SAJS
Review

Hydatid disease of the liver

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Summary

Echinococcus granulosus remains a clinical problem in sheep and subsistence farming communities in South Africa. The most commonly affected organs are the liver and the lung. Most cysts remain clinically silent and are diagnosed incidentally or when complications occur. Clinical examination is unreliable in making the diagnosis. Serological testing has a broad range of sensitivity and specificity and is dependent on the purity of the antigens utilised. Ultrasound examination of the abdomen is both sensitive and cost effective. Computed tomography and endoscopic retrograde cholangiopancreatography (ERCP) are reserved for complicated cases. The differential diagnosis includes any cystic lesion of the liver.

Liver hydatid cysts can be treated by medical or minimally invasive (laparoscopic and percutaneous) means or by conventional open surgery. The most effective chemotherapeutic agents against the parasite are the benzimidazole carbamates, albendazole and mebendazole. Albendazole is more efficacious, but recommended treatment regimens differ widely in terms of timing, length of treatment and dose. Medical treatment alone is not an effective and durable treatment option. PAIR (**p**uncture, **a**spiration, **i**njection, **r**easpiration) is the newest and most widely practised minimally invasive technique with encouraging results, but it requires considerable expertise. Open surgery remains the most accessible and widely practised method of treatment in South Africa. The options are either radical (pericystectomy and hepatic resection) or conservative (deroofing and management of the residual cavity). Various scolicidal agents are used intraoperatively (Eusol, hypertonic saline and others), although none have been tested in a formal randomised controlled trial. Laparoscopic surgery trials are small and unconvincing at present and should be limited to centres with expertise. Complicated cysts (intrabiliary rupture and secondary infection) may require ERCP to obtain biliary clearance before surgery, and referral to a specialist centre may be indicated.

Hydatid disease (echinococcosis) is a parasitic disease that remains a clinical problem worldwide, especially in areas where animal husbandry and subsistence farming form an integral part of community life. The liver is the most commonly involved organ (52 - 77%),[1] but hydatid disease may affect any part of the body either as a primary or secondary event.

Aetiology and life cycle

There are four forms of hydatid disease. *Echinococcus granulosus* (EG) is the most common and gives rise to cystic hydatid disease (CHD), which is the focus of this review. *Echinococcus multilocularis* is uncommon and causes alveolar hydatid disease (AHD), which is far more aggressive and frequently mimics malignancy.[2] The rarest clinical form is *Echinococcus vogeli* or polycystic hydatid disease (PHD), with characteristics between CHD and AHD.[3] Recently a new strain, *Echinococcus shiquicus,* has been identified on the Tibetan plateau but to date no human infection has been described.[4] CHD is a zoonosis infecting a variety of domestic and wild animals. There is no host specificity for the larval stage of EG, but the commonest intermediate hosts are sheep, cattle, buffalo, camels and pigs.[5] The definitive hosts are canids (dogs, jackals, hyenas and foxes in Africa).

Echinococcus granulosus is a small, hermaphroditic tapeworm about 3 - 5 mm in length. The tapeworm comprises 3 - 4 segments and lives in the upper small intestine of the definitive canine host. The eggs produced by the mature tapeworm contain an embryo that has 3 pairs of lancet-shaped hooklets. The contaminated faeces are ingested by the inter-

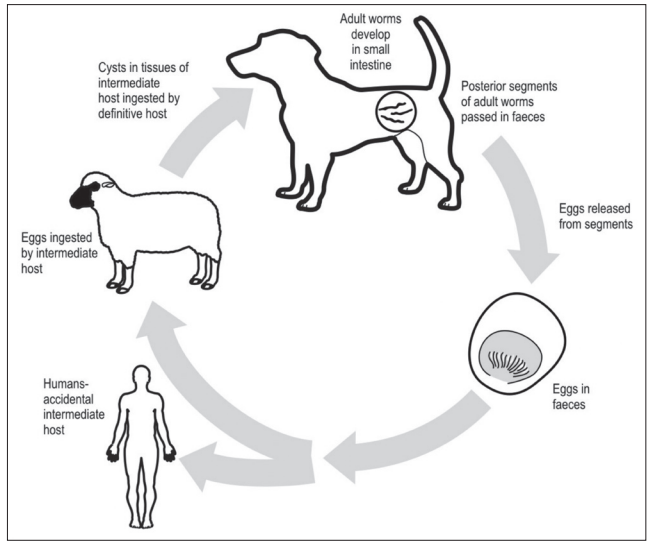

Fig. 1. Life cycle of *Echinococcus granulosus*.

