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Open Surgery for Hepatic Hydatid Disease

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Hydatid disease is a zoonosis caused by the larvae of *Echinococcus granulosus*. Humans are an intermediate host and are usually infected by direct contact with dogs or indirectly by contaminated foods. Hydatid disease mainly involves the liver and lungs. The disease can be asymptomatic. Imaging techniques such as ultrasonography and computed tomography are used for diagnosis. The growth of hydatid cysts can lead to complications. Communication between bile duct and cysts is a common complication. The goal of treatment for hydatid disease is to eliminate the parasite with minimum morbidity and mortality. There are 3 treatment options: surgery, chemotherapy, and interventional procedures. Medical treatment has low cure and high recurrence rates. Percutaneous treatment can be performed in select cases. There are many surgical approaches for managing hydatid cysts, although there is no best surgical technique, and conservative and radical procedures are used. Conservative procedures are usually preferred in endemic areas and are easy to perform but are associated with high morbidity and recurrence rates. In these procedures, the parasite is sterilized using a scolicidal agent, and the cyst is evacuated. Radical procedures include hepatic resections and pericystectomy, which have high intraoperative risk and low recurrence rates. Radical procedures should be performed in hepatobiliary centers. The most common postoperative complications are biliary fistulas and cavity-related complications. Endoscopic retrograde cholangiopancreatography can be used to diagnose and treat biliary system complications. Endoscopic sphincterotomy, biliary stenting, and nasobiliary tube drainage are effective for treating postoperative biliary fistulas.

Key words: Hydatid disease – Echinococcosis – Open surgical approach

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Table 1 World Health Organization–Informal Working Group on Echinococcosis (WHO-IWGE) classification of ultrasound images of cystic echinococcosis cysts

WHO-IWGE Classification	Radiologic characteristics	Definition of cyst
CL	Unilocular cystic lesion with uniform anechoic content, cyst wall not visible	Cystic lesion
CE1	Unilocular cystic lesion with uniform anechoic content, cyst wall visible, snowflake sign	Active cyst
CE2	Multivesicular, multiseptated cysts, daughter cysts present, honeycomb sign	Active cyst
CE3a	Unilocular cyst containing liquid with a floating membrane inside, water-lily sign	Transitional cyst
CE3b	Cysts with daughter cysts in solid matrix	
CE4	Cysts with heterogeneous hypoechoic or hyperechoic degenerative contents, no daughter cysts	Inactive cyst
CE5	Cysts characterized by a thick calcified wall, which is arch shaped, producing a cone-shaped shadow; degree of calcification varies from partial to complete	Inactive cyst

Cystic echinococcosis (CE) or hydatid disease is a zoonosis caused by the larvae of *Echinococcus granulosus*. Cystic echinococcosis is common in areas where sheep and cattle are raised and is endemic to Mediterranean countries, the Middle East, Central Asia, South America, Africa, Australia, and New Zealand.[1–3] The incidence of the disease has been increasing owing to immigration and travel to nonendemic countries.

Dogs are the definitive host for *E. granulosus*, while sheep and cattle are the major intermediate hosts. Humans are also an intermediate host and are infected accidentally, typically by direct contact with canines or indirectly by contaminated food and water. After ingesting cestode eggs, the larvae or oncospheres hatch in the stomach or small intestine, penetrate the intestinal wall, enter the portal circulation, and reach the liver. The organs involved most frequently are the liver (52%∼77%) and lungs (10%∼40%).[4] The kidneys, brain, bone, muscles, thyroid, subcutaneous tissue, and other organs are involved less frequently (20%).[5–8]

The metacestode develops into a single, fluid-filled cyst (hydatid) after an incubation period that differs with the location of the cyst and adjacent tissue; it takes several years because the metacestode develops slowly. The fluid in the cyst is clear, colorless, sterile, and contains highly antigenic material that can lead to anaphylactic reactions when a cyst ruptures. The cysts grow 1 to 30 mm in diameter per year. The growth of the cysts causes a reaction in the host tissue and the formation of a fibrous tissue layer (the pericyst or ectocyst). The endocyst that is formed by the parasite consists of 1 or 2 layers. The outer layer (laminated layer) is an acellular membrane that is permeable to water and electrolytes, which protects the cyst from host enzymes, bile, and bacteria. The inner layer is a cellular membrane that forms brood capsules containing protoscolices. Cysts with a laminated layer only are called univesicular or sterile cysts. Cysts that have both laminated and germinal layers are called fertile or multivesicular cysts. Protoscolices are released into the cyst fluid and daughter cysts develop. This process is called endogenic vesiculation. The process can also occur outside the cyst, within the pericyst, and this is called exogenous vesiculation.[9] Exogenous vesiculation is likely responsible for the major inflammatory and parasitic complications and postoperative recurrence.[10]

