Dear Editor,

A 40-year-old previously healthy woman experienced a four-week history of recurrent postprandial epigastric pain. Following medical investigation, the patient was diagnosed with an ultrasound-documented cholelithiasis, as well as syphilis, since the specific blood tests (venereal disease laboratory research and *Treponema pallidum* hemagglutination assays) were positive. After a three-week course of intramuscular benzathine penicillin, the patient, free from peritoneal signs, was scheduled for laparoscopic cholecystectomy at our surgical department. During the operation, there was no evidence of the gallbladder on the inferior side of the liver. Therefore, after inspecting the liver and its ligaments, gallbladder heterotopy was excluded and the intraoperative diagnosis of agenesis of the gallbladder was made. The day after the intervention, a magnetic resonance cholangiopancreatography confirmed the gallbladder agenesis and excluded further biliary abnormalities or cholelithiasis. Hence, the patient was discharged. After two years and four months of follow-up, she remains asymptomatic.

Through the centuries, syphilis has proven to affect any organ of the human body, including the hepatobiliary-pancreatic system. In fact, late and congenital syphilis may determine hepatitis and obstructive jaundice through the development of hepatic or cephalopancreatic gumma and hepar lobatum, which is the syphilitic cirrhosis of the liver, while early syphilis may cause perportal inflammation.1,2 Conversely, congenital agenesis of the gallbladder (CAGB) is a very rare condition (0.01%-0.04%) that is one of the numerous disorders affecting the gallbladder. Usually asymptomatic, it sometimes manifests with dyspepsia, biliary colic, or jaundice.3 It is known that ultrasound can misdiagnose such an anomaly, thus rendering the diagnosis of CAGB without a laparoscopic intervention somewhat difficult.4 The pertinent literature consists of approximately 400 cases of CAGB; none of these were associated with syphilis. Although there is no certain explanation as to why and how a previously dormant CAGB became symptomatic after a *Treponema pallidum* infection, our patient’s long asymptomatic stage (28 months with no biliary colic) following specific antibiotic treatment is a relevant fact, and prompts the speculation on a potentially underlying immunologic mechanism. Given the unprecedented concurrence of these two conditions, this clinical dilemma could remain indefinitely unresolved.

**Conflict of interest**

All authors declare to have no conflict of interest.

**References**


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