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Case Report

Disseminated hydatid cysts as incidental finding in an unconscious female with abdominal distention, King Saud Medical City, Al-Riyadh, Saudi Arabia

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ABSTRACT

Hydatid disease, which is a parasitic infection caused by *Echinococcus granuloses*, primarily affects the liver and lungs, and there is a rare primary peritoneal involvement (2%). The diagnosis requires imaging (ultrasonography, computed tomography, and magnetic resonance imaging) and serology. Its management encompasses surgery and antiparasitic therapies. Our case underscores the diagnostic challenges, complications, and the importance of early detection and optimal treatment. A 25-year-old Ethiopian female presented with abdominal pains, distensions, and shortness of breath and was diagnosed with disseminated hydatid diseases with portal vein thrombosis. Imaging revealed extensive peritoneal cysts, hepatic involvement, and bronchopleural–peritoneal fistula. She was transferred to King Saud Medical City, where she received antiparasitic medications, anticoagulants, and blood transfusions before undergoing exploratory laparotomy, hydatid cyst excisions, and bladder repair due to intraoperative injury. Postoperatively, she was mechanically ventilated in the intensive care unit, hemodynamically stable, and received multidisciplinary care, including infectious disease, hepatobiliary surgery, and urology specialists. This case underscores the complexity in diagnosing and managing disseminated hydatid disease and thus emphasizes the significance of early detection, imaging, and multidisciplinary care. Surgical excision of the cyst remains essential, whereas antiparasitic therapy and anticoagulation support early recovery, thereby preventing recurrence and vascular complications in extensive peritoneal hydatidosis.

Keywords: Peritoneal hydatid cysts, *E. granulosus*, Hydatid disease, Cystic echinococcosis, Hepatic cyst rupture, Peritoneal involvement

INTRODUCTION

Hydatid disease, a parasitic infection, is caused by the larval stages of *E. granulosus*, a tapeworm that primarily affects humans through accidental parasite egg ingestions.[1] The disease is endemic in livestock-rearing regions, where close interactions between humans, dogs, and sheep facilitate the parasite's life cycle.[2] The liver (60-70%) and lungs (20-30%) are the most commonly affected organs owing to their rich vascular supply.[3] However, primary peritoneal hydatid cysts are rare (2%), frequently occurring secondary to hepatic cyst rupture, spontaneous seeding, or surgical spillage.[4,5] Primary peritoneal hydatidosis is extremely uncommon, making its diagnosis and management particularly difficult.

Hydatid disease is endemic in various regions globally, particularly in the Mediterranean regions, the Middle East, Africa, South America, and Central Asia.[6] In these regions, agricultural communities and animal husbandry practices contribute to the persistence of the disease. Higher infection rates are noted in rural populations

owing to heightened exposure to infected dogs and livestock.

The gradual growth of peritoneal hydatid cysts can lead to years of asymptomatic disease. However, as the cysts enlarge or rupture, complications arise.[7] Cyst rupture represents the most life-threatening complication, causing peritoneal seeding, secondary echinococcosis, and abscess formation, potentially resulting in peritonitis, sepsis, anaphylaxis, bowel obstruction, hydronephrosis, vascular compromise, and surgical challenges due to adhesions.[8]

The diagnosis of peritoneal hydatid cysts requires clinical suspicion, imaging, and serology. Ultrasonography (USG) is the first-line modality, whereas computed tomography (CT) and magnetic resonance imaging (MRI) provide detailed visualization of daughter cysts, calcifications, or membranes.[1] Serological tests (enzyme-linked immunosorbent assay and indirect hemagglutination) facilitate confirmation but may yield false negative results. Fine-needle aspirations are avoided owing to the risk of ruptures and anaphylaxes.

The management of peritoneal hydatid cysts comprises medical therapy, surgical intervention, or a combination of both. To reduce cyst viability and lower the risk of recurrence, albendazole or mebendazole is preoperatively administered.[9] Surgery remains the primary treatment modality, with procedures including open or laparoscopic cystectomy, pericystectomy, or omentoplasty, depending on the cyst size and location.[10] The puncture, aspiration, injection, and reaspiration (PAIR) technique is an alternative for inoperable cases (cysts smaller than 6 cm); however, its use in peritoneal hydatid cysts is limited owing to the risk of peritoneal spread.[10] To minimize recurrences, complete surgical excisions with peritoneal lavage using scolicidal agents are crucial.

