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Case report

Disseminated hepatic and peritoneal hydatidosis mimicking liver cirrhosis: A case report

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Abstract

Introduction: Hydatidosis, caused by Echinococcus granulosus, is a zoonosis endemic to rural areas of Peru that rarely presents as a condition mimicking portal hypertension. **Case presentation:** This is the case of a 47-year-old woman from Cerro de Pasco with a four-year history of progressive abdominal distension and fatigue, initially diagnosed as liver cirrhosis due to chronic ascites. Computed tomography revealed multiple hepatic cysts and massive peritoneal collections; Western Blot serology confirmed the diagnosis of disseminated hydatidosis. The team performed an exploratory laparotomy and drained more than 10 liters of hydatid content, followed by a laparoscopic reintervention because of infectious complications. After continuous treatment with albendazole, the patient showed a favorable clinical course at nine months. **Conclusions:** Clinicians should consider disseminated hydatidosis in the differential diagnosis of chronic ascites in patients from high-Andean regions. Successful treatment depends on comprehensive medical-surgical management and long-term follow-up.

Keywords

Echinococcus granulosus, echinococcosis, liver cirrhosis, portal hypertension, zoonosis.

INTRODUCTION

Hydatidosis, or cystic echinococcosis, remains a critical public health challenge in regions with extensive livestock farming, where the transmission cycle between canids and herbivores persists because of inadequate sanitary and environmental conditions. This zoonotic disease is caused by the larval stage of the cestode *Echinococcus granulosus* and represents one of the most complex scenarios for this disease in Latin America in Peru[1,2]. In departments of the central Andes, such as Pasco and Junín, prevalence in rural communities may reach 9%. This figure reflects not only close contact with definitive hosts but also key risk factors such as limited access to safe water and the continued practice of feeding dogs raw viscera[2,3].

Humans become accidental hosts after ingesting eggs excreted in the feces of infected dogs. Once inside the body, oncospheres cross the intestinal barrier and travel through the bloodstream before lodging, in most cases, in the liver (50%–70%) or lungs (20%–40%)[1-3]. However, the parasite's behavior is unpredictable, and it can affect virtually any organ system.

Peritoneal hydatidosis is a particularly complex presentation, with a frequency of only 5%–16%. This presentation usually results from the rupture, either spontaneous or traumatic, of preexisting hepatic cysts[4]. Because of its rarity

and nonspecific symptoms, it can be mistaken for chronic ascites of other etiologies, such as abdominal tuberculosis, peritoneal carcinomatosis, or even decompensated liver cirrhosis[5].

Accurate diagnosis requires a high degree of clinical suspicion supported by imaging and serologic testing. Although ultrasonography is usually the first step because of its accessibility, computed tomography (CT) is the modality that best defines the extent of peritoneal involvement and guides surgical planning[6]. In cases of disseminated disease, tests such as Western blot are crucial for confirming the etiology[7].

CASE PRESENTATION

A 47-year-old woman from a rural community in Cerro de Pasco, with no known medical history or significant harmful habits, was evaluated. She reported that her illness had begun four years before admission with progressive abdominal distention and mild right upper quadrant pain. During an initial evaluation at a private healthcare facility, collateral circulation and shifting dullness were noted. Abdominal ultrasonography showed increased hepatic echogenicity and an anechoic collection in the right upper quadrant, interpreted as loculated ascites. Diagnostic paracentesis yielded clear fluid with the following values: adenosine deaminase (ADA), 6 U/L; glucose, 29.36 mg/dL; total protein, 0.05 g/dL; and albumin, 0.01 g/dL. With a serum-ascites albumin gradient (SAAG) of 3.10 and limited availability of additional testing because of the restrictions imposed during the COVID-19 pandemic, a presumptive diagnosis of liver cirrhosis was established. Geographic barriers to accessing higher-complexity healthcare facilities from her place of residence also prevented subsequent medical follow-up and completion of additional diagnostic studies.

Four months before admission to our hospital, the patient developed general malaise, fatigue, anorexia, a subjective febrile sensation, and a marked increase in abdominal girth. Her condition progressed to constant abdominal pain, postprandial fullness, and weight loss. These symptoms resulted in severe functional impairment and prompted her transfer to a referral center in Lima.

