

Liver cirrhosis and complications of chronic fascioliasis in childhood. A case report

Cirrosis hepática y complicaciones de la Fascioliasis crónica en la edad pediátrica. A propósito de un caso

Giovanni Callizaya-Macedo¹ | ORCID: 0009-0002-7509-5537

José Miguel Cussi Romero² | ORCID: 0009-0004-3895-1538

Jhossmar Cristians Auza-Santivañez³ | ORCID: 0000-0002-7703-2241

José Antonio Lagos Monasterios² | ORCID: 0009-0006-2732-8876

Angel Simon Chuquimia Calderon² | ORCID: 0009-0002-2121-924X

María Angela Murillo Alcázar⁴ | ORCID: 0009-0006-6296-9548

Daniela Nicole Pacheco de la Torre⁴ | ORCID: 0009-0002-7192-8095

Freddy Ednildon Bautista-Vanegas^{5,6} | ORCID: 0009-0005-5827-7066

¹ Hospital del Niño "Dr. Ovidio Aliaga Uría". Departamento de Cirugía Pediátrica. La Paz Bolivia.

² Hospital de Clínicas Universitario La Paz. Unidad de Imagenología. La Paz Bolivia.

³ Hospital del Gran Chaco "Fray Quebracho" de III nivel. Instituto Académico Científico Quispe-Cornejo. Tarija, Bolivia.

⁴ Hospital del Niño "Dr. Ovidio Aliaga Uría". Unidad de Imagenología. La Paz, Bolivia.

⁵ Kliniken Beelitz GmbH Neurologische Rehabilitationsklinik: Beelitz Heilstätten, Brandenburg, DE. Alemania.

⁶ Neurologisches Fachkrankenhaus für Bewegungsstörungen / Parkinson: Beelitz Heilstätten, Brandenburg, DE. Alemania

Submitted: 15-04-2026 | Revised: 20-05-2026 | Accepted: 01-06-2026 | Published: 02-06-2026

ABSTRACT

Introduction: Fascioliasis, a zoonosis caused by *Fasciola hepatica*, is a significant public health problem in the Bolivian highlands, especially among children. Although the disease is usually benign, in undiagnosed cases, it can progress to severe forms such as liver cirrhosis. **Case report:** We describe a 5-year-old patient from an endemic area who presented with abdominal distension, hepatomegaly, ascites, and edema. Laboratory tests revealed severe hypoalbuminemia, coagulopathy, eosinophilia, and a cholestatic pattern. Imaging confirmed chronic liver disease with portal hypertension. The patient's condition deteriorated, leading to neurological complications and death associated with sepsis and malnutrition. **Discussion:** This case demonstrates a consistent pathophysiological progression from chronic parasitic infection to liver fibrosis and cirrhosis. It highlights the diagnostic challenges in advanced stages and the reliance on clinical and epidemiological criteria in settings with limited access to confirmatory tests. It also reveals shortcomings in early diagnosis and timely access to treatment. **Conclusions:** Chronic fascioliasis caused by *Fasciola hepatica* can progress to liver cirrhosis in children, although this is rare, confirming its potential as a cause of severe chronic liver disease. In Bolivia, it is not a rare disease but rather an invisible one. Access to early diagnosis remains limited. Controlling this disease requires an intersectoral approach involving health, education, and basic sanitation. Fascioliasis should be considered a priority differential diagnosis for liver disease in childhood.

Keywords: Fascioliasis; *Fasciola hepatica*; hepatic cirrhosis; pediatric; portal hypertension; Bolivia; zoonosis.

RESUMEN

Introducción: La fascioliasis, zoonosis causada por *Fasciola hepatica*, constituye un problema de salud pública relevante en el altiplano boliviano, especialmente en población pediátrica. Aunque su evolución suele ser benigna, en casos no diagnosticados puede progresar a formas graves como cirrosis hepática. **Caso clínico:** Se describe una paciente de 5 años procedente de zona endémica, con distensión abdominal, hepatomegalia, ascitis y edema. Los estudios evidenciaron hipoalbuminemia severa, coagulopatía, eosinofilia y patrón colestásico. La imagenología confirmó hepatopatía crónica con hipertensión portal. La evolución fue desfavorable, con complicaciones neurológicas y fallecimiento asociada a sepsis y desnutrición. **Discusión:** El caso demuestra una progresión fisiopatológica coherente desde infección parasitaria crónica hasta fibrosis y cirrosis hepática. Resalta la dificultad diagnóstica en fases avanzadas y la dependencia de criterios clínico-epidemiológicos en contextos con acceso limitado a pruebas confirmatorias. También evidencia fallas en el diagnóstico temprano y en el acceso oportuno al tratamiento. **Conclusiones:** La fascioliasis crónica por *Fasciola hepatica* puede evolucionar hacia cirrosis hepática en la población pediátrica, aunque sea infrecuente, confirmando su potencial como causa de hepatopatía crónica grave. En Bolivia no es una enfermedad rara, es una enfermedad invisibilizada. El acceso al diagnóstico temprano sigue siendo limitado. El control de esta enfermedad requiere un enfoque intersectorial entre salud, educación y saneamiento básico. La fascioliasis debe considerarse diagnóstico diferencial prioritario de hepatopatía en la edad pediátrica.

