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Laparoscopic Treatment of Hepatic Cysts: A 10-Years Single Institution Experience

Nicola Tartaglia[1*], Alessandra Di Lascia[1], Pasquale Cianci[1], Alberto Fersini[1], Vincenzo Lizzi[1], Antonio Ambrosi[1] and Vincenzo Neri[1]

[1]Department of Medical and Surgical Science, University of Foggia, Italy.

Authors' contributions

This work was carried out in collaboration between all authors. Authors NT and ADL contributed equally to this work. Authors NT, AF, AA and VN performed surgical operations. Authors PC, ADL and VL analyzed the data. All authors read and approved the final manuscript.

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ABSTRACT

Aims: This study presents our experience in the laparoscopic management of simple hepatic cysts (SHCs) and the polycystic liver disease (PCLD).
Study Design: Retrospective institutional study.
Place and Duration: Department of Medical and surgical Science, University of Foggia, Foggia, from January 2004 to December 2014.
Methodology: Laparoscopic deroofing was performed in 20 consecutive patients. There were 15 cases with SHCs and only 5 cases with PCLD (Gigot's type I). In SHCs group, cyst was single in 10 cases and multiple in 5. Cysts were located in both hepatic lobes in 4 cases. The two groups were homogeneous for age, sex and ASA score. Preoperative investigations include routine laboratory tests, ultrasonography and the use of computed tomography in order to make diagnosis ad to rule out parasitic and neoplastic liver cysts. CT scan was performed in all cases to assess the characteristics, dimensions, and exact position of the lesion. Surgery was planned for all patients because of evident and persistent symptomatology, characterized of the presence from no less of 6 months of typical symptoms such as nausea, vomiting and epigastric pain. In our series, 6 cases of cholelithiasis were associated.

**Corresponding author: E-mail: nicola.tartaglia@unifg.it;*

Results: The analyzed outcome variables included surgical procedure, operative time, blood loss, length of hospital stay, complications, and medium follow-up period. All the patients underwent laparoscopic deroofing of the larger cysts and puncturing of the smaller cysts. There were no conversions. The mean operative time was estimated twice in the group PCLD than SHCs (110 min vs 60 min). The histological examinations revealed the typical pattern of the simple liver cysts in any case, without evidence of malignancy. No significant blood loss was found.
The total morbidity recorded was 25% (5/20), 2 cases in the group of SHCs (13,3%) and 3 cases in the PCLD one (60%) and was characterized of 2 cases of ascites through trocar insertion sites after removal of drainage tube and 3 case of pleural effusion. There were no significant group differences in term of length of hospital stay. The follow-up period (a mean of 22 months) confirmed that all the patients remained free of symptoms and relapse of the disease.
Conclusion: The technical feasibility and the good short- and medium-term results made the laparoscopic approach the procedure of choice for the management of symptomatic liver cysts.

Keywords: Hepatic cyst; liver disease; laparoscopy; laparoscopic deroofing; polycystic liver disease; minimally invasive surgery.

1. INTRODUCTION

Hepatic cysts represent a heterogeneous cluster with regard to pathogenesis, clinical presentation, diagnostic findings and therapeutic management. They are classified as congenital or acquired, based on the pathogenesis. Congenital forms include the simple hepatic cysts (SHCs) and the polycystic liver disease (PCLD). The acquired lesions are represented by hematomas, parasitic cysts, neoplastic and metastatic cysts. These lesions are also classically divided with a different criterion into parasitic hepatic cysts (PHCs) and non-parasitic hepatic cysts (NPHCs), with the latter being the most prevalent worldwide (prevalence of 4-7% in the general population) [1]. In most cases, cysts are asymptomatic and detected as an incidental finding during abdominal exams. Sometimes, these lesions are not easily differentiated at initial presentation from the cases with neoplastic evolution (cystoadenoma, cystoadenocarcinoma) and so their management can become challenging. Some points of discussion are highlighted in relation to the surgical treatment for SHCs and PCLD: which patients, when, with whom operative procedure.

The aim of this study was to assess our single institution experience in the laparoscopic management of the SCHs and PCLD during a 10-year period in order to determine surgical indications, immediate and medium-term results, relapse incidence of the laparoscopic approach.