On admission, she was conscious, afebrile, and hemodynamically stable, with signs of mild malnutrition. On directed history-taking, she denied alcohol use, obesity, and known risk factors for viral hepatitis. Marked abdominal distention with collateral circulation was noted (**Figure 1**). However, physical examination revealed no jaundice, pruritus, peripheral edema, palmar erythema, telangiectasias, or asterixis. The abdomen was distended and tender to deep palpation, with no palpable splenomegaly or signs of peritoneal irritation.

Initial laboratory tests revealed a cholestatic pattern and moderate hepatocellular injury: alkaline phosphatase, 827.3 U/L (reference range [RR], 40–129 U/L); γ-glutamyl transferase, 280 U/L (RR, 8–61 U/L); aspartate aminotransferase (AST), 50.7 U/L (RR, <35 U/L); and alanine aminotransferase (ALT), 42.9 U/L (RR, <35



Figure 1. Clinical image of the patient on admission. Marked generalized abdominal distention with tense skin, everted umbilicus, and whitish striae is observed, consistent with a chronically enlarging mass. Image courtesy of the authors.

U/L). For the differential diagnosis, the absence of typical markers of hepatocellular failure or cirrhotic portal hypertension was a key finding. There was no thrombocytopenia (platelet count, 245,000/μL), prothrombin time was normal (12 seconds), and total bilirubin remained at 0.55 mg/dL. Other findings included a serum albumin level of 3.11 g/dL (RR, 3.5–5.2 g/dL) and a C-reactive protein level of 7.78 mg/L (RR, <5 mg/L). Serologic tests for human immunodeficiency virus (HIV), hepatitis B, and hepatitis C, as well as the hepatic autoantibody profile and tumor markers, were negative.

Admission abdominal ultrasonography showed a multiloculated cystic lesion in the right upper quadrant that displaced the liver, as well as complex collections in the right flank and subhepatic region (**Figure 2**). Portal Doppler ultrasonography ruled out signs of portal hypertension. Subsequent contrast-enhanced CT revealed a multiloculated hepatic mass involving both lobes (210 × 180 mm) and a large, similar intraperitoneal mass (300 × 250 mm). These findings were consistent with disseminated hepatic and peritoneal hydatidosis (**Figure 3**). The diagnosis was confirmed by Western blot for *Echinococcus granulosus* (8-, 16-, and 21-kDa bands).

Treatment with albendazole (800 mg/day) was initiated, followed by exploratory laparotomy. During surgery, a 40 × 30 cm peritoneal cyst containing multiple daughter cysts and purulent material was identified. A 30 × 20 cm hepatic cyst involving segments IVa, VI, VII, and VIII was also found, along with a third cyst in the right fallopian tube that communicated with the peritoneal cavity. In total, more than 10 liters of hydatid fluid were drained.

During the postoperative period, the patient developed residual collections. Two months after admission, a follow-up CT scan showed a 513-mL subdiaphragmatic collection containing air (**Figure 4**). Because of persistent purulent drainage, the patient underwent a second laparoscopic procedure for peritoneal lavage and adhesiolysis. After nine months of follow-up and albendazole therapy, follow-up CT showed resolution of the lesions. Only an encapsulated subcapsular collection remained, without clinical repercussions. Overall, the patient had a favorable clinical course (**Figure 5**).

DISCUSSION

This case illustrates an unusual presentation of cystic hydatidosis in which massive peritoneal dissemination mimicked decompensated liver cirrhosis. This diagnostic mimicry represents a major challenge because ascites is commonly associated with chronic liver disease, whereas a parasitic