Palabras clave: Fasciolosis; *Fasciola hepática*; cirrosis hepática; pediátrico; hipertensión portal; Bolivia; zoonosis.

INTRODUCTION

Fascioliasis is a parasitic zoonosis caused by the trematode *Fasciola hepatica*, which has a cosmopolitan distribution, and *Fasciola gigantica*, which is found primarily in tropical regions of Africa and Asia. These parasites commonly affect ruminants and, incidentally, humans. (1,2) In recent years, fascioliasis has been recognized as an emerging and re-emerging infection capable of progressing to chronic forms with progressive deterioration of liver function; this condition has a negative impact on both public health and livestock production. (3) It is estimated that over the past decade, the global prevalence has increased by approximately 11%, which is equivalent to nearly 17 million infected people and approximately 91 million at-risk people—figures that have prompted the World Health Organization to prioritize its control. (1,3) Infection is acquired through the accidental ingestion of viable metacercariae, the encysted forms of the parasite, present in contaminated water or raw vegetables. (2,3) The life cycle of *Fasciola* requires an intermediate host, represented by the freshwater snail of the genus *Lymnaea*, and a definitive host, which corresponds to ruminants such as sheep, goats, and cattle. Humans act as accidental hosts by consuming untreated water or raw vegetables such as watercress, lettuce, or alfalfa contaminated with metacercariae. From a clinical standpoint, fascioliasis manifests in two forms: acute and chronic. (1,2,4) The acute phase includes a typical phase characterized by fever, hepatomegaly, and abdominal pain secondary to larval migration through the hepatic parenchyma. At this stage, the juvenile forms of the parasite can cause hemorrhages in the intrahepatic bile ducts and contribute to the development of anemia. There are also atypical presentations, with respiratory, cardiac, or neurological symptoms, and ectopic forms, in which immature flukes are found in tissues other than the liver, with subcutaneous tissue being the most frequently affected site. (3,4)

The diagnosis of fascioliasis is based on the integration of clinical, laboratory, and imaging data. In the acute phase, serology using ELISA allows for the confirmation of the diagnosis with high sensitivity and specificity. (2,7,8,15) In the chronic phase, confirmation is achieved by identifying eggs in the stool or duodenal aspirate or by finding adult parasites during surgical procedures. The detection of fecal antigens or circulating antigens is a useful tool in both phases of the disease. (2,10) Although antibody detection does not allow differentiation between active and past infections, it has significant epidemiological value for assessing the distribution of the parasite in a population, especially when techniques such as Western blotting are used. (8,15) The evaluation of liver function tests also provides relevant information. In patients with nonspecific symptoms, measurements of glutamate dehydrogenase, aspartate aminotransferase, and Γ -glutamyl transpeptidase are useful, whereas decreased albumin, increased alkaline phosphatase, and elevated transaminases and bilirubin reflect cholestasis and parenchymal damage. (8,10,15)

Abdominal ultrasound is usually the initial test and may reveal thickening of the gallbladder wall, intravesicular echogenic images, and hypo- or hyperechogenic subcapsular lesions with tortuous courses or ductal involvement. (9,11) Computed tomography is particularly useful, especially in the acute phase, where hypodense nodular areas or tortuous, branched linear lesions—known as “branched tunnels”—are observed, with peripheral enhancement and a predominantly subcapsular location, corresponding to the parasite's migration route.

Despite these diagnostic advances, progression to complicated chronic forms, such as hepatic fibrosis or cirrhosis, is rare in the pediatric population and is often underdiagnosed, especially in endemic areas. In this context, the presentation of our clinical case is essential for expanding the knowledge about the natural history of the disease, its complications, and diagnostic challenges. This report describes a case of liver cirrhosis secondary to chronic fascioliasis in a pediatric patient, highlighting the clinical, laboratory, and imaging findings, as well as their relevance in the differential diagnosis of chronic liver diseases in children.