mediate host (humans are accidental intermediate hosts), in which the eggs hatch and the embryo migrates through the intestinal wall into the portal system. Most embryos lodge in the liver, mainly the right lobe due to preferential portal flow, where they either die or develop into hydatid cysts within months to years.[5] Approximately two-thirds of patients develop liver cysts. The embryos may escape this first filter and lodge in the capillaries of the lung where they develop. A small percentage of embryos may find their way into the systemic circulation, where they may involve brain, bone or any other site. Most patients (approximately 80%) have single-organ involvement.[3] The life cycle is complete when the definitive host ingests infected offal containing hydatid cysts (Fig. 1).

Pathology

The developing hydatid cyst has three layers. The outer pericyst is composed of host fibroblasts, eosinophils, giant cells and modified hepatocytes. The middle laminated membrane is acellular and impermeable to bacteria, and the innermost layer, the germinal layer or brood capsule, is translucent and is the origin of scolices and daughter cysts within the primary cyst.[2] The cyst usually contains crystal-clear fluid which is strongly antigenic and may cause anaphylaxis if released into the circulation of the host.

Most cysts remain silent when small and present only when complications such as rupture into the biliary tree, bacterial superinfection or free intra-abdominal rupture occur. Owing to the lack of symptoms in the early stages, the actual accurate assessment of the growth rate of these cysts is difficult.

Classification of cysts

Several classifications of CHD exist. All were developed in endemic areas, and are important because they enable the most appropriate treatment option to be selected. The classifications are not comparable, however, which makes comparative analysis difficult. The two most widely used classifications are the morphological classifications proposed by Gharbi *et al.*[6] in 1981 (Table I) and Lewall and McCorkell[7] in 1985, which are based on pathology and natural history. In 1997, the WHO Informal Working Group classification on Echinococcosis (WHO-IWGE) proposed a new standardised classification based on ultrasound images.[8] This classification is intended to follow the natural history of CHD and is divided into three groups. The first group are active, fertile cysts containing viable scolices, the second group are in a transitional stage owing to compromise either by host defence or chemotherapy, and the third group are inactive, having lost their fertility, and are degenerative.

Clinical features

CHD is frequently silent and only diagnosed incidentally during abdominal investigation for other pathology. The most common symptom, when it occurs, is right upper quadrant or epigastric pain and the most common findings on examination are an enlarged liver and a palpable mass.[1] Patients may also present with complications of the cyst such as biliary communication (major or minor), intraperitoneal rupture (spontaneous or post-traumatic) and, rarely, intrathoracic or intrapericardial rupture. Cyst rupture can be associated with anaphylaxis secondary to the highly antigenic content of the cyst fluid or may be silent and present with multiple intraperitoneal cysts.

TABLE I. GHARBI CLASSIFICATION OF HYDATID CYSTS

Type	Description
I	Pure fluid collection
II	Fluid collection with a detached membrane
III	Fluid collection with multiple septa and/or daughter cysts
IV	Hyperechoic with high internal echoes
V	Cyst with reflecting calcified thick wall

Diagnosis

There are several modalities that can confirm the diagnosis of CHD, as an adjunct to careful history (exposure) and examination.

Serology and immunological tests

Full blood count may reveal eosinophilia in the presence of cyst leakage, or may be normal. Tests to determine cellular immunity, such as the Casoni intradermal test, have largely been abandoned owing to low sensitivity.