2. MATERIALS AND METHODS

From January 2004 to December 2014, a total of 570 patients with hepatic cysts were admitted to the Department of Medical and Surgical Science, University of Foggia; 20 consecutive patients were referred for evaluation and treatment of simple liver cysts (solitary and multiple cysts) or PCLD. The study group consisted of 7 males and 13 females with a mean age 49 years (range 35-60 years). There were 15 cases with SHCs and only 5 cases with PCLD (Gigot's type I) [2]. These patients had not a family history of PCLD and Autosomal Dominant Polycystic Kidney Disease (ADPKD). No Previous treatment of cysts had been performed in our patients. No patients with hepatic cysts needed major resections or liver transplantation. Follow-up in asymptomatic patients, without other pathologies, has been one year and then every 3 years. All the patients signed an informed consent to the use of personl data.

Demographic data, size of the dominant cyst, cysts sites and ASA score were reported in Table 1.

The most common symptoms were epigastric pain and abdominal swelling, the others were early satiety, heartburn, nausea, vomiting, supine dyspnea and palpable mass (Table 2).

The mean duration of symptoms was 6 months (range, 3-24 months).

A careful traveling history was taken and a thorough examination with laboratory tests, ultrasonography (US) and computed tomography (CT) was used to make diagnosis ad to rule out parasitic and neoplastic liver cysts. Routine laboratory investigations, including liver and renal function tests and bleeding profile, were done in all patients and were normal; serum antibodies

for Echinococcus were negative such as the tumor markers (α-fetoprotein, carcinoembryonal antigen and carbohydrate antigen 19-9). On clinical examination, hepatomegaly and palpable mass were present in 4 patients. According to American Society of Anesthesiologists (ASA) classification, ASA I was observed in 16 patients (80%) and ASA II in 4 patients (20%). The initial imaging modality was the US in all cases, followed by CT scan. Simple liver cysts typically presented on US as monolocular, anechoic and sharply demarcated lesions with thin wall and posterior acoustic enhancement. CT scan was performed in all cases to assess the characteristics, dimensions, and exact position of the lesion. On CT scan, SHCs and PCLD appeared as well-demarcated lesion with uniform fluid-density without enhancement after contrast administration (Fig. 1, Fig. 2).

Table 1. Demographic data, size of the dominant cyst, cysts sites and ASA score of all patients

Data	SHCs (15)	PCLD (5)
Sex (M/F)	5/10	2/3
Median age (years)	50 (35-60)	46 (43-59)
Median size, cm (range)	8 (5-17)	11 (10-13)
Bilateral location, *n* (%)	4 (27%)	5
Right hemiliver, *n* (%)	5 (33%)	/
Left hemiliver, *n* (%)	6 (40%)	/
ASA I, *n* (%)	14 (95%)	2 (4%)
ASA II, *n* (%)	1(5%)	3 (6%)

Table 2. Clinical findings in all patients

Indications symptoms	Simple cysts (15) *n* (%)	PCLD (5) *n* (%)
Epigastric pain	13 (87%)	3 (60%)
Abdominal swelling	12 (80%)	3 (60%)
Early satiety	6 (40%)	3 (60%)
Heartburn	2 (13%)	1 (20%)
Nausea	6 (40%)	2 (40%)
Vomiting	3 (20%)	2 (40%)
Supine dyspnea	0	1 (20%)
Palpable mass	2 (13%)	2 (40%)

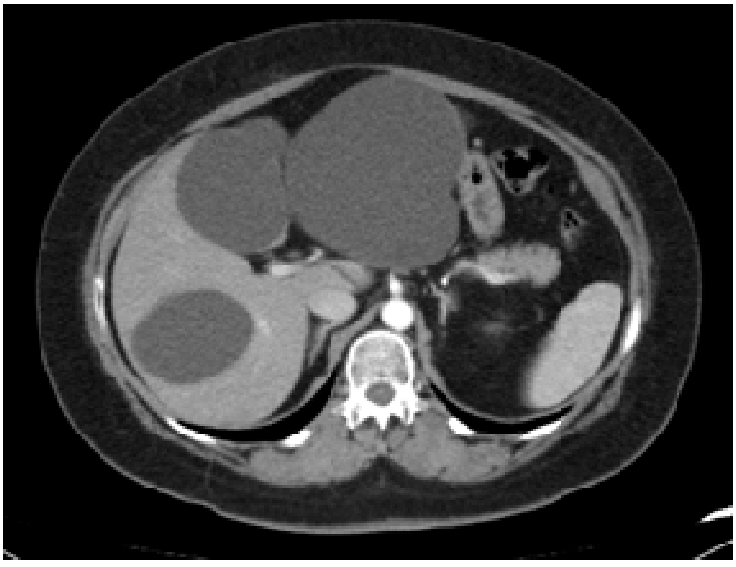

Fig. 1. Abdominal CT of a patient with multiple simple cysts, involved both hepatic lobes; arterial phase