CLINICAL CASE

A 5-year-and-9-month-old female patient from Pucarani, an endemic area of the Andean highlands, was referred from the Hospital Holandés with a two-month history of progressive abdominal distension, intermittent episodes of mild abdominal pain localized in the right hypochondrium, and fluctuating edema of the lower extremities. accompanied by periods of unquantified low-grade fever and mild but sustained weight loss. On admission, the patient was in fair general condition, pale and well hydrated, with evident abdominal distension. Pulmonary auscultation revealed decreased vesicular breath sounds at both lung bases. The abdomen was distended, with palpable hepatomegaly approximately 3 cm below the costal margin that was firm in consistency and not tender on deep palpation. The abdominal circumference was 65 cm, with hypoactive bowel sounds and mild superficial collateral circulation. Edema of the lower extremities was noted at the time of the physical examination.

Laboratory tests revealed abnormalities consistent with advanced hepatocellular failure. The liver profile revealed severe hypoalbuminemia (1.8 g/dL), elevated transaminases with a mixed pattern (AST 69 IU/L, ALT 45 IU/L), and a marked increase in Γ -glutamyl transpeptidase (334 IU/L), indicating chronic cholestasis. Metabolic abnormalities, including hypocalcemia (7.8 mg/dL) and hypophosphatemia (2.6 mg/dL), as well as coagulopathy evidenced by a prolonged prothrombin time (16.1 s; INR 1.50), were also documented. C-reactive protein was moderately elevated (4.8 mg/dL), and the admission blood test showed significant eosinophilia (18%), suggesting a parasitic etiology.

Initial abdominal ultrasound revealed morphological findings consistent with chronic liver disease: a liver with regular contours but heterogeneous parenchyma, scattered hypoechoic areas with poorly defined margins, and a cephalocaudal diameter of 15 cm, suggesting hepatomegaly. Thickening of the gallbladder wall and abundant free fluid in all abdominal quadrants were also observed, with collapsed intestinal loops whose peristalsis could not be assessed because of the surrounding fluid. The Doppler study revealed a portal vein flow velocity of 11.2 cm/s, a peak systolic velocity in the hepatic artery of 64.8 cm/s, and a resistance index of 0.75, findings consistent with incipient portal hypertension. (Figure 2)

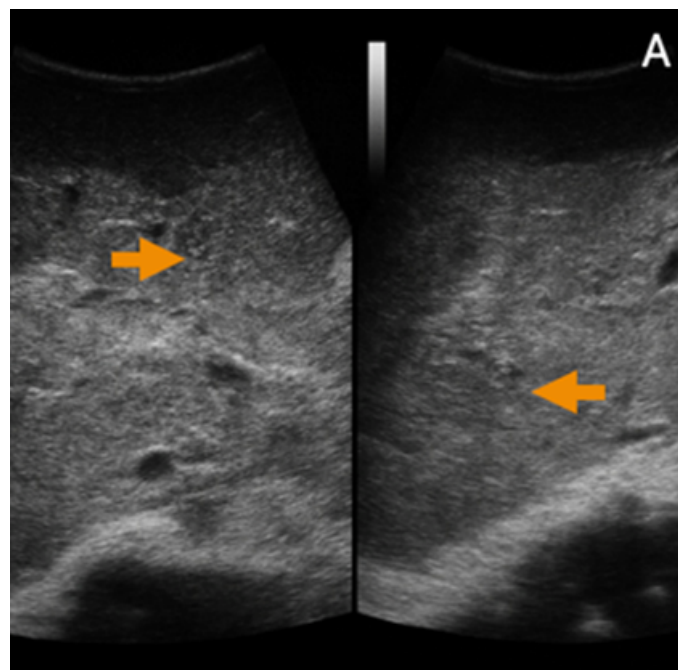


Figure 1. Abdominal ultrasound (B-mode, grayscale). Liver with regular contours, obtuse angles, parenchyma with heterogeneous echogenicity, and scattered hypoechoic areas with poorly defined margins (orange arrow). Cephalocaudal liver length of 15 cm, consistent with hepatomegaly.