Tests of humoral immunity are still widely used to confirm the diagnosis. The sensitivity and specificity of any humoral test depends largely on the quality of the antigens utilised. Antigens can be derived from whole parasites or organelles, or soluble antigens from cyst fluid. Indirect immunofluorescence assay (IFA) is the most sensitive test (95%) in patients with hepatic CHD.

The sensitivity and specificity of enzyme-linked immunosorbent assay (ELISA) is highly dependent on the method of antigen preparation, and cross-reactions with other helminthic diseases occur if crude antigens are used. Purified fractions may yield high sensitivities (95%) and specificity (100%).[9]

Imaging

Imaging modalities range from simple to complex and invasive. Plain radiographs of the abdomen and chest may reveal a thin rim of calcification delineating a cyst, or an elevated hemidiaphragm. Both signs are nonspecific.

Ultrasound is readily available and cost effective. A cyst containing daughter cysts and hydatid sand (debris) are highly suggestive. Several studies have documented the excellent sensitivity (100%) of ultrasound.[10,11]

A computed tomography (CT) scan of the abdomen gives better information concerning location, accessibility and possible complications. It is also helpful in identifying exogenous cysts, and the volume of the cyst can be estimated. CT is an important investigation when there is diagnostic uncertainty on ultrasound, when planning surgical intervention or when recurrent disease is diagnosed (Fig. 2). Magnetic resonance imaging (MRI) adds little to CT scanning and is not cost effective.

Endoscopic retrograde cholangiopancreatography (ERCP) remains an important tool in cases where rupture into the biliary tree has occurred, allowing both the diagnosis of major biliary communication and clearance of the common bile duct (CBD) prior to surgery or intervention (Fig. 3).

Differential diagnosis

This includes all cystic lesions of the liver, and is encompassed in the WHO-IWGE classification category of CL (cystic lesion). Congenital cysts, either single or multiple, and neoplasms, both primary and secondary, must be considered.

Management

Most liver hydatid cysts require treatment because of the risk of complications as they grow. The type of intervention is determined by the nature and location of the cyst and the surgical fitness of the patient. Small densely calcified cysts, presumed to be dead, require no further intervention and should be monitored. Viable daughter cysts may, however, persist in the calcified shell, and excision should still be considered in good-risk surgical candidates. Small cysts (< 4 cm) located deep within the liver parenchyma, if uncomplicated, can be managed conservatively, while those sited peripherally in a fit patient are best managed operatively.[1] PAIR (**p**uncture, **a**spiration, **i**njection, **r**easpiration) is an effective alternative in the management of suitable cysts.

Chemotherapy

Chemotherapy forms an integral part of the management of CHD. The most widely used agents with anti-echinococcal activity are the benzimidazole carbamates mebendazole (MBZ) and albendazole (ABZ). Albendazole is more effective *in vitro* than mebendazole, and has improved gastrointestinal absorption and bioavailability.[12,13] Both drugs interfere with glucose absorption through the wall of the parasite, leading to glycogen depletion.

Chemotherapy alone

This should be considered only in patients in whom there is a contraindication to surgery or PAIR. Patient refusal, poor surgical risk and multiple recurrences following intervention are possible indications.

In a prospective, randomised control trial assessing parasite viability in patients receiving medical therapy, Gil-Grande *et al.*[14] showed protoscolex viability to be significantly lower in patients receiving medical therapy than in those receiving no therapy. Furthermore, the data showed that ultrasound response (increased echogenicity) correlates well with cyst non-viability (Table II).

WHO guidelines for primary chemotherapy are ABZ 10 - 15 mg/kg/d in divided doses for a minimum of 3 months, with 10 - 14-day intervals between cycles, dependent on clinical and radiological improvement. Improvement is defined as reduction in cyst size (> 25%), membrane separation from the pericyst, or calcification. To date, the available data are unclear whether daily dosage, total dosage or treatment duration is the most important factor.[15] Studies show ABZ to be more efficacious than MBZ (82% v. 56%). About 25% of cysts regenerate once medical therapy is stopped.[16] Praziquantel has been tested in a few clinical studies as an efficacious agent in treating echinococcosis in humans. A small study has suggested that combination therapy with ABZ[17] or MBZ may be more efficacious than either agent alone.