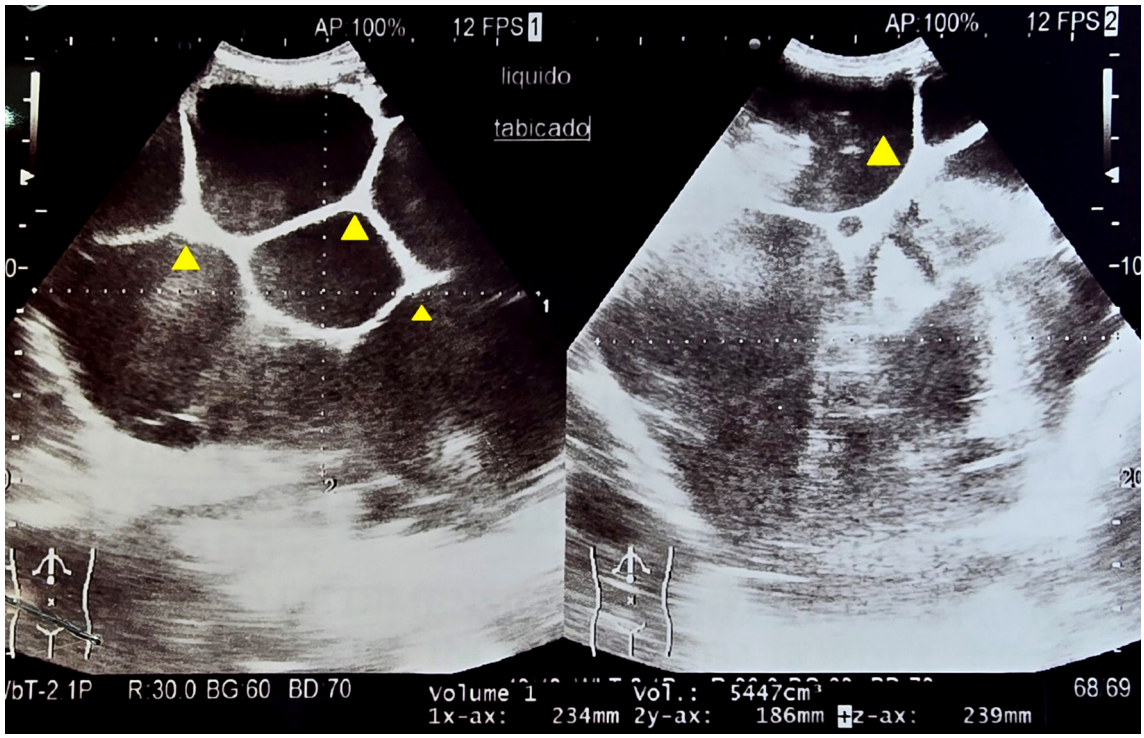


Figure 2. Abdominal ultrasonography. Multiple multiloculated, septated cystic-appearing lesions (arrowhead) with anechoic contents are identified. No solid excrescences are observed within the septa. Image courtesy of the authors.

