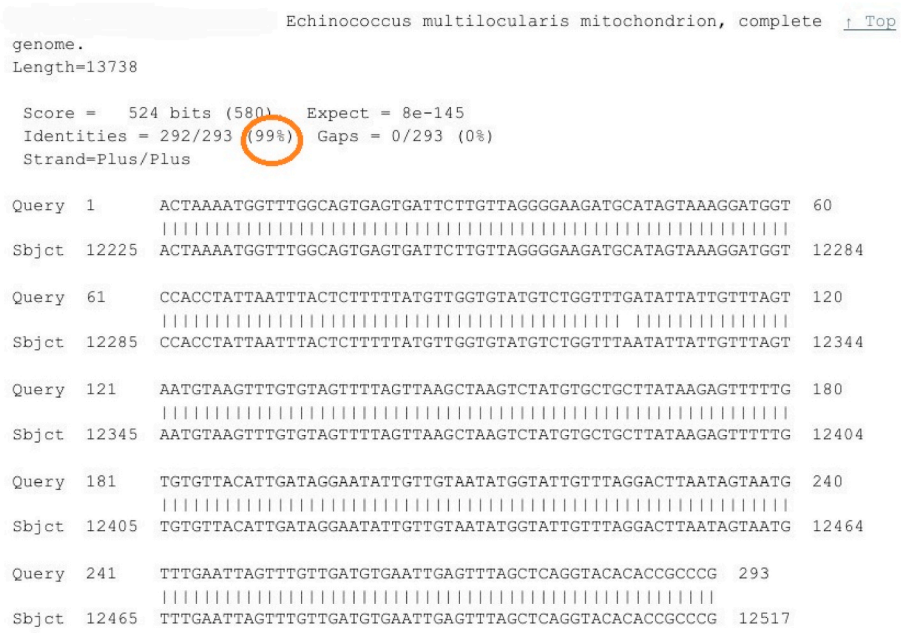

Fig. 2. Molecular genotyping of the intraoperative specimen identified *Echinococcus multilocularis*.

(*continued*)

Date	Event/Intervention	Diagnostic/Clinical detail
Current	Postop follow-up	Good general condition; Albendazole to be continued ≥2 years post-resection; imaging surveillance planned

3. Discussion

This case illustrates the forms of diagnostic anchoring that are particularly prevalent in migrant medicine: the tendency to attribute an infectious etiology to the patient's region of origin. The patient came from Eritrea, where *E. granulosus* CE is endemic [8] and *E. multilocularis* has not been described [8]. This background information guided clinical reasoning towards CE over a two-year period. The natural history of AE involves a median of 5-15 years from infection to clinical manifestation due to slow hepatic infiltration [9]. The patient's years of residence in southern Germany --- an established *E. multilocularis* endemic region --- were therefore sufficient for local acquisition and lesion development before initial detection.

Both imaging morphology and serology proved insufficient for definitive species discrimination in this case. The lesion's polycystic, partially calcified appearance on CT and MRI can be seen in both CE (particularly CE3b–CE5 under WHO-IWGE classification) and AE [10–12]. Cross-reactivity between *E. granulosus* and *E. multilocularis* antigens in standard IgG assays is documented and limits serological species differentiation [13]. Here, PCR and sequencing of DNA from resected tissue provided the definitive answer. Targeting mitochondrial markers allows unambiguous species and genotype identification with high sensitivity. This result changed the postoperative management: while CE would require albendazole for a defined, typically shorter course, the diagnosis of AE leads to a minimum of two years of adjuvant benzimidazole therapy post-resection and long-term imaging surveillance given the risk of recurrence and metastatic spread [14].

Southern Germany, and Bavaria in particular, is one of the regions in Europe where *E. multilocularis* is highly endemic. Red fox population density and prevalence of *E. multilocularis* in foxes in this region are among the highest documented in Central Europe [15]. Urbanization of fox populations has further extended the geographic risk zone beyond rural areas [15,16]. Transmission to humans occurs via ingestion of eggs shed in the feces of foxes and other canids, most commonly through contact with dogs that hunt and consume rodents, but also through gardening, berry or mushroom foraging, contact with contaminated soil, or consumption of unwashed produce [17,18]. These exposure routes are entirely independent of the patient's country of origin.

Migrants are not only at risk from pathogens endemic to their countries of origin - they are also continuously exposed to the infectious environment of their country of residence. This basic principle may easily be overlooked in clinical practice when the patient's background biases the differential diagnosis towards imported conditions. Analogous situations exist for other Central European zoonoses: tick-borne encephalitis, Lyme disease, hantavirus infections, and others can all affect migrants residing in endemic regions of Central Europe. The differential diagnosis in any patient with prolonged European residence should routinely include locally endemic pathogens, weighted by actual exposure history rather than country of birth.

For echinococcosis specifically, the European Centre for Disease Prevention and Control (ECDC) surveillance data from 2022 showed that of cases with available travel information, approximately two-thirds were acquired domestically or within the EU/EEA [15]. Disaggregation by migration background is not routinely reported, leaving an important epidemiological data gap. Cases such as the one presented here may contribute to misclassification if molecular confirmation is not pursued.

4. Conclusion

This case of molecularly confirmed *E. multilocularis* infection in a long-term resident from an *E. granulosus*-endemic country demonstrates that geographic origin is an unreliable proxy for echinococcal species attribution after prolonged residence in Central Europe. PCR-based molecular diagnosis was essential for definitive species differentiation and informed postoperative management. Clinicians should maintain *E. multilocularis* in the differential diagnosis of hepatic lesions in any patient residing in southern Germany, irrespective of migration background. When imaging and serology are inconclusive, molecular testing of tissue material should be considered.