Hydatid disease can be asymptomatic, and the diagnosis is often made incidentally. The presenting symptoms depend on the size, number, and location of the cysts, organ involved, and complications. The most common symptoms and signs are abdominal pain, a palpable mass in the right upper quadrant, hepatomegaly, and dyspepsia. Cholangitis, jaundice, fever, anaphylaxis, and acute abdominal pain are symptoms of complicated hydatid disease.

Hydatid disease is diagnosed using imaging techniques such as ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI). Serologic tests used to diagnose hydatid disease include immunofluorescence assays, indirect hemagglutination, immunoelectrophoresis, and enzyme-linked immunosorbent assays (ELISA).[11,12] US has the best diagnostic efficiency, while CT helps in the accurate anatomic localization of cysts.[13] There used to be several US classifications for liver hydatid disease, but the World Health Organization–Informal Working Group on Echinococcosis (WHO-IWGE) standardized the classification system in 2001 (Table 1).[4]

Hydatid disease should be treated once it is diagnosed because the cysts usually grow and cause complications, although they can rarely collapse spontaneously and might disappear or calcify. The main complications are rupture into the peritoneal cavity, infection, compression of or communication

with the biliary tree, anaphylaxis, and secondary hydatidosis. Complications are observed in one third of patients. The most frequent complications are communication between the cyst and biliary tree (9%∼25%) and peritoneal perforation (10%∼16%).[3]

The ideal treatment should completely eliminate the parasite and prevent recurrence of the disease with minimum morbidity and mortality. There are 3 treatment choices for hydatid disease: medical (chemotherapy), percutaneous drainage or puncture-aspiration-injection-reaspiration (PAIR), and surgery. Surgery is the only therapeutic option in complicated cases. The treatment for uncomplicated hepatic hydatid disease should be individualized. Medical treatment alone is not satisfactory. It has low cure (10%–30%) and high recurrence (3%–30%) rates.[14] It should be used as an adjuvant to surgery and percutaneous treatment. Medical treatment can be used alone in patients who have disseminated disease, who have comorbid disease, and who have small (<5 cm) CE1 and CE3 cysts in the liver and lungs.[1]

Percutaneous drainage is suitable for CE1 and CE3a cysts >5 cm. It is an option in patients who are not suitable for surgery or refuse surgery or who have disease recurrence or disease that is refractory to medical treatment. It is contraindicated in CE2, CE3b, CE4, and CE5 cysts and lung hydatid disease.[1]

Surgical treatment is indicated in patients who have large CE2 and CE3b cysts with multiple daughter cysts, for superficial liver cysts that might rupture, infected cysts, cysts communicating with the biliary tree, and cysts exerting pressure on adjacent vital organs. Contraindications to surgery are multiple cysts, cysts that are difficult to access, dead cysts, inactive cysts that are partially or totally calcified, very small cysts, and patients who are not suitable for surgery because of their general condition.

Medical therapy with antihelminthic drugs (*e.g.*, albendazole, mebendazole, praziquantel) is used to prevent recurrence and secondary hydatidosis. Treatment with albendazole 10 to 15 mg/kg daily for 4 days to 3 months before surgery and 1 to 3 months postoperatively is recommended. During albendazole treatment, liver function tests and leukocyte counts should be carried out monthly, and albendazole should be used in 2-week intervals after 1 month of use. The preoperative administration of albendazole can reduce the viability of cysts, lower intracystic pressure, and soften cysts, facilitating their evacuation and aspiration.

The surgical procedures for hydatid disease are conservative, radical, and laparoscopic surgery. Conservative procedures are easier, safer, and take less time, but have high rates of postoperative morbidity and recurrence. Radical procedures are associated with low recurrence rates, but high intraoperative risk for a benign disease.