Our case highlights a rare peritoneal hydatid cyst, underscoring the diagnostic challenges and complications. Unlike typical cases of hepatic rupture, this case highlights primary peritoneal involvement. Documenting such cases helps raise clinical awareness, facilitates early detection, and informs optimal treatment strategies.

CASE REPORT

A 25-year-old Ethiopian female with unknown past medical history presented to the emergency department (ED) of Al Iman General Hospital on January 9, 2025, after being transferred by the Red Crescent. She reported a 1-week history of generalized abdominal pains, which progressively worsened over time. The pain was diffuse, nonradiating, and associated with abdominal distensions for the past 3 days. Moreover, the patient complained of shortness of breath but denied fevers, chest pains, coughs, hemoptysis, or hematemesis. No history of urinary symptoms, recent travels, sick contacts, outdoor activities, or animal exposure was reported. The patient had been residing in Saudi Arabia for the past 10 years with no previous hospital admissions or known medical conditions.

Figure 1: Abdominal distention with multiple prominent masses on examination.

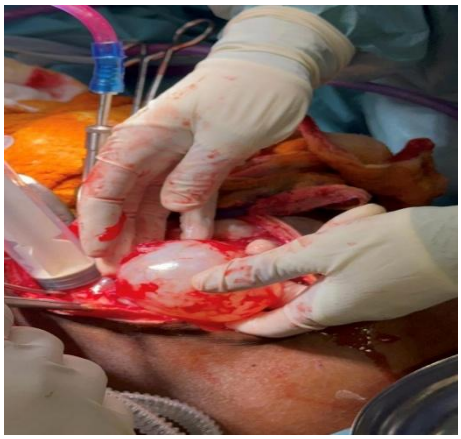

Figure 2: Hydatid cyst observed upon opening the peritoneal cavity.

Figure 3: Hydatid cyst removed from the abdomen.

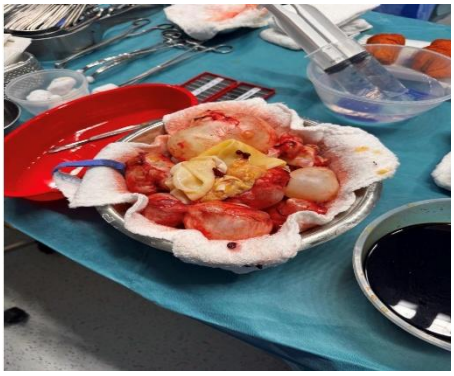

Figure 4: Multiple hydatid cysts dissected from the right side of the abdomen.

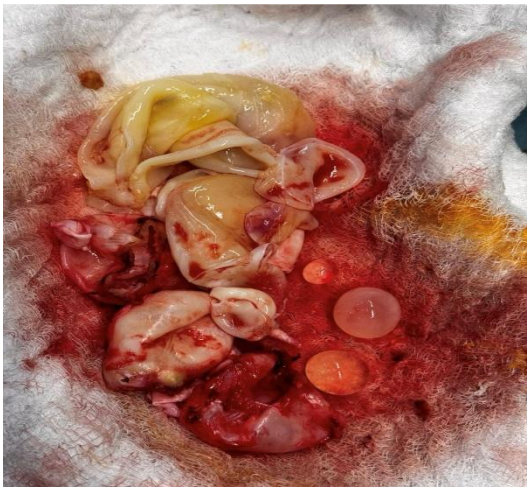

Figure 5: Ruptured hydatid cyst showing multiple daughter cysts.

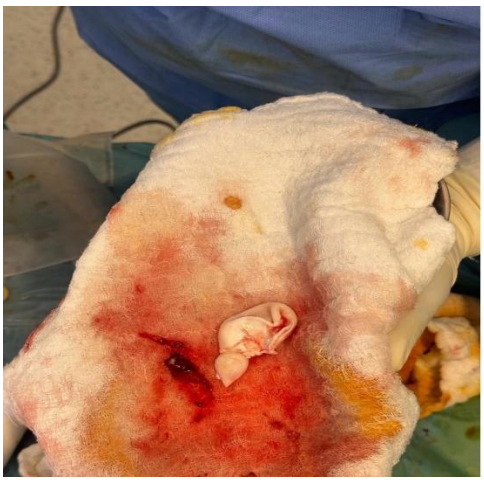

Figure 6: Ruptured hydatid cyst.