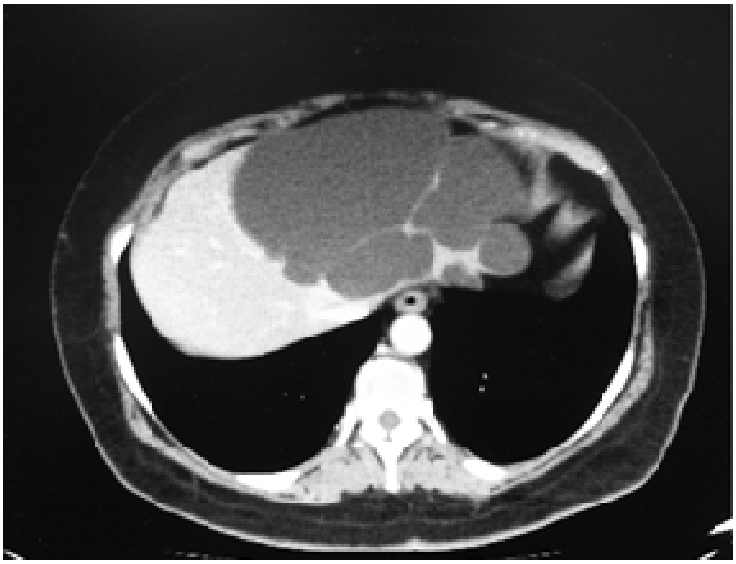

Fig. 2. Abdominal CT of a patient with PCLD (Gigot's type I), arterial phase

The imaging examinations excluded the presence of septations, calcifications, papillary projections and split wall. None of the cysts was complicated, infected or ruptured. Morphological definition and topography of the cystic lesions was also determined by magnetic resonance imaging (MRI) in few cases with typical aspect of homogeneous hypointensity in T1-weighted imaging and homogeneous hyperintensity in T2-weighted imaging. In SHCs group, cyst was single in 10 cases and multiple in 5. Cysts were located in both hepatic lobes in 4 cases. Cysts' median size was 8 cm (range, 5-17 cm). In PCLD, cysts were several, involved any hepatic segment and the dominant cysts had a median diameter of 11 cm (range, 10-13 cm). In our series, 6 cases of cholelithiasis were associated. Elective laparoscopic surgery was planned for all patients because of the presence of evident and persistent symptomatology (Table 2). All the patients underwent laparoscopic deroofing of the larger cysts and puncturing of the smaller cysts. The first step is the decompression of the cyst with the aspiration of its content; the cyst collapse and provides the adequate access to the superior part of the cyst wall in order to facilitate the mobilization and the radical excision of its roof. The roof of the cyst is then largely excised with monopolar hook and sent for histological examination. Then, the residual cavity was widely in communication with the peritoneum. The evaluation of the cystic fluid, typically clear and serous, is important in order to define the benign nature of the cyst. It is important to examine the cavity of the cyst carefully to preclude both the contingent tissue proliferation and the connection to the biliary ducts. In case the cyst was loculated, all the septa were broken and laid open. Great care was taken to avoid vascular and biliary tract injury within the cystic septa and on the cyst edges. In case of deeply cysts their fenestration occurred in a second time and through the superficial ones. In PCLD (Gigot's type I), this procedure has to be repeated for each cyst and so can become tedious and time consuming, but it is crucial that all cysts are deroofed, because the success of the operation depends on this meticulous treatment [3,4]. Peritoneal drainage was placed in all patients.

3. RESULTS

There was no intraoperative or postoperative mortality in this series. The mean operative time was 60 minutes in simple liver cysts and 110 minutes in PCLD, because of the necessity of treating numerous cysts. There were no conversions. No significant blood loss was found. Liquids were started on the same evening as surgery, and a normal diet was started on the first postoperative day (POD). Analgesia was required only in 1 POD. Fluid drainage was present for 2 days to 5 days. Between 30 mL and 370 mL of fluid was recorded per patient during the 3 days (average). At discharge, the complete resolution of symptomatology was reached in all cases. The histological examinations revealed the typical pattern of the simple liver cysts in any case, without evidence of cystoadenoma or

carcinoma. Intraoperative complications were not detected in our series. Postoperative morbidity consisted of 2 cases of ascites through trocar insertion sites after removal of drainage and 3 case of pleural effusion. All cases treated conservatively with medical therapy.

