



FIGURE 1. A: Liver tissue showing granuloma formation around *Calodium hepaticum* eggs. The granulomata were nodules with sizes between 1 mm to 3.5 × 2.0 cm containing eggs and worms or necrotic materials and nucleic debris surrounded by a rim of diverse inflammatory cells. Liver tissue outside the granulomata appears healthy. B: Transverse section of re-curved necrotic adult female; midbody (left; 115 µm at widest point) and anterior (right; 45 × 27 µm). The BB and thick Cu are visible, as are a necrotic Ov and St. C: Two eggs in longitudinal (left; 41 µm long) and transverse (right; 36 × 27 µm) section. The external envelop contains numerous minipores and is thinner than the internal envelop with SS between them. A PP may be seen in 1 egg. Hematoxylin and eosin stain. BB indicates bacillary bands, Cu, cuticle, Ov, ovary, PP, polar plug, SS, sagittal striations, St, stichocyte.

A Case of Autochthonous Hepatic Capillariasis in a Refugee Child in Belgium

To the Editors:

Hepatic capillariasis is a neglected, but serious, hepatic disease with a worldwide distribution caused by the nematode *Calodium hepaticum*.¹⁻⁴

The nematode has a monoxenous life cycle and can infect any mammalian liver but mainly affects rodents, while humans are an accidental host.^{1,3,4} Infection occurs after ingestion of embryonated eggs from contaminated soil. The larvae hatch in the intestine, migrate, mature, and mate in the liver parenchyma, with hundreds unembryonated eggs laid.^{1,5} The eggs remain viable in the liver and are released into the environment after death of the host.^{3,4} In the soil, the eggs reach the infective stage.^{3,5} Poor sanitation and living conditions predispose to infection.^{1,5} There is a higher incidence in children attributable to higher soil-to-hand-to-mouth contact. Pica is an additional risk, particularly when presenting as geophagia.¹

A 3-year-old Afghan girl born in Belgium and living in an asylum seeker center was referred for persistent high fever and abdominal pain. A history of pica was present. Pallor, weakness, and hepatomegaly were observed on clinical examination. Blood tests revealed elevated C-reactive protein (151 mg/L), ALT (360 IU/L), AST (207 IU/L), IgM levels (13.05 g/L), microcytic anemia (8.3 g/dL), and eosinophilia (12,750/µL). Stool cultures were negative. Abdominal ultrasound examination showed hepatosplenomegaly with ascites. FibroScan revealed minor fibrosis with a stage F1. Serology for *Fasciola hepatica* and *Toxocara canis* was positive. Liver biopsy revealed adult nematodes and eggs. The child was treated with triclabendazole (10 mg/kg/day) for 2 days then albendazole (50 mg/kg/day) and methylprednisolone (1 mg/kg/day) for 5 days. She responded well. Twenty days after treatment, she presented with a relapse. A review of the liver biopsy identified the diagnostic features of *C. hepaticum* (Fig. 1). This diagnosis was confirmed by PCR on the liver tissue. The positive *Fasciola* and *Toxocara* serologic tests were determined to be cross-reactions. Albendazole course was continued for 100 days (25 mg/kg/day) and corticosteroids (1 mg/kg/day) for 50 days followed by degressive weaning with positive clinical and biological response.

The clinical presentation of hepatic capillariasis is nonspecific. Prolonged fever, anemia, hepatomegaly, eosinophilia, hyperglobulinaemia and elevated levels of liver enzymes are usually present.^{1,2,5} Clinical, biochemical and radiological correlation may assist in diagnosis but definitive diagnosis requires liver biopsy.^{1,2,5} The parasite

and its eggs are undetectable in feces.^{1,2} Serological detection of antibodies to *C. hepaticum* may be useful but is limited by antigen availability¹ and the absence of a commercially available test.² Antibodies to *C. hepaticum* can cross-react in serological tests for *Toxocara*,⁵ *Schistosoma*, and *Dirofilaria immitis*.^{1,5}

Clinical prognosis is defined by the intensity of infestation and ranges from mild to severe or fatal.^{2,5} Survival rates increases with early diagnosis.¹

Treatment consists of a combination of corticosteroids and anthelmintics.^{1,2,5}

Hepatic capillariasis is rare, but should be taken into account in a child presenting with persistent fever, hepatomegaly and eosinophilia. Currently, biopsy remains the cornerstone of diagnosis. The development of serological tests would be beneficial to early diagnosis and treatment, which improves prognosis.