The patient was initially evaluated at Al Falah Private Hospital, where an abdominal USG revealed disseminated abdominal hydatidosis with portal vein thrombosis (PVT) and coarse liver textures. Subsequently, she was referred to Al Iman General Hospital for further evaluation and management.

Initial evaluation and management (at Al Iman General Hospital)

Upon ED arrival, the patient appeared clinically stable and was not in distress despite her symptoms. She was conscious, alert, and oriented to time, place, and persons. Her vital signs were within normal limits as follows: temperature, 36.5 °C; blood pressure, 116/60 mmHg; heart rate, 89 beats/min; respiratory rate, 20 breaths/min; and oxygen saturation, 96% on room air. On physical examination, the patient exhibited jaundice, as evidenced by the yellow sclera. Abdominal examination revealed significant distension with multiple prominent masses, which were suspected to be hydatid cysts, although no tenderness was noted (Figure 1). No hepatosplenomegaly was detected on palpation. Bedside USG further confirmed multiple cystic lesions surrounding the liver and uterus.

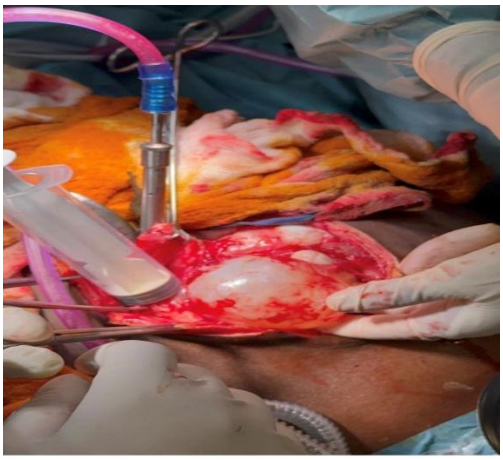

Figure 7: Peritoneal cavity flushing with 18.4% normal saline.

The patient's initial laboratory results showed severe anemia (hemoglobin, 6.1 g/dl), leukocytosis (white blood cells, $13.04\times10^3/\mu l$; neutrophil, 76.7%), and thrombocytosis (platelet, $697\times10^3/\mu l$). Hyponatremia (sodium, 126 mmol/l) and elevated ALP (220 U/L) were noted. Renal function was normal, malaria and β-HCG tests were negative, and the blood type was O+.

The patient was maintained on a nothing-by-mouth status and started on intravenous (IV) fluid at 120 ml/h. Empiric antibiotic therapy was initiated with piperacillin/ tazobactam (Tazocin) 4.5 g IV every 6 h. Owing to her severe anemia, she was transfused with one unit of packed red bloods cells (PRBCs) for 4 h.

Abdominal and pelvic contrast-enhanced CT, performed at King Saud Hospital on January 10, 2025, revealed extensive disseminated hydatid cysts throughout the peritoneal and pelvic cavities. These cysts varied in size and exhibited multiple internal septations and daughter cysts, with some showing peripheral calcifications. The largest cyst, measuring 15×10 cm in the right abdominal

region, demonstrated ruptured lateral borders, which was associated with a significant collection of free intraperitoneal fluids. Moreover, the cysts exerted pressure on adjacent structures, leading to right kidney compression, inferior displacement of the uterus, and a leftward shift of the inferior vena cava and porta hepatis. Additionally, marked intrahepatic biliary radicle dilatations were noted. CT further revealed right lower lobe lung collapse, accompanied by right hemidiaphragmatic elevation, probably secondary to the mass effect from the extensive cystic involvement.

Considering the progressive nature of her condition, an urgent transfer to King Saud Medical City was arranged for definitive surgical intervention. Upon transport, she was closely monitored, with emergency epinephrine, a crash cart, and two units of PRBCs on standby to manage potential decompensation.

Management at King Saud Medical City

Upon arrival, the patient was admitted under general surgery and initiated treatment with a combination of antiparasitic medications, broad-spectrum antibiotics, anticoagulants, and blood transfusions. Her treatment regimen comprised praziquantel 1,200 mg orally three times daily, albendazole 400 mg orally two times daily, piperacillin/tazobactam (Tazocin) 4.5 g IV every 6 h, and heparin 5,000 IU two times daily to prevent thrombotic complications. Owing to her severe anemia, she received additional three units of PRBCs to optimize her hemoglobin level preoperatively.