Side-effects from chemotherapy are important. Between 10% and 20% of patients may develop transient elevation in transaminases, usually reversible on stopping therapy. Idiopathic marrow suppression is a major side-effect and both ABZ and MBZ are contraindicated in pregnancy. The available evidence indicates that chemotherapy alone is not the ideal treatment strategy for CHD of the liver, with high failure rates and recurrence rates on stopping treatment.[18]

Preoperative chemotherapy

The aim of preoperative therapy is to sterilise the cyst contents, thereby diminishing the chances of secondary implantation by accidental spillage during surgery and local

TABLE II. CHEMOTHERAPY: ABZ v. MBZ OR PLACEBO (RANDOMISED CONTROLLED TRIALS)

Study	Regimen	Regression/improvement[a] or non-viable parasite[b]	*p*-value	Failure	Surgical removal
Gil-Grande,[14] 1993 (*N* = 55)	**Placebo (*N* = 18)**	**50%[b]**	**–**	**–**	**–**
	ABZ 10 mg/kg/d for 1/12 (*N* = 18)	**72%[b]**	**0.039**	**–**	**–**
	ABZ 10 mg/kg/d for 3/12 (*N* = 19)	**94%[b]**	**0.018**	**–**	**–**
***Teggi,[13] 1993 (*N* = 337)**	**MBZ 50 mg/kg/d 3 - 12/12 (*N* = 121)**	**50.5%[a]**	**< 0.001**	**48.1%**	**30**
	ABZ† 12 mg/kg/d 3/12 (*N* = 216)	**77.9%[a]**		**31.1%**	**18**
***Franchi,[16] 1999 (*N* = 448)**	**MBZ 50 mg/kg/d continuously for 3 - 6/12 (*N* = 125)**	**46.6%[a]**	**< 0.001**	**24.3%**	**–**
	ABZ‡10 - 12 mg/kg/d continuously for 3 - 6/12 (*N* = 323)	**82.1%[a]**		**26.7%**	**–**
Keshmiri,[40] 2001 (*N* = 29)	**Placebo (*N* = 7)**	**14%[a]**	**< 0.001**	**–**	**–**
	ABZ 400 mg BD, 3 cycles of 6/52 (*N* = 22)	**82%[a]**		**–**	**–**

*Both studies included cysts other than liver hydatids, but responses analysed individually for each organ.
† 90 patients after 1989 not randomised with evidence that ABZ more effective than MBZ.
‡ Patients enrolled from 1980 received ABZ alone.
ABZ = albendazole; MBZ = mebendazole.

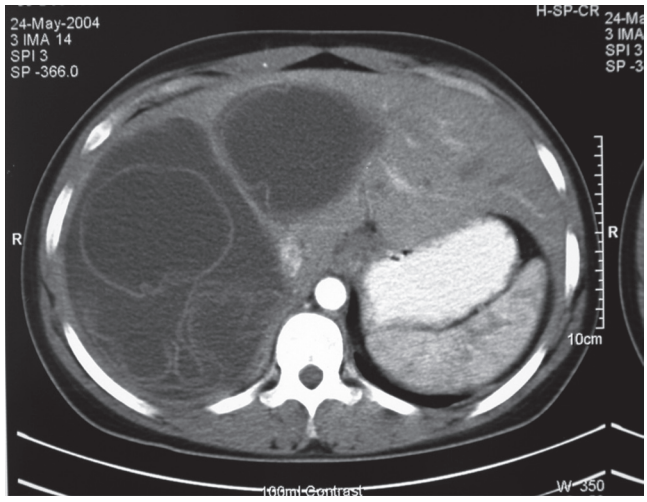

Fig. 2. CT scan showing typical type III cyst in right lobe of liver.