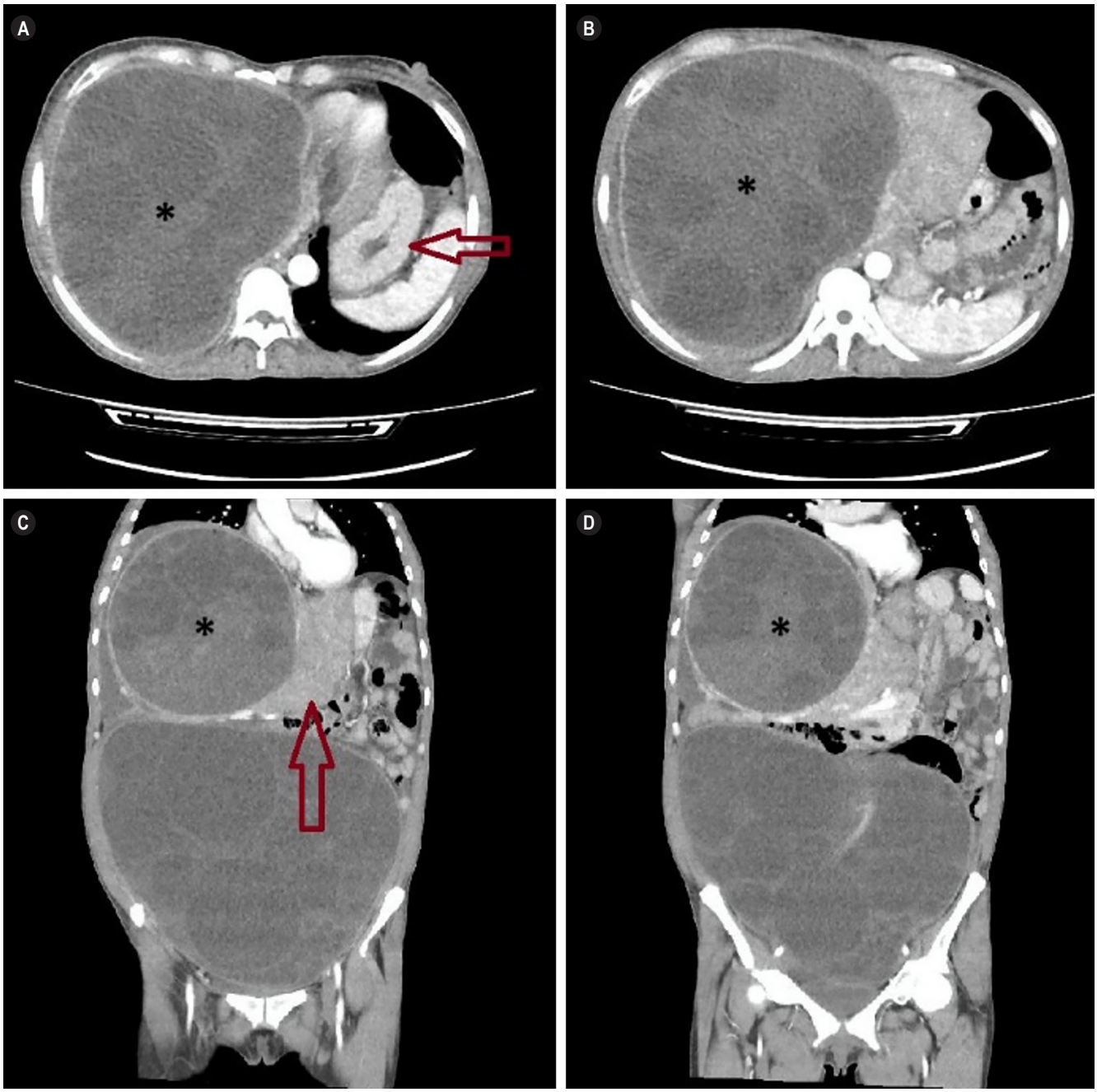


Figure 3. Preoperative contrast-enhanced computed tomography (CT) of the abdomen and pelvis. **A** and **B.** Axial sections: giant mass (*) compressing and displacing the stomach and solid viscera. **C** and **D.** Coronal reconstructions: extension of the lesion from the diaphragm to the pelvis. Images courtesy of the authors.

etiology is uncommon and rarely considered in clinical practice in urban settings. This can lead to treatment delays and increase the risk of iatrogenic complications[5,8].

A critical finding was the elevated serum-ascites albumin gradient (SAAG) (≥1.1 g/dL). Together with collateral circulation and tense ascites, this finding erroneously suggested cirrhotic portal hypertension. Although an elevated SAAG has greater than 90% accuracy for identifying portal hypertension, it is not synonymous with cirrhosis[5,8]. In this setting, extensive occupation of the hepatic parenchyma by multiple cysts may cause presinusoidal portal hypertension. This phenomenon results from direct