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Central Diabetes Insipidus: Another Rare Complication of SARS-CoV-2 Infection in Children?

To the Editors:

The clinical course of SARS-CoV-2 infection is extremely variable, ranging from asymptomatic infection in over 40% of cases to acute respiratory syndrome causing distress in about 3% of cases.¹ In the early phase, it became soon

clear that it remained far less aggressive with children,² with inferior mortality and morbidity.³ The impact of SARS-CoV-2 infection in children has changed since January 2020. Development of tools for systematic monitoring of infection, and tracing of individual cases, provided additional information and, in some cases, disclosed the association of SARS-CoV-2 infection with various clinical manifestations. How many of them are proper-hoc or rather simply post hoc, that is one mistake temporal sequence for causal connection, often remains to be assessed. To this issue, reporting the clinical observation of unusual or unexpected events, associated with SARS-CoV-2 infection, may bear some interest under the clinical, but also the research point of view.

We report a previously healthy 17-year-old male who presented to our Pediatric Emergency Department with acute onset, isolated polydipsia and polyuria, in the range of 7L/day. He had no family history of diabetes insipidus (DI), and his medical history was fully uneventful, with the only exception of pauci-symptomatic SARS-CoV-2 infection, diagnosed 3 weeks before.

Initial laboratory examination revealed hypernatremia (147 mEq/L) and increased serum osmolality (307 mOsm/L). His 24-h urinary output was approximately 7.5 liters, with low-specific gravity (1.002 g/mL) and osmolality (100 mOsm/L). Based on those findings, DI was diagnosed.

He tested negative at PCR analysis of his nasopharyngeal swab for SARS-CoV-2.

To discriminate between central diabetes insipidus (CDI) and nephrogenic diabetes insipidus, a desmopressin (DDAVP) stimulation test was performed through intranasal administration of 10 µg of desmopressin: polyuria and polydipsia were immediately corrected (24-h urine output, 2.4 liters), as well as serum sodium (141 mEq/L) and serum osmolality (293 mOsm/l). Urine-specific gravity (1.010 g/ml) and osmolality (207 mOsm/L) defects improved, too; pituitary hormones were normal.

Contrast-enhanced brain magnetic resonance imaging showed the expected disappearance of the normal posterior pituitary bright signal, no evidence of hypothalamic-pituitary mass, or thick pituitary stalk. Chest radiograph excluded lung interstitial, nodular or cystic lesions. radiograph evaluation of the entire skeleton ruled out any osteolytic lesion, either recent or previous. Based on all the above information, the diagnosis of CDI was made and intranasal desmopressin spray, 10 µg twice a day, was started. He rapidly achieved good disease control and could be discharged on day 7 of hospitalization.

Only 5 cases of COVID-associated DI have been reported,^{3–7} none in a patient during pediatric age. They had an age ranging between 28 and 68 years, 2 had diabetes-type2. The diagnosis of SARS-CoV-2 infection preceded acute onset-DI by 2–8 weeks. Clinical manifestation ranged from pauci-symptomatic in 2 cases, to respiratory syndrome in 3 cases; only 1 patient had a fatal outcome of COVID-19 pneumonia. The present case perfectly fits in this frame, with a 3-week delay between SARS-CoV-2 infection (with pauci-symptomatic course) and DI onset. This is apparently the first case of COVID-19-associated DI occurring during the pediatric age.

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