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Gallbladder Agenesia

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Summary

Gallbladder agenesis (GA) is a rare congenital entity. Most patients remain asymptomatic, while those with symptoms report symptoms that mimic bile colic. Initial evaluation for suspected gallbladder pathology, such as ultrasound of the right upper quadrant, May be misleading or inconclusive. As a result, some patients are eventually diagnosed intra-operatively. Therefore, GA should be maintained as a differential diagnosis and should be performed as magnetic resonance cholangiopancreatography (MRI) when other tests are inconclusive.

We present a 39-year-old woman who has chronic symptoms compatible with biliary colic and an equivocal ultrasound reported as scleroatrofic with cholelithiasis. Laparoscopy was performed during which the absence of gallbladder was found. Postoperative CPRM confirmed the diagnosis of GA.

Keywords: Gallbladder Agenesis; Magnetic Resonance Cholangial-Pancreatography (MRCP); Laparoscopy

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Introduction

Gallbladder agenesis (BVS) is a rare condition. It is defined as the congenital absence of the gallbladder (BV), associated or not with the absence of the cystic duct [1]. It is a rare congenital condition of the biliary system, where approximately 400 cases of BVS, both in autopsies and as clinical presentations, have been described in the literature [2].

On the other hand, more than 50% of the cases are isolated and asymptomatic. These asymptomatic patients are mainly healthy and do not require interventions. However, some patients develop symptoms, presenting clinical signs similar to the symptoms of BV lithiasis. Symptoms commonly occur in the fourth or fifth decade of the patient's life [3].

Having treated a patient with BV has motivated us to present within this society.

Material and Method

Clinical case: 39-year-old female patient, referred from the Nephrology Service, with a history of chronic kidney failure, without hemodialysis but likely to have a kidney transplant. She states that the patient began with epigastric pain irradiating to HD for 1 year, accompanied by abdominal distension, nausea and vomiting. On the other hand, on some occasions she manifested heartburn and epigastric burning. Consultation with the Gastroenterology Service where a VEDA was carried out that showed normal esophagus, an antral region where multiple biopsies were performed and that showed antral gastritis according to the pathological result. Helicobacter pylori negative He medicated with pump inhibitors, diet. In turn, an abdominal ultrasound showed a diminished size and scleroatrophic BV. She improved her symptoms and later at 6 months, she started with abdominal pain. New abdominal ultrasound that reveals a decreased size and scleroatrophic gallbladder. Diet was indicated, where it evolved favorably. 1 month ago, new intense pain in the same HD region, accompanied by vomiting and nausea after ingestion. It was decided to consult with the Surgery Service, where a new abdominal ultrasound was carried out, which showed a scleroatrophic gallbladder with lithiasis. New VEDA, which was normal. Given this situation and the repeated abdominal pain crises, a laparoscopic cholecystectomy is proposed. In the laparoscopic exploration, after the placement of the trocars, the BV is not found in the region of the gallbladder liver bed (Figure 1), it is explored on the left liver side where the BV is not found either. It was decided to explore the region of the main bile duct where a

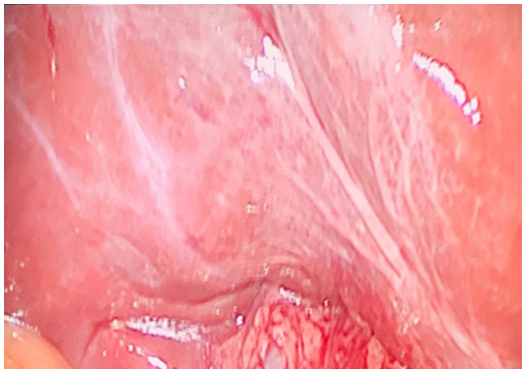

Figure 1: Laparoscopic examination. The gallbladder is not seen.

scleroatrophic organ could be found over said region by placing a clamp (Figure 2). In addition, the Rouviers groove is well observed, where BVP free of the vesicular organ is observed, allowing exploration towards the confluent of the liver where the BV does not appear (Figure 3). Finally, it was decided to release the VBP, to see if there was a gallbladder outline, being negative (Figure 4). Finally, it is decided to end the laparoscopic exploration. Postoperative good evolution. 12-hour hospitalization. A post-operative MRCP was requested, the result of which was normal, not showing the gallbladder. It is referred to the Nephrology Service.