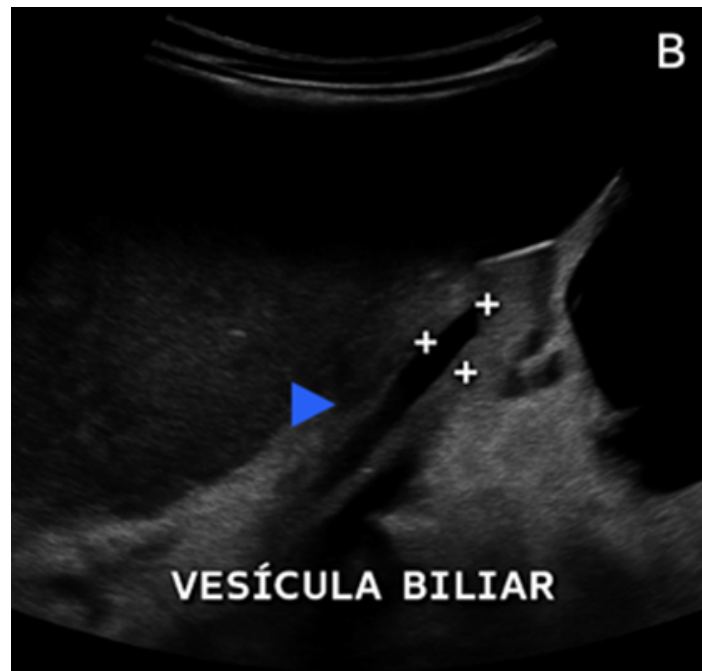


Figure 2. Abdominal ultrasound (B-mode, grayscale). Gallbladder measuring 5.3×3 cm with a parietal wall thickness of 0.7 cm (blue arrow), in the context of portal hypertension and chronic cholestasis secondary to fascioliasis.

A contrast-enhanced abdominal–pelvic computed tomography (CT) scan revealed an overall increase in liver size with a predominance of the left lobe, subtly lobulated margins, and heterogeneous parenchymal enhancement with nodular-appearing hypoechoic areas and multiple scattered hypodense nodules, findings assessed in the coronal and axial planes (Figures 3 and 4). Subcentimeter retroperitoneal lymph nodes and abundant free fluid in all quadrants of the peritoneal cavity were also observed, confirming the clinically evident massive ascites.

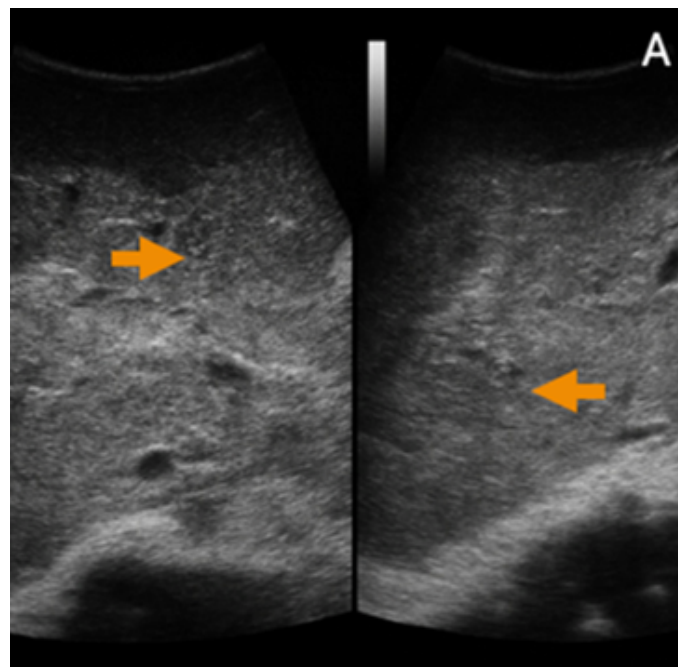


Figure 3. Contrast-enhanced abdominal–pelvic CT scan, coronal view. The liver is generally enlarged, with the left lobe being predominantly enlarged; the margins are subtly lobulated. Areas with a nodular appearance and heterogeneous enhancement of the parenchyma are identified following contrast administration.

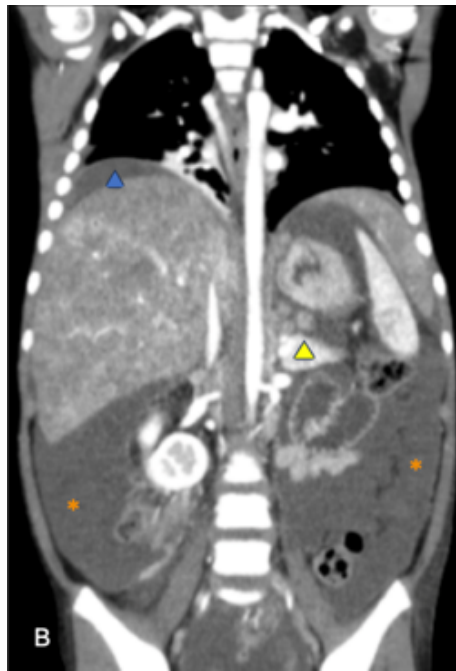


Figure 5. Abdominal CT scan showing density differences in the hepatic parenchyma. The yellow arrows indicate regions of hepatic involvement; the orange arrow points to an adjacent structure with different characteristics. Study performed with intravenous contrast in the portal phase.