Surgical treatment of uncomplicated hydatid disease

The main purposes of surgical therapy are the complete removal of parasites, prevention of spillage, and preservation of healthy liver tissue. Most surgeons in endemic areas prefer conservative procedures. The cyst is exposed safely. The pericystic area and operating field are covered with pads soaked with scolicidal agent to prevent the spillage of parasites into the surrounding tissue and peritoneal cavity. The cyst is punctured and aspirated. The aspirated fluid in uncomplicated cysts is clear and colorless and is called rock water. Before instilling the scolicidal agent, as much fluid as possible is aspirated to prevent dilution of the scolicidal agent. Then, the scolicidal agent is instilled into the cyst cavity and left for approximately 5 to 15 minutes. The most widely used scolicidal agents are hypertonic saline (3%–30%), ethyl alcohol (70%–95%), chlorhexidine, cetrimide (0.5%), silver nitrate (0.5%), povidone iodine (10%), and hydrogen peroxide (3%). Formaldehyde solution was once used for irrigation as a scolicidal agent, but it is no longer used because it causes serious complications, such as sclerosing cholangitis and acute pancreatitis. Most authors prefer hypertonic saline because the risk of complications is lower than with other agents. (Aspiration of cyst fluid containing bile implies a communication between the bile duct and cyst, and a scolicidal agent should not be instilled because it can cause sclerosing cholangitis.) Then, the scolicidal agent is aspirated, and the cyst is unroofed. The cyst contents, such as the germinative membrane and daughter cysts, are evacuated. At this point, the cavity should be explored carefully for any gross communication with the biliary tract and for the presence of exogenous cysts embedded in the wall of the cyst. If there is no evidence of biliary system communication, a clear sponge is put into the cavity and left for approximately 5 minutes. Bile staining on the sponge implies communication between the bile duct and cyst. Biliary communications with visible fistulas are sutured intraoperatively with nonabsorbable sutures.

The next step in conservative treatment is managing the residual cavity. This can be done using various methods such as external drainage, marsupialization, internal drainage, capitonnage, introflexion, and omentoplasty.

Conservative surgery is easy, safe, and rapid, but has high morbidity (6%∼47%) and recurrence (4%∼25%) rates,[15–18] The most common complications are bile leakage and fistulas, cavity infection and abscesses, intraperitoneal spillage, vascular injury and bleeding, sepsis, cholangitis, and anaphylactic shock.

Radical procedures include total or partial pericystectomy and hepatic resections. The cysts are removed completely in radical surgery. A pericystectomy can be performed using an open or closed cyst method. In the open method, the cyst is sterilized with a scolicidal agent, the contents of the cyst are evacuated, and the pericyst is removed. This method is preferred when the cyst wall is thin and there is a risk of rupture or encountering a major vascular structure with the closed cyst method. In the closed cyst method, an *en bloc* pericystectomy is performed. During the procedure, the afferent blood vessels and biliary ducts are ligated between the pericyst and normal liver parenchyma to prevent hemorrhage and postoperative bile leakage. Exogenous daughter cysts adjacent to the main cyst can be identified with the closed cyst method, and no scolicidal agent is needed. However, bleeding can occur from the adjacent vessels because some pericyst tissue adheres strongly to the main vessels. A partial pericystectomy that leaves part of the cyst wall behind might be preferred when the pericyst adheres strongly to major vessels.

Radical procedures have low recurrence (0%∼3%) and complication (0%∼26%) rates but have high intraoperative risk.[10,13,19] These procedures should be performed by experienced hepatobiliary surgeons in hepatic surgery centers.

Surgical treatment of complicated hydatid disease

Complications of liver hydatid disease are secondary infection; obstructive jaundice due to pressure or rupture into the biliary tree, peritoneum, or an adjacent structure; and anaphylaxis.

Rupture into the biliary tree

Intrabiliary rupture is the most common complication of hepatic hydatid disease and is encountered in approximately 10% to 25% of patients.[20,21] The early diagnosis and treatment of cystobiliary communications are mandatory because they are associated with increased postoperative morbidity.

Cystobiliary communications can occur with frank (major, $\geq$5 mm) or occult (minor, $<$5 mm) ruptures. Frank rupture is seen in 3% to 17% of cases.[22,23] The clinical symptoms of cystobiliary communications are obstructive jaundice, abdominal pain, fever, and nausea/vomiting.[21,23] The spill of daughter cysts and fragmented membranes into the biliary tree can lead to obstructive jaundice, cholangitis, and septicemia. Occult rupture is seen in approximately 10% to 37% of cases.[22] An occult cystobiliary communication is usually asymptomatic. It usually appears as a bile leak or fistula postoperatively. Increased bilirubin and alkaline phosphatase (ALP) levels and a cyst diameter $>$10.5 cm suggest an occult cystobiliary communication.[20,22]

US, CT, MRI, and ERCP can be used to diagnose intrabiliary rupture. The US findings of intrabiliary rupture include irregular linear echogenic structures without acoustic shadowing in the bile duct and a dilated biliary system. ERCP can also be used to clean the common bile duct and explore the duct nonoperatively. Furthermore, surgical treatment is not necessary in some cases. However, ERCP is an invasive, expensive procedure and should be performed selectively.