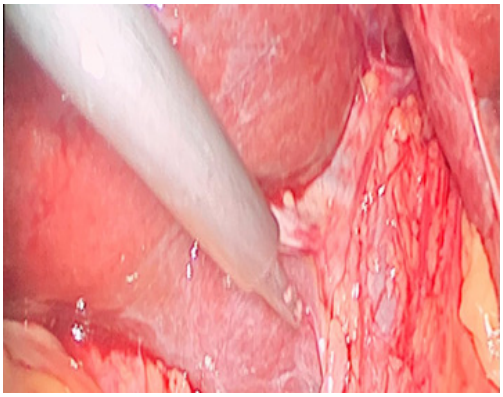

Figure 2: It is separated with forceps over the bile duct. But no gallbladder is found.

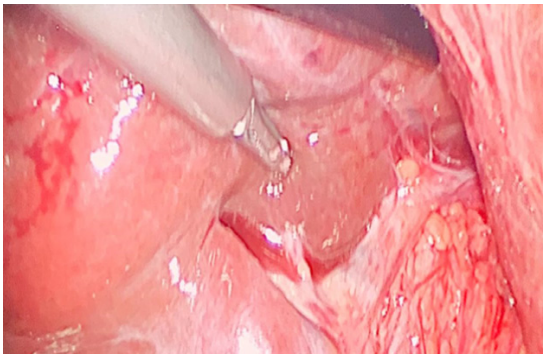

Figure 3: Groove of Rouviers is observed. VBP is observed and confluent without VB.

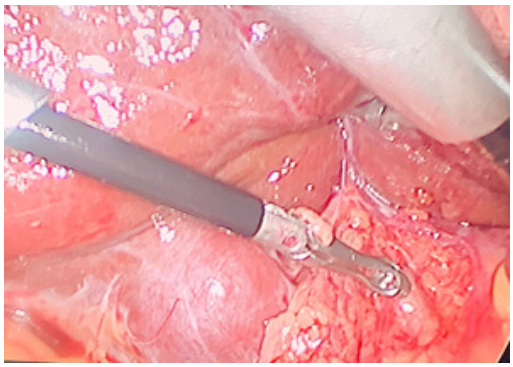

Figure 4: Fatty tissue is removed from VBP. Not finding the VB.

Discussion

The biliary system presents relatively frequent anomalies. BV is subject to these abnormalities in number, location, and morphology. BVS without extra-hepatic biliary atresia is a well-recognized but extremely rare congenital anomaly in which BV does not develop and is subsequently not found in the usual or more common atypical sites [4,5]. The first case was described by Lemery in 1701 [6].

From an epidemiological point of view, the incidence based on autopsy results is approximately one case per 6334 live births [5], or approximately 0.035 to 0.3% [7,8]. Allan S, et al. (1974) [9], observed three cases of VGA in 2451 cholecystectomies. In contrast, Jain BK, et al. (2001) [10], reported four cases among 771 patients who were operated on for bile duct diseases over a 5-year period.

From an embryology and etiology point of view, its congenital malformation is accepted, which may or may not be accompanied by agenesis of the cystic duct [11]. It is accepted that embryogenesis is corroborated by the frequent association of agenesis with congenital malformations [1]. Other forms are the existence of families in which this pathology has occurred in several members, which suggests that there are family hereditary forms of AGV [12]. Likewise, this abnormality may be associated with other biliary and / or extra-biliary abnormalities, such as unusual complications caused by common bile duct cysts [13,14]. Children with VGA and distant malformations tend to have trisomy 13 or another chromosomal abnormality that carries a poor prognosis [8]. In all this context, some authors [6,15], established a classification that can distinguish three groups:

1. Fetal abnormalities (12.9%): Patients who die in the prenatal period of other malformations, such as cardiovascular, of genito-urinary systems and gastrointestinal tract, but VGA is seen at autopsy.