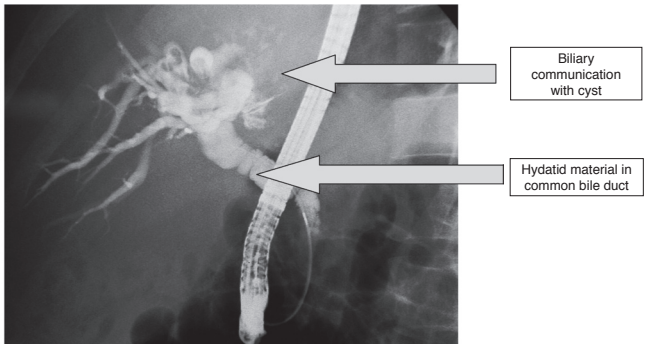

Fig. 3. ERCP showing biliary communication with cyst and hydatid material in common bile duct.

recurrence.[19] There is uncertainty concerning optimal dosing regimens in preoperative chemotherapy. The problem arises in trying to do comparative analyses between studies where dosage regimens differ, as well as methods to evaluate efficacy.

In a recent non-consecutive series, Manterola *et al.*[20] evaluated short-course ABZ (10 mg/kg/d 4 days preoperatively) and found 61% of cysts still viable at surgery. They concluded that short-course ABZ is ineffective as a preoperative manoeuvre.

Longer preoperative treatment with ABZ, however, seems to be beneficial. Aktan and Yalin[21] in a prospective, non-randomised series showed statistical significance in the number of non-viable cysts achieved by giving ABZ for 3 weeks before surgery, using intracystic pressure as an indicator of cyst viability. Intracystic pressure has previously been shown to be a reliable indicator of cyst viability.[22] Similar studies show the trend that 3 - 4 weeks' preoperative treatment significantly decreases the viability of cysts.[23]

At present, postoperative ABZ is recommended if there is spillage of cyst content at the time of surgery, partial cyst removal or biliary rupture. Available evidence suggests that treatment should be commenced 3 - 4 weeks before surgery to be maximally effective, but current WHO guidelines advise commencement of treatment at least 4 days before surgery and continuation for 1 month postoperatively if ABZ is used, or 3 months if MBZ is used.[3,24] The available randomised controlled trials with respect to chemotherapy are shown in Table II.

Chemotherapy and PAIR

In a prospective study by Khuroo *et al.*[25] percutaneous drainage combined with ABZ was an effective and reasonably safe method of dealing with CHD of the liver. There is no consensus regarding the duration of chemotherapy. Chemotherapy is given to provide a 'safety net' to decrease the risk of secondary implantation should spillage occur, to reduce cyst size prior to the procedure, and to ensure a high concentration of ABZ in the bile should biliary spill occur during the procedure.

Surgical management of CHD

Open surgery

The principles of open surgery are to eradicate the macroscopic parasite, prevent intraoperative spillage, and obliterate the residual cavity if resection is not performed. Currently surgery remains the cornerstone treatment for CHD of the liver in fit patients.[18] The surgical procedures are classified as either radical (pericystectomy or hepatic resection) or conservative (deroofing of the cyst and management of the residual cavity).

Pericystectomy involves removal of the cyst together with a rim of host liver in a non-anatomical plane. This is a major procedure accompanied by significant blood loss unless portal inflow occlusion is used, and is not widely practised in South Africa. Liver resection is appealing in that the cyst is removed intact, without risk of spillage, but many regard this as aggressive surgery for benign disease. A case can, however, be made for resection when cysts occupy the left lateral segments (2 and 3) and communicate with major bile ducts. This is technically and physiologically a lesser procedure than a right-sided liver resection.

