



Short Communication

Accessory lobe of the liver in a 14-year-old girl



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1. Introduction

Accessory lobe of the liver (ALL) is an uncommon congenital anomaly caused by embryonic hyperplasia. It is defined as an extra liver lobe attached to the normal liver with or without a stalk, mostly found in the infra-hepatic region. The exact incidence of ALL remains unknown, but the incidences of ALL and ectopic liver have been reported to be less than 1%.¹ More than 30 cases of ALL have been reported in the literature, but only one case involving an adult patient has been documented in Taiwan.²

2. Case report

A previously healthy 14-year-old girl was referred to a gynecology clinic due to delayed menstruation. An abdominal mass was found incidentally by ultrasound, and she was subsequently transferred to our hospital. A physical examination revealed a 7 × 8 cm, soft, smooth, non-mobile, well-defined mass in the right upper quadrant. No spider

angioma or other skin lesion was noted. The results of a complete blood count and white blood cell differential count only revealed thrombocytopenia (92,000/ μ L). Tests of the C-reactive protein level and liver function, including serum aspartate transaminase level, alanine aminotransferase level, prothrombin time, activated partial thromboplastin time, albumin, and bilirubin (direct and indirect) were normal, except mildly elevated alanine aminotransferase level (47 U/L). Serum levels of tumor markers, including carbohydrate antigen 19–9, carcinoembryonic antigen, alpha-fetoprotein, and cancer antigen 125, were all within normal ranges. The results of tests for hepatitis B and C viruses were negative. Abdominal ultrasonography revealed a mass below the liver with a blood supply from the portal vein, as well as portal vein cavernous transformation and splenomegaly. Computed tomography (CT) of the abdomen revealed portal vein cavernous transformation, splenomegaly, and an 11.8 × 7.9 × 7 cm well-defined lobular mass in the right upper abdomen, abutting the inferior margin of the right hepatic lobe through a stalk. The mass had homogeneous attenuation, the enhancement pattern was identical to that of the liver, and its vascular structures were connected to the middle hepatic vein and the main portal vein. Magnetic resonance cholangiopancreatography (MRCP) revealed similar findings and further identified portal vein obstruction by thrombus and bile duct drainage from the mass into the right hepatic duct (Fig. S1). The patient's serum levels of protein C, protein S,

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anti-thrombin III, factor V, and factor VIII were within normal ranges, and she had no history of abdominal trauma, surgery, umbilical vein catheterization, or sepsis. A diagnosis of ALL with portal hypertension due to portal vein obstruction was made. The patient had not previously experienced gastrointestinal bleeding. Upper gastrointestinal endoscopy revealed a F1 varix and a F2 varix without red color sign at distal one-third of esophagus, and endoscopic variceal ligation was performed on the F2 varix. To prevent the possibility of torsion, laparoscopic accessory liver lobe resection was conducted. Pathology revealed liver tissue, and the post-operative course was uneventful. Follow-up serum alanine aminotransferase levels were normalized 1 week later.

3. Discussion

ALL can be classified into two categories according to size: bulky, > 30 g and small, < 30 g.³ Different means of biliary drainage and the presence of a common capsule within the liver have also been used to classify ALL into three categories: (i) ALL without a common capsule within the liver and the ALL bile duct drains into the intrahepatic bile duct of the liver; (ii) ALL without a capsule within the liver, and the ALL bile duct drains into the extrahepatic bile duct of the liver; and (iii) ALL shares a common capsule with the normal liver, and the ALL bile duct drains into the extrahepatic bile duct of the liver.⁴ The ALL of our patient was bulky, it had no capsule, and its bile duct drained into the intrahepatic bile duct of the liver.

The majority of patients with ALL are asymptomatic, similar to our patient, although some may have non-specific symptoms such as abdominal pain, nausea, vomiting, constipation, or abdominal distention.¹ Thus, the diagnosis of ALL is often made incidentally during imaging performed for other reasons or during operations or autopsies. Complications of ALL include portal hypertension, torsion, infarction, and traumatic injury. The majority of these patients present with acute and intense right upper quadrant pain.³ A few cases with fluctuating impaired liver function due to torsion have been reported in the literature.⁵ Our case did not experience complications related to torsion but had transient elevated serum alanine aminotransferase levels, which returned to normal after ALL resection. Previous studies have reported cases with portal vein hypertension caused by external compression of the ALL.⁶ However, in our case, portal vein hypertension was caused by concomitant portal vein obstruction. To the best of our knowledge, no similar case has been described previously. Ultrasonography, CT, and MRI can provide sufficient and accurate information for the diagnosis of ALL.

Most patients with ALL do not require treatment. Indeed, surgical treatment is reserved for ALL patients with complications such as torsion and rupture, and large or pedunculated lobes.^{7,8} Our patient was asymptomatic, but she had a large and pedunculated liver lobe and a risk of torsion. Therefore, she underwent laparoscopic resection of the ALL. The prognosis in cases of ALL is good; even in patients experiencing complications, the majority recover well after surgery.^{3,7} To prevent the risk of esophageal variceal bleeding related to a possible increased portal blood flow following resection of a large ALL, esophageal variceal ligation can be performed prior to the procedure, such as in our case. Moreover, long-term follow-up of our patient is mandatory because of the associated portal vein obstruction.

Conflict of interest

The authors declare that they have no conflicts of interest related to the subject matter or materials discussed in this article.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.pedneo.2018.04.005>.