The total morbidity recorded was 25% (5/20), 2 cases in the group of SHCs (13,3%) and 3 cases in the PCLD one (60%). The Table 3 shows the data compared between the two groups. There were no cases of bile leakage and neither of severe cardiopulmonary complications.

All patients were followed for a mean of 22 months (range, 8 to 48 months). The first follow-up was 30 days after the surgery, the subsequent ones were every 6 months. The follow-up consisted of the clinical observation, laboratory investigations and US. The long-term outcomes confirmed that all the patients remained free of symptoms and relapse of the disease.

4. DISCUSSION

Liver cysts are classified as true or false based on the presence of an epithelial lining. *True liver cysts* include congenital cysts (SHCs and PCLD), parasitic cysts (caused by Echinococcus Granulosus or Echinococcus Multilocularis), neoplastic cysts (cystadenoma, cystadenocarcinoma, metastatic cancers from ovaries, pancreas, kidneys) and biliary duct-related cyst (Caroli disease, bile duct duplication and peribiliary cysts). *False liver cysts* may be caused by spontaneous intrahepatic hemorrhage, post-traumatic hematoma or intrahepatic biloma. SHCs are the most prevalent and have a tendency to follow a benign course. As a result of the frequent use of abdominal imaging techniques in recent years, the incidence of so-called coincidental cysts has increased up to 18% [5]. Women are more commonly affected than men, with female to male ratio 1,5:1 and the incidence is larger in adults older than 50 years [6]. The first important step, regarding liver cystic lesions, is to make a diagnosis of nature of cysts. The second is determining whether the patient's symptoms are related to cystic lesions. The third is deciding the appropriate therapy. Cystic lesions of the liver encompass a wide spectrum of disorders. The vast majority of liver cysts are asymptomatic; they can occasionally be felt as a mass during physical examination. About 5% of patients are symptomatic [7]. They can produce symptoms due to their size, anatomical site, or when they become complicated. Most commonly, cyst enlargement can produce vague symptoms such as epigastric pain, nausea, early satiety, vomiting [7-9]. Complications due to their increased size and central liver localization include obstructive jaundice, portal hypertension and Budd-Chiari Syndrome. Although quite rare, others complications of liver cysts consist of: intracystic hemorrhage, infection, rupture to the peritoneal cavity, torsion [10]. Laboratory findings are predominantly normal, only into a small number of patients it is found a little alteration of liver function tests [11]. US remains the most accurate, non-invasive and cost-effective imaging modality for diagnosis simple cysts with a sensitivity and specificity of approximately 90%; the diagnosis of a simple cyst is based on the following US criteria: anechoic (fluid filled cavity), no septations, smooth borders, strong posterior wall echoes, spherical or oval shaped [7]. Recent advances in CT and MRI technology might result in even higher sensitivity rates [12]. The casual relationship between abdominal pain and the presence of cystic lesions should be always questioned before surgery, and other possible causes of pain or the presence of other concomitant pathologies (i.e. gastroesophageal reflux) should always be excluded [13]. Differential diagnosis among several types of true liver cysts is of paramount importance. Complicated cysts, echinococcosis [14] and cystic neoplasms (cystoadenoma and cystoadenocarcinoma), which cause a diagnostic enigma, demand accurate diagnosis in the early stage because specific therapy could be required [15]. Cystadenoma (HC) and cystadenocarcinoma (HCa) are biliary cyst tumors that originate from the biliary epithelium, represent less of 5% of all cystic lesions, generally asymptomatic, that occur more commonly in female older than 40 years of age. Abnormal serum and cystic fluid markers, such as Ca 19-9 and CEA levels, may favor diagnosis but this is a variable finding. Diagnostic imaging studies include a multiloculated lesion with internal septations, thickened and irregular wall, papillary projections, calcifications and wall enhancement. However, in most cases, differentiation between HC and HCa is not possible [16]. The same problem arises in differentiating hydatid cysts and complicated cysts from malignant ones because in many cases intracystic haemorrhage, calcifications and septations are present in all these lesions [17]. The recent development of microbubble contrast-enhanced ultrasound (CEUS) enables us to

visualize vascular flow within septa or solid components of cysts, which is absent in simple cysts with intracystic haemorrhage [18]. The rupture of blood vessels inside of cystic wall due to rapid enlargement is the mechanism of bleeding in a simple cyst. On CEUS, the HC or HCa presents with septa enhancement during arterial phase and hypoenhancement during the portal and late phases. So, CEUS can be helpful in differentiating HC and HCa from complicated cysts when USG, CT or MRI is inconclusive [19]. Fine needle aspiration (FNA) could be of additional help to exclude complicated cysts [20]; however due to the risk of malignancy, FNA is generally not performed.