The most commonly utilised surgical approach is simple cystectomy. The cyst should abut the surface of the liver for this approach to be utilised. Following exposure of the liver by laparotomy, the most prominent portion of the cyst is identified and packed off from the rest of the abdominal cavity with abdominal swabs. The most superficial swabs are soaked in a scolicidal solution, which prevents accidental spillage of viable scolices into the abdominal cavity. The cyst is then punctured with a 16-gauge needle attached to a closed aspiration/injection system. The cyst is aspirated until no longer tense. Once no further fluid can be aspirated, a scolicidal agent is injected into the cyst and left for 1 - 2 minutes. This procedure is repeated 2 - 3 times. The cyst is then finally aspirated, and a larger incision is made at the site of initial puncture to allow a large-bore suction to be introduced. The remaining cyst fluid and more solid contents are aspirated (smaller daughter cysts and membranes). The cyst is finally deroofed, and remaining daughter cysts and germinal membranes are removed using ovum forceps (Figs 4 and 5). The cavity is carefully inspected for evidence of bile leaks, which are identified by bile staining of the cyst fluid. A useful technique is to place a dry abdominal swab in the cavity and leave it for a few minutes to identify smaller leaks and their location. Any bile leaks are oversewn as well as the edge of the deroofed cyst to prevent bleeding.

The management of the residual cavity remains controversial. A variety of procedures including omentopexy (packing the residual cavity with omentum), simple drainage (internal or external), capitonnage, introflexion and capsulorrhaphy have been described. The complication rate in simple cystectomy ranges from 6% to 47%[26] and includes bile leaks, cavity infections/abscesses and wound infection. There is level II evidence to suggest that omentopexy is efficient in preventing deep abscess formation in both radical and conservative surgery.[27] Table III lists available randomised controlled trials dealing with the residual cavity.

There are no prospective randomised controlled trials comparing radical and conservative surgery. Conservative procedures are easier to perform, and based on current levels of evidence available it is not possible to conclude which treatment is better. More prospective randomised controlled trials are required to provide higher levels of evidence.

Scolicidal agents

There is no ideal protoscolicidal agent that is both safe and effective. Many factors influence scolicidal efficacy, including *in vitro* instability of the substance (e.g. sodium hypochlorite) and unpredictable dilution of the agent within the hydatid fluid. Possible cystobiliary communication may cause chemical cholangitis and subsequent sclerosing cholangitis, and therefore formalin is no longer used as a scolicide. Other effective agents that have been used are 70 - 95% ethanol, 15 - 20% hypertonic saline and 0.5% cetrimide (which is a very effective agent but causes methaemaglobinaemia[28]), 1% povidone iodine and 0.5% silver nitrite. There are no randomised controlled trials comparing various scolicidal agents and their complications. There are, however, numerous reports of the side-effects of all the agents used, including fatal hypernatraemia following the use of hypertonic saline.[29] Most recently, chlorhexidine gluconate in low concentrations (0.04%) has been shown to be an effective scolicidal agent, especially for intraperitoneal spillage.[30] This study has yet to be confirmed in humans.

Laparoscopic surgery

The theoretically appealing advantages of laparoscopic surgery apply to CHD of the liver as well. Shorter hospital stay, lower incidence of wound sepsis, and therefore more cost-effective management are cited. None of the studies available to date are comparative or randomised and there is no universally accepted standard technique.[31]

The laparoscopic approach is reported to be safe and effective, but should be limited to expert laparoscopic surgeons until comparative studies are available.

PAIR

Fear of anaphylactic shock and secondary echinococcosis have previously discouraged the intentional puncturing of hydatid cysts. However, accidental punctures inevitably occurred during investigation of cystic liver lesions with no untoward effects. In 1983, Ben Amor reported the successful management of hydatid cysts in sheep livers using percutaneous puncture and injection of hypertonic saline in combination with benzimidazole derivatives, and subsequent successful treatment of 2 patients in whom the surgical risk was high.[32] Numerous reports followed well as articles on successful treatment of hydatid liver cysts. Khuroo *et al.*[25] published the first randomised controlled trial in 1993 and concluded that percutaneous drainage was effective and safe in the management of CHD (Table IV). Subsequent studies of large numbers show that the risk of anaphylaxis is negligible and that regrowth does not tend to occur.[33]

PAIR is a valuable alternative to surgery in terms of cost-containment and mean hospitalisation time. The technique varies between institutions and is best suited to Gharbi type I - III cysts. Pretreatment with ABZ is started 10 days before the procedure and is continued for 1 - 2 months, depending on the ultrasonographic features of the cyst after the procedure.[3,31]