Radiological evaluation via abdominal CT revealed characteristic findings with alterations in the density of the hepatic parenchyma (Figure 5). The axial image showed marked differences between hepatic regions, with areas of increased attenuation in the anterior segments and a more hypodense posterior zone (Figure 5). These radiological patterns were consistent with the established presumptive diagnosis. Abdominal imaging studies revealed patterns suggestive of simultaneous hepatic and pancreatic involvement. A thoracoabdominal CT scan of axial sections revealed alterations in liver density with parenchymal heterogeneity (Figure 5). At higher levels (mid-abdomen), pancreatic involvement was evident without evidence of necrosis or significant peripancreatic fluid (Figure 6). The combination of these findings suggested a differential diagnosis that included systemic etiologies.

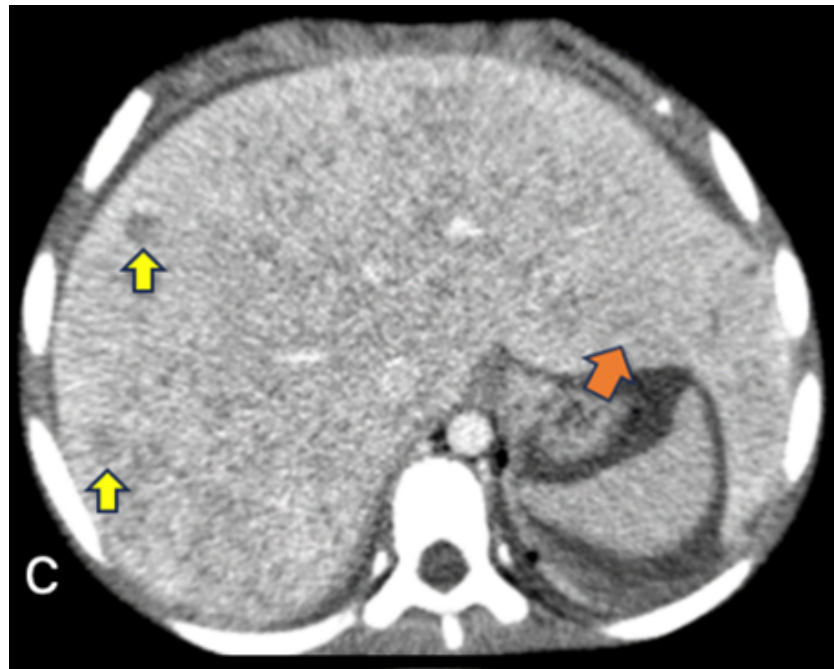
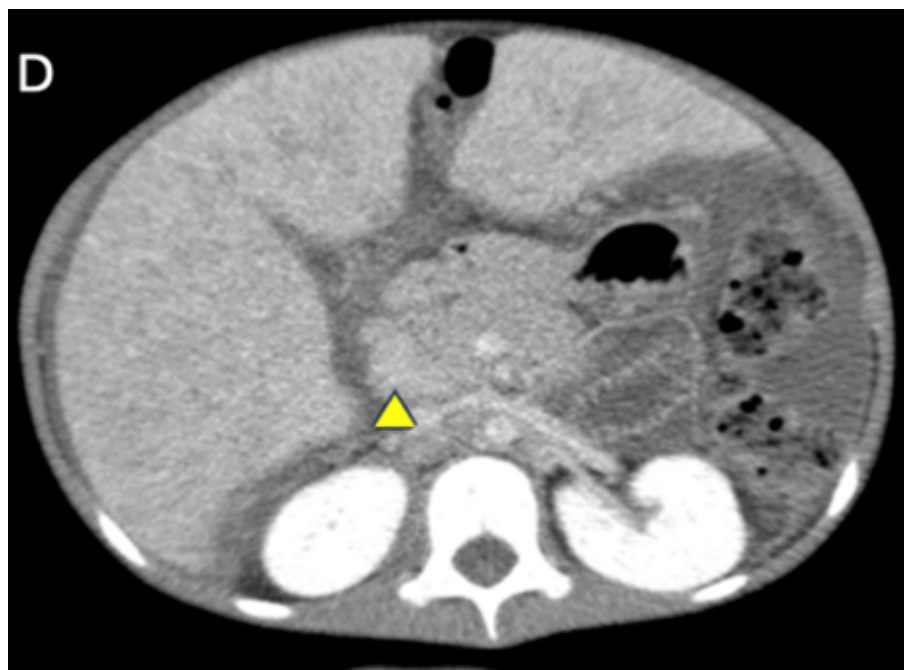


Figure 6. Abdominal CT scan at the pancreatic level. The yellow triangle indicates the pancreatic region with altered characteristics. Bilateral renal enhancement is observed in the portal phase, allowing for adequate evaluation of the hepatic parenchyma and adjacent mesenteric structures.