The data of literature confirms the rarity of HCa/HC findings (2,2%) with an extremely low percentage of malignancy (0,2%); on the other hand the intraoperative histological examination of the cystic wall should be performed to exclude a HCa/HC which requires a resection with clear margins [21]. SHCs arise congenitally from aberrant bile duct cells and contain a clear fluid. Because bile duct epithelium covers the simple cyst inner lining, it is hypothesized that simple cysts arise during embryogenesis when intrahepatic ductules fail to connect with extrahepatic ducts [7].

Furthermore, the presence of multiple hepatic cystic lesions must raise the suspicion of PCLD and require further screening (investigations of both kidneys and extensive family history). PCLD is a genetic disease responsible for the development of multiple hepatic cysts. It presents in two forms, with or without the association with autosomal dominant polycystic kidney disease (ADPKD).

Both have an autosomal dominant transmission and similar clinical presentation. PCLD associated with ADPKD is linked with mutations in the PKD1 (short arm of chromosome 16, encoding polycystin-1) or PKD2 gene (chromosome 4, encoding polycystin-2). Polycystin-1 and polycystin-2 are important for adequate functioning of the primary cilium [22]. Whereas isolated PCLD is associated with heterozygous mutation in PRKC-SH or SEC63 genes. These genes encode hepatocystin and SEC63 proteins, respectively. Hepatocystin acts in the folding process of proteins, while SEC63 acts as part of the endoplasmic reticulum translocon [22]. The pathophysiology of PCLD is linked to a malformation of the hepatic ductal plate and cilia of cholangiocytes. The ductal plate is the anatomical template for the development of the intra-hepatic bile ducts, through a complex sequence of growth and apoptosis. Incorrect involution of the ductal plate results in complexes of disconnected intralobular bile ductules (von Meyenburg complexes) [23]. As consequence, multiple cysts arise from progressive dilatation of these abnormal ductules that display the same epithelium and structures of functioning cholangiocytes. Also, ciliary defects are responsible for the hyperproliferation of cholangiocytes and for the cystogenesis that is a consequence of the alterated balance between fluid secretion and absorption [24]. The natural history of PCLD is characterized by a continuous increase in the volume and the number of cysts.

Overall prevalence is the same in gender, but is more common in the female population. Pregnancy, multiparity and use of exogenous female steroids further increase the risk for severe hepatic cystic disease, because of the cystic increase in size and number [25]. It has been proposed that sporadic cases of PCLD should be diagnosed when a patient has more than 15 to 20 cysts and no previous family history, while four cysts suffice in the presence of a positive familiar history [26].

Several clinical classification have been proposed to grade the severity of PCLD [27].

Gigot's classification [2] is used for staging based on CT findings:

- type I, less than 10 large cysts (more than 10cm in maximum diameter);
- type II, with multiple and widespread cysts of moderate size, but with remaining large areas of non-cystic liver parenchyma;
- type III, massive, diffuse involvement of liver by small and medium-sized cysts with only a few areas of normal liver tissue.

According to *Morino's classification* [28] PCLD is divided into 2 groups:

- Type 1: limited number of large cysts in the anterior segments of liver;
- Type 2: multiple and widespread small cysts.

Qian's classification has been used in the context of familiar screening and relies on the number of cysts and the presence of symptomatic hepatomegaly [29].

Table 3. Short-term results

Parameter	SHCs (15)	PCLD (5)
Hospital stay (days)	5 (range, 3-7)	6 (range, 4-10)
Mean operative time (min)	60 (range, 40-80)	110 (range, 90-130)
Ascites *n* (%)	0	2 (40%)
Pleural effusion *n* (%)	2 (13,3%)	1 (20%)

Finally, *Schnelldorfer's classification* aims at differentiating patients in order to propose the best therapy and it is based on the severity of symptoms, cysts characteristics, the number of normal hepatic sectors and the presence of portal vein or hepatic veins occlusion [30]. The treatment of hepatic cysts is indicated when they became highly symptomatic or complicated and when they have rapid growth [8].

Uniform management of the cystic liver disease has not been defined clearly and multiple therapeutic options have been advocated for symptomatic cases: sclerotherapy (percutaneous aspiration of the cyst followed by instillation of a sclerosant substances), or surgical treatments such as laparoscopic or open deroofing, hepatic resection and liver transplantation [9].