Technique of PAIR

The technique involves puncturing the cyst under direct visualisation with ultrasound and local anaesthesia, using a transhepatic approach. Fluid is aspirated and analysed for bilirubin, identifying a possible biliary communication, and viable protoscolices (centrifuged specimen stained with neutral eosin, showing motile scolices). If viability is confirmed, as much fluid as possible is aspirated, and any other daughter cysts are individually punctured. A third of the volume of fluid aspirated is reinjected with the scolicidal agent of choice (typically 95% ethanol, which has been shown to be highly effective[34]). The scolicide usually causes germinal membrane separation from the pericyst. The fluid is then reaspirated after 15 minutes and re-examined for viable protoscolices. The procedure is then repeated until no viability is demonstrated or there is total separation of the germinal layer from the pericyst that is confirmed on ultrasound. In large cysts (> 6 cm) some prefer prefer to leave a catheter in the cyst cavity, which allows cyst drainage and identification of bile leaks. Once drainage stops and no bile leak is present, ethanol can be injected to sclerose the residual cavity, thereby theoretically preventing the complications associated with a large residual cavity.

Follow-up includes both serology (ELISA) and ultrasound features. The frequency of follow-up is also centre-dependent. Meta-analysis demonstrates a cure rate of 95.8%, a major complication rate of 7.9% compared 25.1% with surgery, and a recurrence rate of only 1.6%.[35]

PAIR and surgery

The first randomised controlled trial comparing PAIR and surgery by Khuroo *et al.*[31] showed a statistically significant reduction in hospital stay and complication rate in patients undergoing PAIR (Table V). In a subsequent meta-analysis[36] the outcome of 769 patients with hydatid cysts treated with PAIR and ABZ/MBZ were compared with 952 historical controls undergoing surgery, and it was concluded that PAIR + ABZ/MBZ is associated with higher parasitological and clinical efficacy, lower morbidity and mortality, lower disease recurrence and

TABLE III. SURGICAL RANDOMISED CONTROLLED TRIALS: DRAINAGE AND OMENTOPLASTY

Study	Procedure	Morbidity/sepsis	*p*-value	Hospital stay (days)	*p*-value	Recurrence
Kama,[38] 1998 (*N* = 59)	**Drainage (*N* = 29)**	**44.7%**		**18.6**		**–**
	No drainage (*N* = 30)	**10%**	**< 0.05**	**8.5**	**< 0.05**	**–**
Dziri,[27] 1999 (*N* = 115)	**Omentoplasty (*N* = 58)**	**10%**		**10**		**–**
	No omentoplasty (*N* = 57)	**23%**	**0.05**	**12**	**–**	**–**
Reza Mousav,[39] 2005 (*N* = 65)	**Drainage (*N* = 30)**	**16.6%**		**6.5**		**0 (18 months)**
	Omentoplasty (*N* = 35)	**5.7%**	**< 0.05**	**15.6**	**< 0.05**	**0 (18 months)**

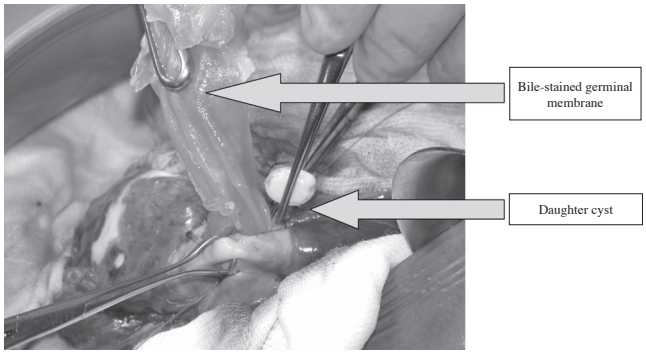

Fig. 4. Germinal membrane and daughter cyst at time of cystectomy.

