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Acute Parasitic Cholitis Revealing Gilbert's Disease: About a Malagasy Case

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Summary

Parasitic migration into the bile ducts is less common. Few cases of parasitic cholitis were reported despite Madagascar being a parasitic endemic country. Our goal is to report the case and stimulate the search for Gilbert's disease in free bilirubin cholitis. A 24-year-old Malagasy woman is hospitalized for febrile jaundice. She is not obese and had no history of vesicular lithiasis. The interrogation reveals the absence of deworming for several years. The clinical examination showed pain on palpation of the right hypochondrium. An abdominal ultrasound showed an intravesicular rail image and extrahepatic bile duct dilation. Biology revealed an elevation of alkaline phosphatase and gamma GT associated with an elevation of predominantly free total bilirubin. There was no anemia or eosinophilia. Blood culture series had come back negative as well as viral serologies (hepatitis A, B, C and HIV). The hemolysis balance was negative. Microscopic examination of the stool for KAOP showed the presence of numerous roundworm eggs. The diagnosis of parasitic cholitis is made associated with Gilbert's disease. The clinico-biolog-

ical evolution is favorable after deworming associated with antibiotic therapy. A control abdominal CT scan was normal after a one-month setback.

Keywords: jaundice; bilirubin; abdominal ultrasound; Madagascar; parasitosis

Introduction

Madagascar is a parasitic endemic country. Gilbert's disease is a benign form of hereditary free bilirubin jaundice transmitted in an autosomal recessive manner [1]. Few cases of acute cholitis on ascariasis have been reported in the country. The association of Gilbert's disease and acute parasitic cholitis is rare. The objective is to report the first Malagasy case of an association of acute acarid cholitis with Gilbert's disease.

Patient and Observation

A 24-year-old Malagasy woman is hospitalized for jaundice progressing two days before her admission associated with a fever at 40°C, without chills and abdominal pain localized at the level of the right hypochondrium without sign of acute surgical abdomen. The interrogation revealed the deworming of the past five years.

Nor travel to endemic malarial areas of Madagascar. She is not obese, nor does she have a history of vesicular lithiasis. Physical examination revealed a stable haemodynamic state, mucocutaneous jaundice and defence in the hypochondrium. The abdominal volume was normal and there was no collateral venous circulation or palpable mass. The digital rectal exam was normal. The stool is not discoloured. The rest of the clinical examination was unremarkable. In biology, there was an inflammatory syndrome (with neutrophil hyperleukocytosis and CRP at 26 mg / L). The haemoglobin level was 14 g/dl and there was no eosinophilia. Hepatic status showed predominantly free hyperbilirubinemia (total bilirubin elevated to 241 µmol/L, free bilirubin to 211 µmol/L), biological cholestasis with Gamma glutamyl transferase elevated 4 times to normal (312 IU/L), elevated alkaline phosphatase 6 times normal (406 U/L) and hepatic cytolysis twice to normal. Lipasemia was increased to 1.5 times normal. Serum protein electrophoresis was normal. Viral liver serologies A, B, C and HIV serology were negative. The thick gout and thin smear looking for malaria were negative. Bacteriological samples and blood cultures were negative. Parasitological examination of the stool found several roundworm eggs. A hemolysis test was requested whose results showed a normal haptoglobin and LDH level, the Emmel, Itano, Coombs tests were negative. The renal assessment was also normal. Abdominal ultrasound showed a thin-walled, alithiasic gallbladder with intravesicular rail image and extrahepatic bile duct dilation (Figure 1). The diagnosis of acute cholitis on ascariasis associated with Gilbert's disease was retained.

Deworming with albendazole 400 mg/d for 3 days combined with dual antibiotic therapy (ceftriaxone 1g/d and metronidazole 1.5g/d) for 10 days was prescribed for this patient.

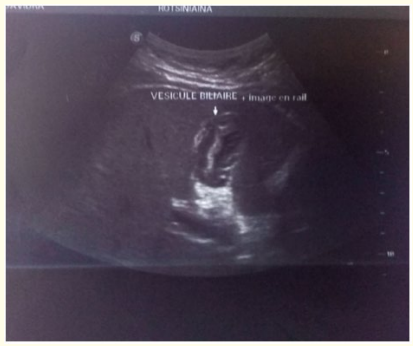

Figure 1: Rail image in gallbladder on ultrasound.

The course of the disease was favourably marked by apyrexia from the third day of treatment, the gradual disappearance of mucocutaneous jaundice and abdominal pain. An abdominal CT scan was requested a month later and showed the disappearance of the intravesicular parasite image and the extrahepatic bile duct dilation. The liver function normalized after a one-month setback of treatment.

Discussion

Although Madagascar is a parasitic endemic country, intravesicular localization by ascariasis is little reported in the country. Cholitis is a condition characterized by febrile cholestatic jaundice. More than 10% of the population in low-income countries are affected by ascariasis. Regarding this observation, this patient had febrile jaundice with free bilirubin without signs of hemolysis and biological cholestasis. Abdominal ultrasound revealed the presence of an image in favor of parasitosis in the gallbladder. KAOP stool examination makes it possible to retain the diagnosis of acute cholitis on ascariasis associated with Gilbert's disease. Endoscopic retrograde cholangiopancreatography (ERCP), liver CT and bili-MRI remain the first-line examination. It makes it possible to visualize characteristic images including a "rail" image, curved fringe image, "spaghetti-like" image on a cross-section, sometimes associated with undulating movements called "dance of verses" [2]. It can also detect other complications such as liver abscess [3]. Abdominal ultrasound is a non-invasive, low-cost and highly accessible examination in low-income countries such as Madagascar. However, this examination is both operator and dependent device.

Gilbert's disease is characterized by free bilirubinemia jaundice in the absence of any hemolysis [4, 5, 7] related to a partial congenital deficiency of bilirubin glucuronyl transferase (UGT1A1) which is a hepatic enzyme ensuring the conjugation of bilirubin. The activity of this enzyme is reduced to 70% of normal during this disease, responsible for an increase in indirect bilirubin and which is manifested by intermittent mild jaundice. The most common genotype of this pathology is represented by the homozygous polymorphism A(TA) 7TAA in the promoter of the gene encoding UDP glucuronosyltransferase 1A [6, 7].

Clinically, it results in a fluctuating subictera visible mainly at the level of the conjunctiva. Frankish jaundice may be accompanied by some nausea and mild abdominal pain can be found following prolonged fastingor infection. The rest of the clinical examination is normal [4, 5]. Biologically, there is amore marked elevation of predominantly free total bilirubinemia without stigma of hemolysis. Gilbert's disease in itself does not require any treatment. It is an autosomal recessive genetic disease with no clinical consequences and no long-term complications. The diagnosis of Gilbert's disease is based on the association of free bilirubin jaundice in the absence of hemolysis and confirmed by the identification of the genetic mutation. For this patient, liver biopsy is not performed because it is an invasive examination and the evolution of jaundice is favourable after anti treatment by atestary. The genetic identification of the mutation was not carried out due to the lack of a local technical platform.

Conclusion

Gilbert's disease is a rare genetic disease of benign evolution. But in a highly endemic area like Madagascar, intravesicular acaridial parasitic superinfection could be responsible for a serious infectious complication that must be systematically investigated.

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