2. Asymptomatic group (31.6%): These patients do not present biliary symptoms and the AGV is discovered during an autopsy or in surgery for another pathology.

3. Symptomatic group (55.6%): These patients between 40 and 50 years old, who do not have another congenital anomaly.

Regarding the symptoms of these symptomatic patients, abdominal pain is in most cases (90%), nausea and vomiting (66%), intolerance to CCK (37%), dyspepsia (30%) [6,16]. The symptoms that our patient presented, with abdominal pain, nausea, dyspepsia, similar to that described in the literature. Jaundice is very rare and can be associated with Choledocholithiasis with or without cholangitis [10,17]. Likewise, the examination may show pain on palpation of the HD. Richards RJ, et al. (1993) [18], reviewed 44 cases of VGA and found that dyspepsia was the predominant symptom in 15 of 44 patients (34%); 24 of 44 (54%) had symptoms suggestive of biliary colic and 12 of 44 (27%) had jaundice. Common duct stones were found in 8 of 12 patients with jaundice, but not in any other.

Symptoms may be secondary to concomitant pathologies, such as stones in the primary ducts and biliary dyskinesia, or may be related to non-biliary causes such as esophagitis and duodenitis [6,16,19 and 20]. In AGV, the hepatic bile duct can replace the absent gallbladder, dilating and assuming the bile storage function [5]. The presence of dyskinesia of the biliary tract, increased resting pressure of the sphincter of Oddi, cholestasis or infection of the bile ducts can initiate the appearance of biliary symptoms and / or the formation of stones in the common bile duct, especially when two or more of These events occur in association [5,8, and 21]. In our case, the patient had two VEDA, where it was demonstrated in one antral gastritis and in another that it was normal.

Regarding diagnosis, abdominal ultrasound is the initial examination for the exploration of biliary pathology. Its sensitivity is 95% in the diagnosis of gallstones [22]. The preoperative diagnosis of VGA has been considered extremely difficult and almost all diagnoses in symptomatic patients are made at laparotomy or during attempted

laparoscopic cholecystectomy [17]. Although ideally, the diagnosis should be made before surgery, this has been documented in only a few reports [23,24]. This is mainly since radiological investigation methods for biliary pathology diseases have a sensitivity of <100% for the identification of BV, and ultrasound is highly operator dependent [8,16]. The confirmed cases of VGA, the report of the abdominal ultrasound is of a lithiasic scleroatrophic BV, the false positives can be explained by the interposition of gases from bowel loops in the gallbladder bed [25], of a peri-portal peritoneal fold [26]. In our case, the patient had two ultrasound scans that showed a decrease in the size of the BV, scleroatrophic and finally a third that showed to be scleroatrophic with gallstones.

Non-visualization of BV by ultrasound can be confirmed by preoperative hepato-biliary scintigraphy, abdominal CT scan, ERCP or MRCP [16,23, and 27]. A preoperative diagnosis based on computed tomography, HIDA scan, and cautious ultrasound would have been reported in twins [28]. Abdominal CT in nine patients with VGA can accurately determine the condition before the operation [29]. These authors suggested that an abdominal CT should be performed in any case in which VB is not visualized on ultrasound. Orlando R, et al. (2001) [23], suggested that diagnostic confirmation is achieved by MRCP, thus avoiding laparotomy, whereas conventional hepato-biliary imaging studies and laparoscopy could not achieve a definitive diagnosis.

Finally, we believe that it is difficult to suspect this congenital malformation and even with studies such as ultrasound where it is operator dependent. But we must be suspicious when faced with a diagnosis of BV that is scleroatrophic and / or not visualized by this method. Faced with this eventuality, the abdominal CT and even more so the MRCP will put us to consider the diagnosis of this BVS, avoiding an exploratory laparotomy or exploratory laparoscopy as happened in our patient. Surgery can be risky in these patients because unnecessary dissection while searching for the missing gallbladder can result in injury to the biliary tree, hepatic vascular tree, or small intestine.

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