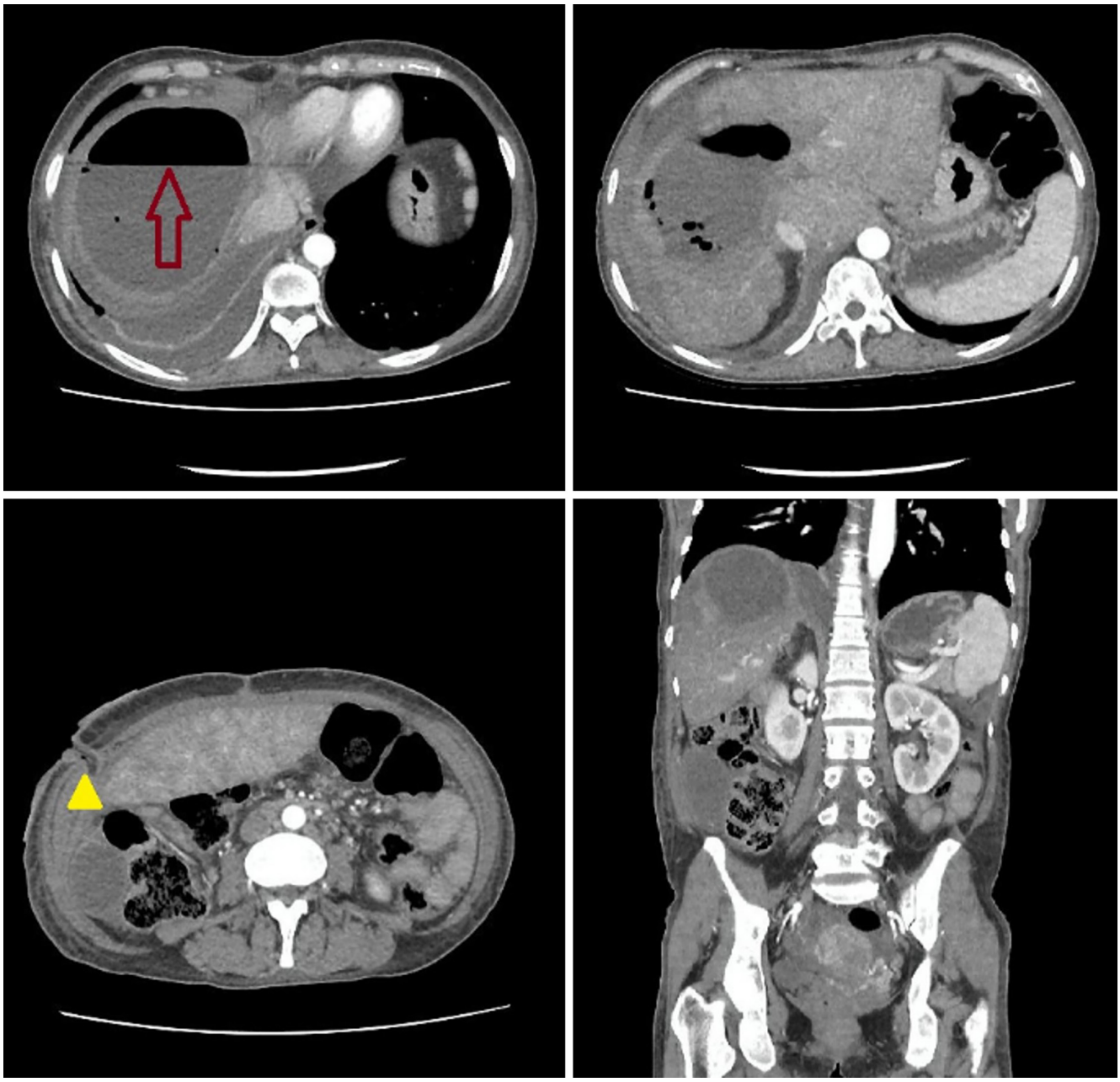


Figure 4. Follow-up CT at 2 months. Persistent abscessed collections with air content (arrow) and an external drainage tube (arrowhead) in the right flank are observed. Image courtesy of the authors.

mechanical compression of the intrahepatic vascular tree or the portal vein at the hepatic hilum. It has been documented in long-term follow-up series as a determinant of morbidity in advanced hydatidosis[9,10]. Disseminated peritoneal hydatidosis may also disrupt the hydrostatic balance and lymphatic drainage within the abdominal cavity. This mechanism, together with the mass effect, could explain the hemodynamic features that mimicked end-stage liver disease despite structurally preserved hepatic parenchyma[10].

Peritoneal hydatidosis is usually a secondary form resulting from rupture of hepatic cysts, whether spontaneous, traumatic, or iatrogenic[4-6,9,10]. In our patient, the paracentesis performed four years before admission under the presumption of cirrhosis may have promoted peritoneal seeding or worsened the condition by facilitating inadvertent