A complementary chest evaluation revealed associated pleuropulmonary involvement. Axial chest CT showed moderate right pleural effusion accompanied by thickening of the perialveolar pleura (red diamond). The distribution of these findings was consistently asymmetric, with relative preservation of the left lung parenchyma. Together with hepatic and pancreatic abnormalities, these findings suggest a multisystemic process with abdominal and thoracic manifestations. Given the progressive deterioration of her clinical condition, the patient was transferred to the Pediatric Intensive Care Unit, where she presented with cerebral intraparenchymal hemorrhage. Her condition deteriorated, and she died as a result of sepsis, with contributing factors, including severe protein-calorie malnutrition attributable to the underlying liver disease.

DISCUSSION

Fascioliasis, caused primarily by *Fasciola hepatica*, is a parasitic zoonosis of growing public health significance and is considered an emerging and re-emerging infection worldwide. Its impact is particularly significant in endemic regions such as the Bolivian Altiplano, where municipalities such as Pucarani have historically had high infection rates, with prevalences of up to 67% documented in groups of children aged 8 to 11 years, making it one of the hyperendemic areas with the highest disease burden in the world. (18)

This endemicity is closely related to local environmental and sociocultural factors, such as the presence of wetlands and the consumption of water or raw vegetables contaminated with metacercariae, which constitute the infective form of the parasite. In this cycle, humans act as accidental hosts, with ingestion of these items being the primary route of infection. The pediatric population, especially school-aged children, is particularly vulnerable, with a significantly increased risk of developing complications such as anemia and severe liver damage.

This case is a representative illustration of the progression to advanced disease. Chronic fascioliasis typically has an insidious onset, with nonspecific symptoms that can delay diagnosis. However, progression to chronic liver disease with signs of portal hypertension and hepatocellular failure, as observed in this case—marked abdominal distension, hepatomegaly, severe hypoalbuminemia, and coagulopathy—constitutes the most severe manifestation of persistent infection. (19)

The clinical, biochemical, and imaging findings in this patient are fully consistent with the pathophysiological process of advanced chronic fascioliasis. The marked increase in GGT, along with increased transaminases, reflects the mixed pattern of cholestasis and hepatocellular damage secondary to sustained periductal inflammation caused by adult parasites. Coagulopathy and severe hypoalbuminemia indicate a liver with a compromised functional reserve, whereas persistent eosinophilia, although attenuated compared with the acute phase, remains an indirect marker of chronic parasitic activity. (20) Taken together, these findings, along with the CT scan showing heterogeneous nodular parenchyma and massive ascites, are consistent with those of established liver cirrhosis and portal hypertension; the systematic review by Machicado et al. confirmed *F. hepatica*'s ability to induce progressive liver fibrosis and cirrhosis in humans, reinforcing the pathophysiological plausibility of the present case. (21)

Diagnosis in advanced stages can be complex. While the identification of eggs in fecal or bile samples remains the confirmatory method, clinical and epidemiological suspicion plays a fundamental role. In this context, imaging studies have significant diagnostic value. In the present case, abdominal ultrasound revealed hepatomegaly, parenchymal heterogeneity, thickening of the gallbladder wall, and abundant ascites, indicating a pattern suggestive of chronic liver disease with portal hypertension in an endemic setting.

The management of advanced cases such as this one requires a multidisciplinary approach that integrates specific antiparasitic treatments, intensive nutritional support, and the management of complications arising from chronic liver disease. Triclabendazole is the only drug recommended by the WHO for the treatment of human fascioliasis, and timely access to triclabendazole is crucial for halting fibrosis progression. (22) Studies conducted in the Bolivian highlands have demonstrated that mass administration of triclabendazole is feasible, safe, and effective in hyperendemic communities, with cure rates exceeding 97% after two treatment cycles, underscoring the viability of preventive chemotherapy strategies as a public health measure in these regions. (23)

CONCLUSIONS

Chronic fascioliasis caused by *Fasciola hepatica* can progress to liver cirrhosis in the pediatric population, although this is rare, confirming its potential as a cause of severe chronic liver disease. The absence of timely diagnosis and treatment makes it a potentially fatal but preventable disease. Imaging (ultrasound + CT scan) is highly useful as a diagnostic tool in advanced stages, especially in settings where parasitological confirmation is limited. Fascioliasis in Bolivia is not a rare disease; it is an invisible disease. This case demonstrates that the problem is not a lack of scientific knowledge but rather its application in the field. There is structural inequity in rural health; children in endemic areas are at higher risk for preventable diseases. Access to early diagnosis remains limited. Controlling this disease requires an intersectoral approach involving health, education, and basic sanitation. Fascioliasis should be considered a priority differential diagnosis for liver disease in children.