Although the easiness and the effectiveness of percutaneous sclerotherapy advocated by several studies, symptomatic recurrence rate is around 20% after 4 months [31]. Due to high recurrence rates, management by aspiration followed by sclerotherapy should be reserved for those patients who are not elegible for surgery and general anesthesia. Surgical fenestration, also known as deroofing, consists of an excision of the cystic roof to provide communication between the cyst and the peritoneal cavity. Fenestration may be achieved by laparoscopy or laparotomy.

Laparoscopic deroofing was first reported in 1991 [32], and has become the procedure of choice for SHCs [4,5], because it is associated with reduced postoperative pain and discomfort, early mobilization, shorter hospital stay and cosmetic benefits.

To avoid recurrence, it is necessary to resect as much of the wall as possible to prevent closure of the remnant wall and reaccumulation, such as we have done in our series. Hemorrhage and biliary injury [33] are, although rare, the major intraoperative complications. Infection and ascites may occur in the postoperative phase. Laparoscopy deroofing shows morbidity ranges of 0%-50% [11], with recurrence rates at 0-25% [13,33] and reoperation rates at 4-9% [8,13]. No perioperative mortality was present in our experience, as also reported by most authors [11,13]. The management and the therapeutic approach in the cases of PCLD appears to be even more complex and controversial. In these cases, the current recommended surgical management is: laparoscopic fenestration in PCLD type I; open deroofing and hepatic resection in PCLD type II; resection or liver transplantation in PCLD type III [6]. Combined hepatic resection and fenestration is more effective for reducing the hepatic mass and relieving gastric compression [6]. In the cases of PCLD the laparoscopic deroofing shows a recurrence rates up to 25% [23], while the hepatic resection and transplantation are associated to a higher morbidity rates [27]. Orthotopic liver transplantation (OLT) is indicated for patients with progressive PCLD after resection/fenestration, patients with concurrent liver dysfunction and renal failure, and patients with diffuse PCLD without segmental sparing (Gigot's type III). In these cases, OLT is effective, but survival is limitated, perioperative morbidity is 40-50%, whereas overall mortality is 10-17% [23]. Medical management may be valuable in symptomatic patients with Gigot's type II/III. The somatostatin analogue, lanreotide or octreotide, reduces the volume of polycystic livers but has a modest clinical effect [27]. The rationale for somatostatin analogue therapy is its inhibitory effect on cholangiocyte proliferation and cyst fluid production [34,35]. Currently, somatostatin analogues are indicated only for a selected group of patients with symptomatic PCLD in whom the risks for surgical intervention are not justified, or in whom the surgical intervention is technically challenging [27]. Sclerotherapy showed ineffective in the management of PCLD, only 20% will have partial or full regression of their disease [36]. Nowadays, the selection of the appropriate approach for the treatment of cystic liver disease remains a clinical challenge. The risks and the benefits of each of the possible therapeutic options have to be carefully evaluated and put in the contest of the clinical presentation and condition of each patient. In our series, we have treated with laparoscopic

deroofing patients with both SHCs and PCLD type I. In fact, we think that in Gigot's type I, symptoms might not be related to the size of the entire liver but to the size of the largest cysts and that the main aim of laparoscopic deroofing is to achieve complete and definitive decompression of cysts, leading to disappearance of the patient's symptoms. Our study focused on a selected group of patients who underwent laparoscopic fenestration whose symptoms were specifically related to cysts. For this reason, this therapeutic approach provided immediate complete relief of symptoms for both SHCs and PCLD.

The procedure was completed successfully by laparoscopy for all 20 cases. The location of cysts was not a contraindication to the laparoscopic approach. In our series, we recorded neither mortality nor intraoperative complications; the postoperative morbidity has been 25%, with no case of recurrence and without the necessity of reoperation, because of the complete regression of the cystic disease and the symptomatology.

5. CONCLUSIONS

The long-term outcomes confirmed that all the patients remained free of symptoms and of radiologic refilling of cysts. By choosing laparoscopic surgery, it is possible to accomplish important goals: symptom relief equivalent to that with open fenestration with the benefits of laparoscopic surgery by reducing the hospital stay. The technical feasibility and safety as well as the good short- and medium-term results made the laparoscopic approach the procedure of choice for the management of symptomatic liver cysts.

ETHICAL APPROVAL

The authors have obtained all necessary ethical approval from suitable Institutional Committee. This confirms either that this study is not against the public interest, or that the release of information is allowed by legislation.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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