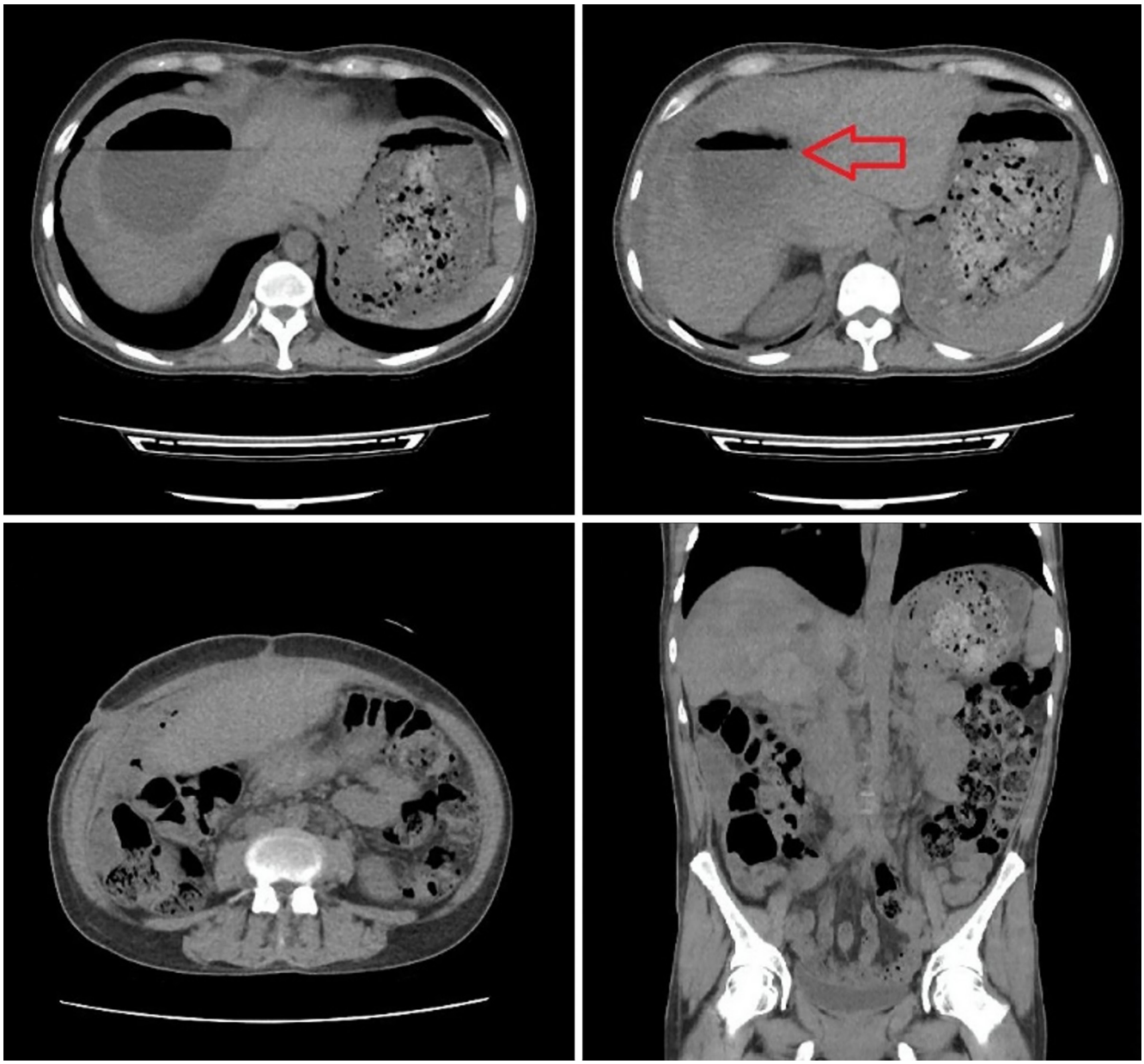


Figure 5. Follow-up CT at 9 months. Near-complete resolution of the disease is observed, with a small residual subcapsular collection (arrow) and a favorable clinical course. Image courtesy of the authors.

release of cystic contents into the abdominal cavity. Because rupture of small cysts may be clinically silent and may not trigger allergic reactions or overt peritonitis, daughter cysts can implant and develop slowly. This can produce chronic ascites that is difficult to characterize initially, as observed in our patient[4,9].

The epidemiologic context should also be emphasized. The patient came from an Andean region of Peru where hydatidosis remains endemic. This should have increased clinical suspicion from the outset[1-3]. The epidemiology of hydatidosis in Peru shows a focal distribution in rural areas of the southern and central regions of the country, with considerable underreporting and limited active surveillance[2,3]. This reality requires physicians, even in urban areas, to maintain a high index of clinical suspicion in patients from endemic regions. In this case, the impact of the COVID-19 pandemic compounded the patient's geographic barriers to healthcare access and created a dual barrier that prevented timely follow-up. This situation not only delayed specialist evaluation but also restricted access

to more sensitive imaging modalities, such as contrast-enhanced CT. As a result, etiologic diagnosis was delayed, allowing the disease to progress silently.