Repeat abdominal and pelvic contrast-enhanced CT on January 10, 2025 confirmed an extensive hydatid cystic disease with significant mass effects. Other findings encompassed multiple large cystic liver lesions, with the largest cyst measuring 15.5×13.5×19 cm. A potential rupture of the dominant cyst's lower wall was noted, along with mild intrahepatic biliary ductal dilatations. The portal and splenic veins were deviated but remained patent, and numerous peritoneal and pelvic cysts with daughter cysts were detected. Mild-to-moderate free abdominal fluid was observed, suggesting intraperitoneal leakage, whereas right lower lobe lung consolidation and minimal right pleural effusion were also noted.

Chest contrast-enhanced CT on January 13, 2025 further revealed a large intraparenchymal cystic lesion in the right lower lung lobe, highly suggestive of a hydatid disease, with associated right pleural effusion and passive atelectasis. Additionally, mediastinal lymph node enlargement was noted, suggesting a systemic inflammatory response or secondary involvement.

To evaluate potential biliary system involvement, abdominal MRI with magnetic resonance cholangiopancreatography (MRCP) was performed on January 13, 2025. The scan confirmed a ruptured hepatic hydatid cyst with intrabiliary rupture, resulting in moderate intrahepatic biliary dilatations. Several bi-lobed hepatic abscesses were noted, with the largest abscess measuring 2.3 cm. Furthermore, the imaging revealed an extensive peritoneal hydatid disease, with numerous scattered cysts throughout the abdominal cavity, further emphasizing the need for urgent surgical intervention (Figures 8-10).

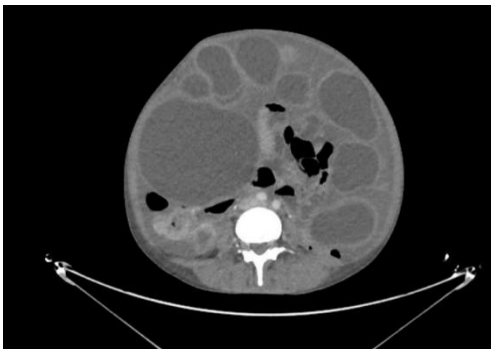

Figure 8: CT of the abdomen, pelvis, and chest multiple hydatid cyst in abdomen cavity.

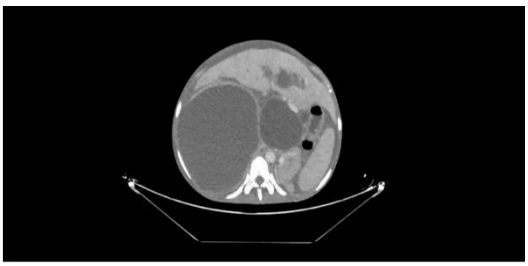

Figure 9: CT of the abdomen, pelvis, and chest large hydatid cyst mass effect.

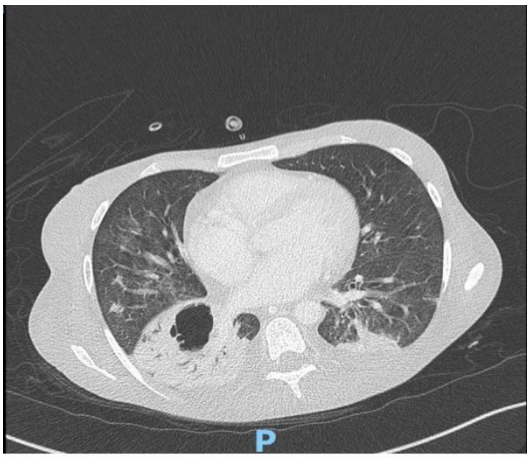

Figure 10: Chest contrast-enhanced CT large intraparenchymal cystic lesion in the right lower lung lobe.

Surgical management

On January 16, 2025, the patient underwent exploratory laparotomy with hydatid cyst surgical excision and deroofing under general anesthesia and total operative

time was 9 h. The abdomen was dissected in layers, revealing ascitic fluid throughout the cavity. Several hydatid cysts were detected, predominantly in the anterior abdominal cavity, and were carefully resected and removed (Figures 2 and 3).