REFERENCES

1. Mas-Coma S, Bargues MD, Valero MA. Fascioliasis and other plant-borne trematode zoonoses. *Int J Parasitol.* 2020;50(4):263-280. Available at: <https://doi.org/10.1016/j.ijpara.2020.01.007>
2. World Health Organization. Fascioliasis: epidemiology, control and prevention. Geneva: WHO; 2021. Available at: <https://www.who.int/news-room/fact-sheets/detail/foodborne-trematode-infections>
3. Cordero del Campillo M, Rojo-Vázquez FA. Fasciolosis humana en América Latina. *Rev Panam Salud Publica.* 2020;44:e52. Available at: <https://doi.org/10.26633/RPSP.2020.52>
4. Marcos LA, Terashima A, Gotuzzo E. Update on human fascioliasis in children. *Curr Opin Infect Dis.* 2021;34(5):435-441. Available at: <https://doi.org/10.1097/QCO.0000000000000755>
5. Valero MA, Gironès N, García-Bodelón MA, Portus M, Mas-Coma S. High-risk groups in human fascioliasis. *Parasites & Vectors.* 2020;13:473. Available at: <https://doi.org/10.1186/s13071-020-04349-8>
6. Fürst T, Keiser J, Utzinger J. Global burden of food-borne trematodiasis. *PLoS Negl Trop Dis.* 2020;14(4):e0008149. Available at: <https://doi.org/10.1371/journal.pntd.0008149>
7. Callizaya Macedo G, Auza-Santivañez JC, Cabezas-Soliz IN, Alarcón Caba MFF, Murillo Alcázar MA, Atila Lujá ME, et al. Inflammatory myofibroblastic tumor in a pediatric patient. A case report from Bolivia. *SAP Nursing Depths Series [Internet].* 2025 Aug. 6 [cited 2026 Apr. 28];4:374. Available from: <https://nds.southam.pub/index.php/nds/article/view/374>
8. Ashrafi K, Bargues MD, O'Neill S, Mas-Coma S. Diagnosis of fascioliasis. *Clin Microbiol Rev.* 2021;34(3):e00021-19. Available at: <https://doi.org/10.1128/CMR.00021-19>
9. Lim JH. Radiologic findings of human fascioliasis. *AJR Am J Roentgenol.* 2020;214(3):W34-W45. Available at: <https://doi.org/10.2214/AJR.19.21716>
10. Evangelista-Ricci FJ, Miranda-Choque EA, Meza-Cloud GA, Sosaya-Zúñiga BS. Diagnóstico por imágenes de fasciolosis en niños. Instituto Nacional de Salud del Niño Breña 2011-2016. *Rev Per Radiol.* 2019;19:26-36. Available at: <https://doi.org/10.1016/j.rpradiol.2019.05.001>
11. Kabaalioglu A, Ceken K, Alimoglu E, Apaydin A. Imaging features of hepatobiliary fascioliasis. *Insights Imaging.* 2021;12:92. Available at: <https://doi.org/10.1186/s13244-021-01036-5>
12. Keiser J, Utzinger J. Triclabendazole for fascioliasis: a review and overview of pharmacology and clinical use. *Clin Microbiol Rev.* 2021;34(2):e00003-20. Available at: <https://doi.org/10.1128/CMR.00003-20>
13. World Health Organization. Triclabendazole in human fascioliasis. Geneva: WHO; 2022. Available at: <https://www.who.int/docs/default-source/neglected-tropical-diseases/fascioliasis/triclabendazole.pdf>
14. Kubatz La Madrid ME, Collazo Cruz J, Cruz Cruz Y, Auza-Santivañez JC, Bautista-Vanegas FE, Aguirre-Cruz B, et al. Asymptomatic multiorgan cysticercosis. A case report from Bolivia. *SAP Health and Policy [Internet].* 2025 Aug. 5 [cited 2026 Apr. 28];4:324. Available from: <https://shp.southam.pub/index.php/shp/article/view/324>
15. Marcos LA, Maco V, Terashima A, Samalvides F, Gotuzzo E. Características clínicas de la infección crónica por *Fasciola hepatica* en niños. *Rev Gastroenterol Peru.* 2002;22(3):228-233. Available at: http://www.scielo.org.pe/scielo.php?script=sci_arttext&pid=S1022-51292002000300006
16. Jara-Campos CA, Escalante-Añorga HM, Davelois KR, Benites AR, Casana-Mantilla WM. Infección por *Fasciola hepatica* en escolares de la provincia de Pataz (La Libertad, Perú): prevalencia y perfil hepático. *Rev Méd Trujillo.* 2019;14(2):66-78. Available at: <http://dx.doi.org/10.17268/rmt.2019.v14i02.01>
17. Jara-Campos CA, Escalante-Añorga HM, Davelois KR, Benites AR, Casana-Mantilla WM. Infección por *Fasciola hepatica* en escolares de la provincia de Pataz (La Libertad, Perú): prevalencia y perfil hepático. *Rev Méd Trujillo.* 2019;14(2):66-78. Available at: <http://dx.doi.org/10.17268/rmt.2019.v14i02.01>
18. Parkinson M, O'Neill SM, Dalton JP. Endemic human fasciolosis in the Bolivian Altiplano. *Epidemiol Infect.* 2007;135(4):669-674. Available at: <https://doi.org/10.1017/S0950268806006935>
19. Cabada MM, Malaga JL, Castellanos-Gonzalez A, White AC Jr. A comparison of the risk for chronic fascioliasis between children 3 to 5 years and children 6 to 12 years of age in the Cusco region of Peru. *Am J Trop Med Hyg.* 2021;105(5):1432-1437. Available at: <https://doi.org/10.4269/ajtmh.21-0399>
20. Ismail A, Abdelsalam MA, Shahin MH, Ahmed Y, Bahcecioglu IH, Yalniz M, Tawheed A. Hepatobiliary fascioliasis: a neglected re-emerging threat, its diagnostic and management challenges. *World J Gastrointest Pathophysiol.* 2025;16(2):107599. Available at: <https://pmc.ncbi.nlm.nih.gov/articles/PMC12186194/>
21. Machicado C, Machicado JD, Maco V, Terashima A, Marcos LA. Association of *Fasciola hepatica* infection with liver fibrosis, cirrhosis, and cancer: a systematic review. *PLoS Negl Trop Dis.* 2016;10(9):e0004962. Available at: <https://doi.org/10.1371/journal.pntd.0004962>
22. Cabada MM, Goodrich MR, Graham B, Villanueva-Meyer PG, Deichsel EL, Callacondo D, et al. Human fascioliasis: current epidemiological status and strategies for diagnosis, treatment, and control. *Res Rep Trop Med.* 2020;11:149-158. Available at: <https://doi.org/10.2147/RRTM.S237461>
23. Villegas F, Angles R, Barrientos R, Barrios G, Valero MA, Hamed K, et al. Administration of triclabendazole is safe and effective in controlling fascioliasis in an endemic community of the Bolivian Altiplano. *PLoS Negl Trop Dis.* 2012;6(8):e1720. Available at: <https://doi.org/10.1371/journal.pntd.0001720>