CRediT authorship contribution statement

Tomáš Palla: Writing – original draft, Investigation. **Klaus Brehm:** Methodology, Investigation. **Thomas Glück:** Investigation. **Frank Hanses:** Writing – review & editing, Resources, Conceptualization.

Ethics declaration

Written informed consent to take part in the study and to publish the article has been obtained from all participants or their legal representatives. The privacy rights of participants have been observed.

This study was performed in compliance with relevant laws, regulatory frameworks and guidelines where the research took place. Ethics committee approval was not required under relevant laws and institutional guidelines. Ethics approval was not required for this case report in accordance with applicable German laws, regulatory frameworks, and institutional guidelines. Written informed consent for publication of the case and accompanying clinical information was obtained from the patient.

This research follows the CARE guidelines and the CARE checklist.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Data availability

The data that support the findings of this case report are not publicly available due to patient privacy and confidentiality restrictions. Further information may be available from the corresponding author upon reasonable request, subject to applicable privacy and ethical restrictions.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) for language editing and proofreading. The authors reviewed and edited the output and take full responsibility for the final content of the manuscript.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Robert Koch-Institut. Echinokokkose [internet]. Berlin: RKI; [cited 2026 Mar 31].
[2] Robert Koch-Institut. Alveoläre Echinokokkose: eine Herausforderung für Diagnostik, Therapie und klinisches Management. Epidemiol Bull 2019;41:423–30 [cited 2026 Mar 31].
[3] Brunetti E, Kern P, Vuitton DA, et al. Expert consensus for the diagnosis and treatment of cystic and alveolar echinococcosis in humans. Acta Trop 2010;114(1):1–16.
[4] Kern P, Ammon A, Kron M, et al. Alveolar echinococcosis in humans: clinical course and outcome. Lancet Infect Dis 2003;3:139–46.
[5] WHO Informal Working Group on Echinococcosis. Guidelines for treatment of cystic and alveolar echinococcosis in humans. Bull World Health Organ 1996;74(3):231–42.
[6] Grimm J, Krickl J, Beck A, Nell J, Bergmann M, et al. Establishing and evaluation of a polymerase chain reaction for the detection of Echinococcus multilocularis in human tissue. PLoS Neglected Trop Dis 2021;15(2):e0009155.
[7] Kern P, et al. WHO-IWGE guidelines for the clinical management of alveolar echinococcosis: PNM classification and follow-up recommendations. Parasitol Int 2017;66:314–22.
[8] Aregawi WG, Levecke B, Ashenafi H, et al. Epidemiology of Echinococcus granulosus sensu lato in the greater Horn Of Africa: a systematic review. PLoS Neglected Trop Dis 2024;18(1):e0011944.
[9] Aydin F, Yalcin A, Karaman A, Sade R, Ozturk G, Alper F. Diagnostic and management perspectives in alveolar echinococcosis: review of literature. Eur Asian J Med 2022;54(Suppl 1):10–5. https://doi.org/10.5152/eurasianjmed.2022.22308.
[10] Renette C, Mendiratta-Lala M, Salgia R, Wong RJ, Sauer BG, Pillai A. ACG clinical guideline: focal liver lesions. Am J Gastroenterol 2024;119(7):1235–71. https://doi.org/10.14309/ajg.0000000000002857.
[11] Zhang H, Lai L, Zhang L, Lai L, Zhang C, Zhu Y, et al. CT imaging features and diagnostic algorithm for hepatic cystic echinococcosis. Abdom Radiol 2024;49(3).
[12] Kantarci M, Bayraktutan U, Karabulut N, Aydinli B, Ogul H, Yuce I, et al. Alveolar echinococcosis: spectrum of findings at cross-sectional imaging. Radiographics 2012;32(7):2053–70. https://doi.org/10.1148/rg.327125708.
[13] Schweiger A, Grimm F, Tanner I, Müllhaupt B, Bertogg K, Müller N, et al. Serological diagnosis of echinococcosis: the diagnostic potential of native antigens. Infection 2012;40(2):139–52. https://doi.org/10.1007/s15010-011-0205-6.
[14] McManus DP, Zhang W, Li J, Bartley PB. Echinococcosis. LANCET (N AM ED) 2003;362(9392):1295–304. https://doi.org/10.1016/S0140-6736(03)14573-4.
[15] Casulli A, Abela B, Petrone D, Šoba B, Dezsényi B, Karamon J, et al. Unveiling the incidences and trends of alveolar echinococcosis in Europe: a systematic review from the KNOW-PATH project. Lancet Infect Dis 2026;26(1):e49–61. https://doi.org/10.1016/S1473-3099(25)00283-X.
[16] Freuling CM, Hampson K, Selhorst T, Schröder R, Meslin FX, Mettenleiter TC, et al. The elimination of fox rabies from Europe: determinants of success and lessons for the future. Philos Trans R Soc Lond B Biol Sci 2013;368(1623):20120142. https://doi.org/10.1098/rstb.2012.0142.
[17] Conraths FJ, Probst C, Possenti A, Boufana B, Saulle R, La Torre G, et al. Potential risk factors associated with human alveolar echinococcosis: systematic review and meta-analysis. PLoS Neglected Trop Dis 2017;11(7):e0005801. https://doi.org/10.1371/journal.pntd.0005801.
[18] Kern P, Ammon A, Kron M, Sinn G, Sander S, Petersen LR, et al. Risk factors for alveolar echinococcosis in humans. Emerg Infect Dis 2004;10(12):2088–93. https://doi.org/10.3201/eid1012.030773.