A large ruptured cyst extending to the hepatic flexures was noted, containing daughter cysts, which were aspirated and treated with 18.4% hypertonic saline (Figures 4-6).

Additional multiple cysts attached to the lower abdominal wall were excised; however, one large cyst caused accidental bladder injury, prompting immediate urologic intervention (Figure 7). The bladder was repaired in two layers using running 2-0 Vicryl sutures, and a suprapubic catheter was inserted and fixed. The fascia was closed using loop Vicryl sutures, and four surgical drains were placed, including perigastric, suprapubic, perihepatic, and pelvic. The abdomen was thoroughly irrigated, and the skin was closed using surgical staples. The estimated blood loss was 500 ml, with no intraoperative transfusion required.

Postoperative course and complications

She was transferred to the intensive care unit (ICU) postoperatively, deeply sedated, and mechanically ventilated. On January 27, 2025, abdominal and pelvic CT revealed an interval decrease in peritoneal hydatidosis; however, a 5-cm pelvic cyst, right subdiaphragmatic collection, and loculated abdominopelvic fluid collection persisted. Moreover, a large pneumoperitoneum was noted with no bowel leak, suggesting a bronchopleural fistula, which was later supported by thoracic imaging. On January 30, 2025, the patient underwent ERCP with biliary sphincterotomy and CBD stent placement for the persistent bile leak. On February 1, 2025, the patient's condition rapidly worsened, with increasing oxygen requirements, desaturation, and respiratory distress. She was intubated and subsequently placed on mechanical ventilation. CT pulmonary angiogram revealed no PE. On February 4, 2025, thoracic surgery noted a right lower lobe cavitary lesion communicating with the pleural space (confirmed fistula) and was conservatively monitored. Follow-up MRCP on February 6, 2025 confirmed bile leak into the right subdiaphragmatic collection and reduced collection size. The hydro-pneumoperitoneum increased in volume; however, other collections demonstrated partial regression. On February 9, 2025, CT cystogram revealed no active urinary bladder leak, confirming the healing of the surgical repair. On February 13, 2025, the patient underwent USG-guided drainage of the large abdominal collection by IR, with two 20-F pigtail catheters inserted; the fluid was thick and green, suggesting infected contents. Tracheostomy was also performed on the same day. On February 22, 2025, she remained on the Thermovent with 3 liters/min of oxygen flow via tracheostomy.

Follow-up

The most recent follow-up CT of the abdomen, pelvis, and chest was performed on February 26, 2025. Abdominal and pelvic CT revealed an interval decrease in the size of the large abdominopelvic loculated collection, now measuring 15×9.3 cm compared with 22×12 cm previously. The right subdiaphragmatic collection remained stable in size at 5×2.4 cm, whereas the pelvic bi-lobed collection demonstrated a further decrease in size. A large volume of ascites remained evident; however, no new fluid accumulations were noted. The biliary stent was visualized in a stable position, and all major abdominal vessels, including the hepatic, portal, splenic, and superior mesenteric veins, were patent. Chest CT revealed a further reduction in the size of the peritoneal cyst, which was inseparable from the right lower lobe. The bilateral patchy ground-glass opacities were markedly improved, and the previously noted bilateral lower lobe atelectasis remained stable. Small bilateral pleural effusions persisted, and no evidence of active hydatid cysts was observed in the lungs. Therefore, the imaging findings suggested radiological improvement in both the thoracic and abdominal compartments.

DISCUSSION

Hydatid disease, known as cystic echinococcosis, is a zoonotic infection caused by the larval stages of *E. granulosus*.[11] It primarily affects the liver (95%); however, peritoneal hydatidosis is less common and accounts for only 2-13% of cases.[12] The present case involved a disseminated hydatid disease with intra-abdominal, intrahepatic, intrabiliary, and pulmonary involvement, complicated by cyst rupture, peritoneal seeding, and secondary infection, underscoring the diagnostic challenges and severe complications of advanced hydatidosis.