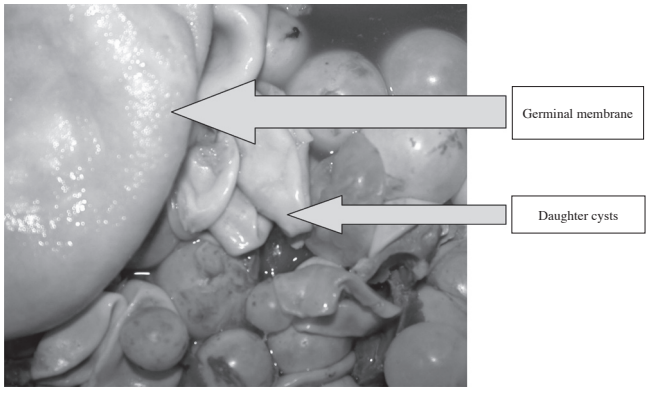

Fig. 5. Germinal membrane and daughter cysts.

shorter hospital stay than surgery. However, despite the claimed safety and efficacy, PAIR is not yet accepted as treatment of choice for uncomplicated hydatid cysts. A Cochrane systematic review concludes that there is insufficient evidence to support or refute PAIR with or without benzimidazole coverage in treating patients with uncomplicated hydatid cysts as being superior to other methods of management.[41]

TABLE IV. PAIR v. ABZ

Study	Treatment arm	Reduced size/ non-viable echo pattern
Khuroo,[25] 1993 (*N* = 33)	**PAIR (*N* = 11)**	**100%**
	PAIR + ABZ 10 mg/kg/d (*N* =11)	**100%**
	ABZ 10 mg/kg/d (*N* = 11)	**18%**

Complicated hydatid cysts

Biliary complications caused by the communication of the hydatid cyst with the biliary system generally only produce symptoms when hydatid material enters the common bile duct, resulting in obstructive jaundice or cholangitis. Less commonly, secondary infection of the cyst may occur. Intrabiliary rupture is usually managed by ERCP with sphincterotomy and clearance of the hydatid material from the CBD, followed by surgical cystectomy and careful closure by direct suturing of the biliocystic fistula with external drainage. If a subsequent bile leak develops, an ERCP and stent may be required.

Recurrence

This is defined as the appearance of new, active cysts after therapy. The lack of prospective trials and differences in treatment regimens and length of follow-up invalidate accurate comparison of recurrence rates. After open surgery recurrence rates of 2.2 - 11.3% have been reported, while after PAIR rates of 0 - 2% have been reported.[37]

New developments

New techniques for the management of hydatid cysts include radiofrequency-assisted cystectomy and pericystectomy[42] and modifications to percutaneous and laparoscopic techniques.

TABLE V. SURGERY v. PAIR

Khuroo,[31] 1997 (*N* = 50)	PAIR + albendazole (*N* = 25)	Surgery (*N* = 25)	*p*-value
Mean hospital stay (days)	**4.2 ± 1.5**	**12.7 ± 6.5**	**< 0.001**
Cyst disappearance	**22 (88%)**	**18 (72%)**	**0.29**
Procedure-related complications	**8 (32%)**	**21 (84%)**	**< 0.001**
Follow-up (months)	**17.5 ± 7.0**	**17.4 ± 6.5**	**0.96**
Serum IgG negativity	**19 (76%)**	**17 (68%)**	**0.74**

The continued advent of new techniques in managing CHD will undoubtedly decrease the chances of future well-constructed prospective randomised control trials on simpler and more accessible management.

Conclusion

Hydatid disease remains a common problem in South Africa. Limitations of expertise and resources dictate that most uncomplicated liver hydatid cysts are managed by conventional surgical means, usually combined with ABZ treatment. Complicated cysts are optimally managed in referral centres with the expertise to perform more complex and minimally invasive procedures (ERCP, PAIR or laparoscopy). Although the initial results of minimally invasive procedures are promising, at present their use is most suited to surgically unfit patients or those with recurrent disease. Well-constructed randomised controlled trials are necessary to assess their true efficacy, durability and precise indication in the management of CHD.

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