FUNDING

The authors received no funding for this study.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest.

AUTHORSHIP CONTRIBUTIONS

Conceptualization: Giovanni Callizaya-Macedo, Jhossmar Cristians Auza-Santivañez

Data curation: José Miguel Cussi Romero

Formal analysis: Giovanni Callizaya-Macedo

Research: Giovanni Callizaya-Macedo, Jhossmar Cristians Auza-Santivañez

Methodology: José Miguel Cussi Romero, Jhossmar Cristians Auza-Santivañez

Project management: Jhossmar Cristians Auza-Santivañez

Resources: José Miguel Cussi Romero

Software: José Antonio Lagos Monasterios

Supervision: Giovanni Callizaya-Macedo

Validation: José Miguel Cussi Romero, María Angela Murillo Alcázar

Visualization: José Antonio Lagos Monasterios

Drafting – original draft: Giovanni Callizaya-Macedo, José Miguel Cussi Romero, Jhossmar Cristians Auza-Santivañez, José Antonio Lagos Monasterios, Angel Simon Chuquimia Calderon, María Angela Murillo Alcázar, Daniela Nicole Pacheco de la Torre, Freddy Ednildon Bautista-Vanegas.

Writing – review and editing: Giovanni Callizaya-Macedo, José Miguel Cussi Romero, Jhossmar Cristians Auza-Santivañez, José Antonio Lagos Monasterios, Angel Simon Chuquimia Calderon, María Angela Murillo Alcázar, Daniela Nicole Pacheco de la Torre, Freddy Ednildon Bautista-Vanegas.