The treatment principles of cystobiliary communications are evacuating the cyst contents, managing the cavity, and restoring bile drainage. Some authors favor radical surgery for treatment, whereas others favor conservative procedures. Common bile duct exploration should be performed following T-tube drainage or choledocoduodenostomy if ERCP is not available. T-tube drainage has the advantages of not damaging the sphincter of Oddi or disrupting the normal anatomy and physiology; in addition, cholangiography and endoscopic removal of any residual debris in the common bile duct can be performed postoperatively.[24] Choledocoduodenostomy is preferred in elderly and high-risk patients, when a dilated common bile duct allows a wide anastomosis, and if there is suspected residual debris in the bile tree.[24]

Treatment of peritoneal rupture

The reported incidence of peritoneal perforation is approximately 10% to 16%, and this can lead to an

anaphylactic reaction in approximately 1% to 12% of patients with intraperitoneal perforation.[3,25,26] Intraperitoneal perforation can occur during surgery or percutaneous treatment. Trauma is another cause of intraperitoneal perforation. The symptoms of intraperitoneal rupture are abdominal pain, nausea, vomiting, allergic reactions, and anaphylaxis. US and CT are the main diagnostic methods, and they have high sensitivity. The treatment of intraperitoneal perforation is controversial. Radical or conservative approaches can be used. After intervention for a perforated cyst, the peritoneal cavity is irrigated with sufficient scolicidal agent, and all of the cyst contents are removed. Medical treatment with albendazole 10 mg/kg daily for 3 months is recommended postoperatively to prevent recurrence. The morbidity (12%∼63%) and mortality (0%∼12%) are high after intraperitoneal perforation.[25,27]

Treatment of postoperative complications

External biliary fistulas and bile leakage are the most common postoperative complications in patients with cystobiliary communication. If bile leakage continues for more than 10 days, it is defined as biliary fistula. The reported rates of bile leakage and biliary fistula are approximately 2.5% to 28.6% and 1% to 10%, respectively.[27,28] The main reason for fistula development is cystobiliary communication. The intracystic pressure (30–80 mmH_2O) is higher than the biliary system pressure (15–20 mmH_2O).[29] Therefore, bile might not be seen in the cyst fluid during surgery. The pressure gradient is reversed after the cyst is evacuated and bile leakage and a fistula develop. Bile-stained cyst fluid, large cysts, cysts located in the liver hilum, and elevated liver function tests are risk factors for the development of a postoperative biliary fistula. Some bile leakage and fistulas can close spontaneously, but most persist and require endoscopic or surgical intervention. The aim of fistula treatment is to decompress the biliary system. Endoscopic treatment with sphincterotomy, stent insertion, or nasobiliary tube drainage is effective and safe and results in a shorter fistula closure time and hospital stay. There are several suggestions regarding endoscopic intervention and the choice of treatment. Fistulas draining $\leq$100 mL per day can be treated conservatively.[28] Endoscopic sphincterotomy is the first line of treatment for low-output fistulas. Stent insertion and nasobiliary tube drainage are usually suggested for high output and persistent fistulas.[27,30–33] Stent insertion is better tolerated and avoids bile loss but requires further intervention for exchange and removal. Nasobiliary drainage allows cholangiography for control of the fistula and biliary tree but prolongs the hospital stay and causes patient discomfort.

Cavity-related complications can occur postoperatively. Fever and purulent drainage are signs of cavity infection and abscess. They should be treated with antibiotics and percutaneous drainage. Surgical intervention is required when conservative and percutaneous treatments fail.

The best treatment choice for hydatid disease is still controversial. Conservative surgical treatment is safer and easy to perform but has high recurrence and complication rates. Radical surgical treatment has high intraoperative risk, but low recurrence and complication rates. Radical surgical interventions should be performed in high-volume hepatobiliary centers. Cystobiliary communications are the most important complication of hydatid disease, and further endoscopic and surgical intervention might be required.

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