From a diagnostic standpoint, the misinterpretation of the initial abdominal ultrasonography underscores the need for standardized assessment[1]. Visualization of heterogeneous lesions within the hepatic parenchyma may lead operators who are unfamiliar with the Gharbi or World Health Organization (WHO) classifications to misdiagnose structural cirrhosis[3,4,11,12]. In contrast, contrast-enhanced CT served as the reference standard in this case. It allowed the extent of multiorgan involvement to be defined and facilitated surgical planning[6,13]. Western blot confirmation was also essential. Its high specificity for detecting antibodies against specific antigens, such as p7, supports confirmation of active disease even at atypical sites[2,5,6].

The therapeutic management of disseminated disease requires a dual approach. Exploratory laparotomy allowed drainage of more than 10 liters of cystic contents and resection of complicated cysts. Therefore, it is the treatment of choice in cases of extensive involvement or secondary infection[12,14]. Continuous albendazole administration (10–15 mg/kg/day) was also essential to reduce protoscolex viability and minimize the risk of recurrence. This risk is significantly higher in peritoneal hydatidosis than in isolated hepatic disease[12,15].

Finally, this case highlights barriers to healthcare access and the persistent impact of neglected infectious diseases in Peru. Although residence in an endemic area (Pasco) should have prompted early clinical suspicion, geographic dispersion and the restrictions imposed during the COVID-19 pandemic delayed the diagnosis. The interruption of surveillance programs during the public health crisis exacerbated vulnerability in rural populations and compromised the goal of disease elimination by 2030[16]. Strengthening epidemiologic surveillance and clinical suspicion at the primary care level is imperative to prevent misdiagnosis at advanced stages.

CONCLUSIONS

Massive hepatic and peritoneal hydatidosis should be systematically included in the differential diagnosis of chronic ascites and portal hypertension in patients from high-Andean regions. Diagnostic success depends on the general practitioner recognizing the absence of classic stigmata of cirrhosis, including jaundice, splenomegaly, thrombocytopenia, and prolonged prothrombin time, before performing invasive procedures. This case demonstrates that severe cystic involvement can mimic liver cirrhosis both clinically and hemodynamically, as evidenced by an elevated SAAG. This can lead to diagnostic errors and potentially hazardous procedures, such as inadvertent paracentesis. Paracentesis should be avoided when cystic masses are suspected to prevent peritoneal seeding or anaphylaxis.

Ethical considerations

This case report was prepared in accordance with the ethical principles established in the Declaration of Helsinki. Patient confidentiality and anonymity were preserved. No procedures were performed beyond those required for clinical care solely for the purposes of this publication.

Informed consent

Written informed consent was obtained from the patient for publication of her clinical case, as well as the radiologic images and intraoperative photographs obtained during her care. Patient anonymity and confidentiality were ensured by removing any information that could identify her from the text and figures presented.

Conflicts of interest

The authors declare no conflicts of interest.

Use of artificial intelligence

The Gemini language model (Google) was used exclusively as a support tool for stylistic improvement, writing, and technical translation of the manuscript. The authors fully reviewed and validated the final text and assume full responsibility for its content and conclusions.

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Author contributions

- Study design and conception: Anna Ángeles and Johan Azañero-Haro.
- Data acquisition: Valeria Aponte and Quesly Cachay.
- Data analysis and interpretation: Johan Azañero-Haro and Anna Ángeles.
- Manuscript drafting: Anna Ángeles, Valeria Aponte, Quesly Cachay, and Johan Azañero-Haro.
- Critical revision of the manuscript: Anna Ángeles, Valeria Aponte, Quesly Cachay, and Johan Azañero-Haro.

