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Polycystic Liver Disease Treatment: A Mini Review

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Abstract

Polycystic liver disease (PLD) can be autosomal dominant or isolated with no renal impairment. The natural course of liver cysts is for them to grow in size and number, resulting in progressive disease that can lead to extremely large and incapacitating hepatomegaly. Only symptomatic hepatomegaly (pain, inability to eat, weight loss, dyspnea) or cystic complications like infection or intracranial haemorrhage should be treated. PLD treatment thus encompasses a wide range of therapeutic options, from non-intervention to liver transplantation, including needle aspiration evacuation with sclerosant injection, laparoscopic fenestration, and fenestration by laparotomy combined with liver resection. The choice of treatment is determined by the symptomatology, intrahepatic extension of the lesions, and the patient's overall health.

Key words: PKD • Renal impairment • Polycystic liver disease

Introduction

Because the vast majority of PLD consists of multiple small cysts that are impossible or difficult to fenestrate, hepatic resection is commonly chosen. Because cysts are inhomogeneously distributed in the hepatic parenchyma, with most areas less affected; the preservation of this less-involved territory allows liver regeneration to occur in a cyst-free environment. PLD hepatotectomies are technically challenging because the cysts compress the planes as well as the vascular and biliary structures. Liver transplantation, whether isolated or in conjunction in cases of severe malnutrition and/or end-stage renal disease, or if the volume of remnant parenchyma is insufficient and suggests failure of a partial hepatectomy [1].

Description

Although some cases occur sporadically, polycystic liver disease (PLD) is primarily a genetic disease. Several clinical manifestations of impaired hepatic or renal function, or both, are described. Polycystic kidney disease (PKD) is a common genetic disease that affects nearly 100,000 people in France and 12 million people worldwide. Hepatocellular insufficiency is never a feature of renal involvement, which eventually leads to destruction of the renal parenchyma and renal insufficiency PLD. Patient symptomatology is primarily caused by hepatomegaly [2], which causes pain, digestive problems, and respiratory discomfort.

The decision between all of these treatments is still difficult and should only be undertaken by specialised multidisciplinary teams, especially since this requires partial hepatic resection. The indication has early morbidity that may be turbulent .The purpose of this article is to summarise therapeutic methods their indications and outcomes in the treatment of PLD.

PLD symptoms and complications

Symptoms are primarily caused by the volume of the hepatomegaly, with digestive and pulmonary symptoms caused by compression of adjacent organs such as the stomach, duodenum, diaphragm, or, in rare cases, the vasculobiliary structures. PLD may occasionally cause ascites or symptoms related to haemorrhage or infection involving some dominant cysts.

The liver volume can increase up to tenfold, causing progressive functional discomfort such as difficulty bending at the waist to put on shoes or sleeping in certain positions, particularly lying prone. Hepatomegaly is a serious condition. Although not synonymous with pain, some patients describe a significant and troubling feeling of heaviness. In patients with severe hepatomegaly, there is frequently gastric and/or duodenal compression, resulting in postprandial nausea and vomiting gastroesophageal reflux disease, and digestive intolerance [3,4]. Even with only mild hepatomegaly, cysts in the left liver or segment I can cause gastric or duodenal compression when these symptoms are mild, the patient will limit the number of meals he or she consumes.

The development of cysts at the level of the hepatic dome, which elevate the diaphragm to the level of the pulmonary hila, causes pulmonary symptoms. This thoracic volume restriction is most likely exacerbated by diaphragmatic dysfunction caused by stretching, particularly on the right side. Patients report exertional dyspnea, which can be disabling. Some hepatic hilum cysts can compress the portal bifurcation, causing portal hypertension (PHT) and ascites, especially if the patient is malnourished. The development of cysts at the level of the hepatic dome, which elevate the diaphragm to the level of the pulmonary hila, causes pulmonary symptoms. This thoracic volume restriction is most likely exacerbated by diaphragmatic dysfunction caused by stretching, particularly on the right side. Patients report exertional dyspnea, which can be disabling. Some hepatic hilum cysts can compress the portal bifurcation, causing portal hypertension (PHT) and ascites, especially if the patient is malnourished.

Treatment

Symptomatic treatment: When PLD is asymptomatic or the symptomatology cannot be attributed to liver volume, cyst size, or changes, therapeutic abstinence is the rule. Because the natural history of this progressive disease is unknown, it is impossible to predict whether the patient's PLD will become symptomatic, in what form, and when. On the other hand, the patient can be reassured that there is no risk of serious complications. Except in the two rare cases of haemorrhage and infection, the treatment goal in symptomatic patients is to reduce the volume of hepatomegaly [5]. Aside from medical treatment to reduce intracystic secretion and treatment techniques for hepatomegaly include needle aspiration/alcoholization, laparoscopic cyst wall fenestration, partial liver resection combined with contralateral fenestration, and total liver resection followed by transplantation, which results in an increase in cyst volume.

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infection, the treatment goal in symptomatic patients is to reduce the volume of hepatomegaly. Aside from medical treatment to reduce intracystic secretion and treatment techniques for hepatomegaly include needle aspiration/alcoholization, laparoscopic cyst wall fenestration, partial liver resection combined with contralateral fenestration, and total liver resection followed by transplantation, which results in an increase in cyst volume. Renal artery embolization of a polycystic kidney prior to a renal transplant has been shown to reduce kidney volume and facilitate the transplant [6]. Embolization of a branch of the hepatic artery (usually on the right) to reduce hepatomegaly has been done in several series, but only one has been published within Japan The most significant series included 30 patients who had their hepatic artery embolized without serious complications and with some efficacy in terms of volume reduction and symptom improvement. We tried this treatment on two patients and found no difference in volume or symptoms (unpublished data); this suggests that more research is needed.

Because cyst sclerosis is extremely painful, it is performed under general anaesthesia with ultrasound guidance. The cyst is evacuated before being filled with 95% alcohol to a maximum volume of 150 mL, before re-aspiration of the sclerosing product. It is effective for symptom relief in 85% of cases, but it takes 6 months after alcoholism to reduce the size of the treated cyst. Sclerosis, or simple puncture aspiration in some cases, allows one to attribute the cause of symptoms to the presence of the cyst. A suction drain may also be required for a few days.

Complications

Analgesics are the only treatment recommended for an episode of acute bleeding because, with the exception of patients on anticoagulants, there is no indication for hospitalisation or transfusion. For the persistence of chronic pain away from one can propose a puncture alcoholization during the acute episode. In practise, this procedure is performed three months after the bleeding episode to prevent the theoretical passage of alcohol into the bloodstream, which could occur in the case of recent haemorrhage [7]. However, the extra space created by a previous resection may make access and exposure easier. Vascular and biliary anastomoses are particularly complex and risky due to frequent dilation of the common bile duct (CBD), which results in a mismatch for anastomotic calibre and greater fragility of the arterial walls, which may result in thrombosis.

Conclusion

Only symptomatic PLD requires treatment. The treatment options are not yet completely standardised. For comparatively tiny, voluminous cysts, needle aspiration with alcohol sclerosis and fenestration, preferably via laparoscopy, are the preferred treatments. Partial hepatectomy, on the other hand, should be preferred for incapacitating PLD with major hepatomegaly, especially in the absence of renal insufficiency. Liver transplantation should be reserved for Gigot Class III PLD patients, as well as those for whom a technically feasible left lobectomy would not be sufficient in terms of volume reduction and/or who require a combined kidney transplant.

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Conflict of Interest

There are no conflicts of interest by author.

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