Notably, this case had extensive peritoneal hydatidosis, a rare and severe echinococcosis form, presenting with ruptured hepatic and peritoneal cysts, resulting in peritoneal spread and multiple cyst seeding. Similarly, Akbulut et al showed that intra-abdominal hydatid cyst rupture with peritoneal seeding is documented but infrequent, accounting for 0.9-16% of cases.[8] Studies including that of Ismail et al have suggested that spontaneous cyst rupture can result from increased intracystic pressure, trauma, or infection, leading to secondary peritoneal echinococcosis.[13]

Moreover, imaging detected PVT, confirming vascular involvement. This complication increases the risk of portal hypertension, liver ischemia, and further hepatic dysfunction, necessitating urgent surgical intervention. Similarly, Kirmizi et al reported that PVT is a rare complication of hepatic hydatid disease caused by mechanical compression, local inflammation, or cyst rupture.[14]

Moreover, this case had a right lower lobe hydatid cyst with associated lung collapse and pleural effusion, possibly attributed to direct extension from intra-abdominal dissemination. This finding is correlated with those of previous studies that reported the association of ruptured hepatic cysts with secondary lung involvement and respiratory distress.[15] Furthermore, imaging revealed a right lower lung lobe cavitary lesion, initially suggestive of pulmonary hydatid disease, but ultimately noted to be communicating with the right pleural space, confirming the presence of a bronchopleural–peritoneal fistula. This complication is extremely rare and probably resulted from the upward extension of intra-abdominal pressure and cyst rupture. The massive pneumoperitoneum in the absence of bowel perforation further supports this diagnosis. Pleuroperitoneal fistulas are sparsely reported in hydatid disease but have been associated with significant respiratory distress and hypoxia, both of which were observed in the present case. Although previous studies have not reported on bronchopleural-peritoneal fistulas, some studies on broncho–biliary fistulas in peritoneal hydatid cysts have been conducted Akbulut et al.[8] Moreover, postrupture anaphylaxis is a known complication, which fortunately did not occur in our patient.[16]

In the present case, bladder injury occurred intraoperatively owing to extensive cystic adhesions to the lower abdomen, requiring primary bladder repair. Kc et al indicated that urinary or pelvic hydatid cysts are rare (2-2.25%), frequently secondary to peritoneal spread and bladder infiltration requiring partial cystectomy.[17] However, in the present case, primary bladder repair was successfully performed intraoperatively, precluding the necessity for more extensive bladder resection.

Notably, in this case, severe abdominal distension, PVT, and lung involvement made the diagnosis difficult. Similarly, previous research Alshoabi et al demonstrated that advanced hydatid disease poses diagnostic challenges owing to atypical presentations mimicking peritonitis or malignancies; however, imaging modalities, including CT and MRI, are essential for early and accurate diagnosis.[1]

Remarkably, our patient underwent exploratory laparotomy, hydatid cyst excision, and deroofing, aligning with the surgical guidelines. The procedure was complicated by intraoperative bladder injury, requiring primary repair, a rare but known complication in extensive hydatid disease. Surgical removal of hydatid cysts remains the gold standard, particularly in ruptured cysts with complications.[18] The PAIR technique is considered in select cases; however, extensive peritoneal involvement requires surgical excision.[19]

Postoperatively, our patient was managed with albendazole and praziquantel, which is the standard therapy for preventing residual cyst growth and recurrence. Albendazole is highly effective against peritoneal hydatidosis and is recommended for at least 3–6 months postoperatively.[20] Anticoagulation with heparin was also initiated owing to PVT, as per current guidelines.[21] The postoperative course of the present case was also complicated by persistent bile leakage, confirmed by MRCP, indicating extravasation into the right subdiaphragmatic collection following hepatic cyst rupture. This event required an ERCP with biliary sphincterotomy and CBD stenting, aligning with the standard management protocols for post-hydatid cyst biliary fistulas.[22]

CONCLUSION

This case highlights the rare and severe presentation of disseminated hydatid disease with multiorgan involvement, including the hepatic, peritoneal, biliary, pulmonary, and urinary systems. Complications, including cyst rupture, PVT, bile leak, and broncho–peritoneal fistula, were managed through a multidisciplinary approach encompassing surgery, ERCP, interventional radiology, antiparasitic therapy, and extended ICU care. Imaging played a crucial role in the diagnosis and follow-up. Despite the complexity, the patient demonstrated steady clinical and radiological improvement, underscoring the significance of early detection and integrated management for advanced hydatidosis.

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