REFERENCES

1. Azañero-Haro J, Reyes J, Castro F, Salazar L, Soto A. Hidatidosis diseminada con afectación pulmonar, hepática, pancreática y esplénica: a propósito de un caso. Rev Cuerpo Med HNAAA. 2023;16(3):e1932. https://doi.org/10.35434/rcmhnaaa.2023.163.1932
2. Sanchez L, Mayta H, Jara LM, Verástegui M, Gilman RH, Gómez-Puerta LA, et al. *Echinococcus granulosus sensu stricto* and *E. canadensis* are distributed in livestock of highly endemic area in the Peruvian highlands. Acta Trop. 2022;225:106178. https://doi.org/10.1016/j.actatropica.2021.106178
3. Acosta-Jamett G, Tamarozzi F, Castro N, Santivanez SJ, Laurente RE, Mazzi C, et al. Community risk of environmental-borne cystic echinococcosis transmission in South America: Results from the multistep cross-sectional and case-control PERITAS study. PLoS Negl Trop Dis. 2025;19(8):e0013382. https://doi.org/10.1371/journal.pntd.0013382
4. Mihetiu A, Bratu DG, Tanasescu C, Vintilă BI, Sandu A, Sandu M, et al. Laparoscopic management of multiple liver, omental, mesenteric, peritoneal, and round ligament hydatid cysts-a rare report of a case and a systematic literature review. J Pers Med. 2024;14(2):205. https://doi.org/10.3390/jpm14020205
5. Campos NMF, Almeida V, Curvo Semedo L. Peritoneal disease: key imaging findings that help in the differential diagnosis. Br J Radiol. 2022;95(1130):20210346. https://doi.org/10.1259/bjr.20210346
6. Kechiche N, Makhlouf D, Lamiri R, Zouaoui A, Sahnoun L, Mekki M, et al. Peritoneal hydatid cysts in children: a case series of rare echinococcosis localization. Iran J Med Sci. 2021;46(1):68-72. https://doi.org/10.30476/ijms.2020.82004.0
7. Aslan M, Yüksel P, Polat E, Cakan H, Ergin S, Öner YA, et al. The diagnostic value of Western blot method in patients with cystic echinococcosis. New Microbiol. 2011;34(2):173-7.
8. Du L, Wei N, Maiwall R, Song Y. Differential diagnosis of ascites: etiologies, ascitic fluid analysis, diagnostic algorithm. Clin Chem Lab Med. 2023;62(7):1266-76. https://doi.org/10.1515/cclm-2023-1112
9. Kosmidis C, Efthimiadis C, Anthimidis G, Vasileiadou K, Koimtzis G, Tzeveleki I, et al. Management of peritoneal hydatid cysts: a fourty-year experience. Heliyon. 2018;4(12):e00994. https://doi.org/10.1016/j.heliyon.2018.e00994
10. Collado Aliaga J, Romero-Alegría Á, Alonso-Sardón M, Prieto-Vicente V, López-Bernus A, Velasco-Tirado V, et al. Portal hypertension as a complication of cystic echinococcosis: a 20-year cohort analysis. Am J Trop Med Hyg. 2021;105(3):692-7. https://doi.org/10.4269/ajtmh.21-0274
11. Benhamiche H, Sottier D, Funes De La Vega M, Cuisenier B, Mejean N, Krausé D. Peritoneal hydatidosis and hepatic hydatid cyst perforation. Diagn Interv Imaging. 2013;94(11):1157-60. https://doi.org/10.1016/j.diii.2013.04.009
12. World Health Organization. WHO guidelines for the treatment of patients with cystic echinococcosis [Internet]. Geneva: World Health Organization; 2025.
13. Calu V, Enciu O, Toma EA, Pârvuleţu R, Pîrîianu DC, Miron A. Complicated liver cystic echinococcosis-a comprehensive literature review and a tale of two extreme cases. Tomography. 2024;10(6):922-34. https://doi.org/10.3390/tomography10060070
14. Evers G, Polania-Liscano HA, Polania S. Equinococosis abdominal diseminada: reporte de caso. Rev Colomb Gastroenterol. 2022;37(1):108-13. https://doi.org/10.22516/25007440.762
15. Almulhim AM, John S. Echinococcus Granulosus. 2023 Aug 7. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan–.
16. World Health Organization. Global report on neglected tropical diseases 2025: consolidating gains for 2030 targets. Geneva: WHO; 2025. Disponible en: https://www.who.int/publications/i/item/9789240114043