

Review

Spinal hydatid disease

M Necmettin Pamir^{*1}, Koray Özduman¹ and İlhan Elmacı¹

¹Department of Neurosurgery, Marmara University Faculty of Medicine, Istanbul, Turkey

Study design: Review article on spinal involvement of hydatid disease.

Objectives: A better understanding of this rare but clinically challenging disease is intended. An overview of the epidemiology, pathogenesis, presentation and diagnosis of spinal hydatid disease is provided. Management problems and frequent pitfalls are discussed as well as current therapeutic options, results and outcome.

Methods: Thirty-seven reports of spinal hydatid disease published between 1964 and 2000 were reviewed.

Results: Most of the reported cases of spinal hydatid disease presented with spinal cord compression syndrome. Due to the relative rarity of the problem the diagnosis was frequently made during surgery. Surgical intervention by decompressive laminectomy was the most frequent first management. Reports of anterior procedures as well as spinal stabilization exist. Intraoperative prophylaxis to reduce spillage as well as pharmacotherapy were usually instituted. Results of surgery were generally reported to be poor. Progressive neurological and mechanical deterioration over the years was the most frequently reported disease course. Anecdotal reports of alternative management strategies exist.

Conclusion: Spinal hydatid disease should be considered in the differential diagnosis of spinal cord compression syndrome in endemic countries and sought after with imaging and serology. Treatment is based on surgical decompression. Despite therapy the disease frequently relapses with progressive destruction of the vertebral column and neurological deterioration. Retention of spinal stability is the major long term concern. Overall outcome is poor with few reports of disease-free long term survival.

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Introduction

Hydatid – Greek for ‘watery cyst’ – disease is a rare but significant parasitic disease in endemic areas. Bone involvement is a rare complication of this disease. According to İşlek et al¹ bone disease was first described by Bidloo in 1708, the first description of spinal hydatid disease was made by Churrier in 1807 and the first surgical intervention was reported by Reydellet in 1819.

Despite the introduction of modern surgical and pharmacological therapy the disease still continues to be difficult to cure and highly prone to recurrence. The infestation persistently erodes the spinal column, eventually leading to its destruction and neurological deterioration. The prognosis is poor, comparable to that of malignancies.²

Pathogenesis

Hydatid disease is caused by the larval stage of the tapeworm *Echinococcus Granulosus*. Far less commonly infestation by *Echinococcus Multilocularis* (alveolar echinococcus) or *Oligoarthrus* may be responsible for the disease.^{3–6}

As is the case with other cestodes, *Echinococcus* has definitive and intermediate hosts. The 3–6 mm long adult worm resides in jejunum of the definitive host: mainly a domestic dog and occasionally other carnivores. The parasite has three proglottids: One immature, one mature and one gravid. The gravid proglottid is shed in the excreta of the dog and ruptures to release 500 to 800 ova which are accidentally ingested by cows, sheep, mice or humans. In the intermediate hosts, after digestion of the chitin, outer layer embryos (oncospheres) develop in the intestine to penetrate the duodenal mucosa and

*Correspondence: MN Pamir, Marmara University Hospital, Department of Neurosurgery, Tophanelioğlu caddesi, No: 13/15 Altunizade, Istanbul, 81190 Turkey

disseminate via the portal circulation. They then either develop into cysts in tissues or are destroyed by the host response. The cysts consist of an outer fibrous layer and a cestode-derived inner germinal membrane containing scolices.³⁻⁸

The liver is the most commonly involved site, followed by the lungs. Bone involvement is rare and in most of these cases involves the spine. Infestation is thought to begin primarily in the center of the vertebral body and subsequently to extend extradurally or paravertebrally. Isolated extradural spinal infestation has been reported.⁹ Secondary extension from paravertebral tissues (costal, mediastinal lesions or cysts involving the Erector Spina muscles) or even from pulmonary cysts are occasionally seen.^{3,10,11}

In 90% of the cases the disease is confined to the bone and the epidural space.^{6,11-16} Intradural extramedullary involvement is found in 9% of the cases.^{12,13,17-19} This can be primary or complicate prior to surgery with dural opening or tear. Occasionally, extension from paravertebrally located cysts is encountered. Intramedullary hydatid disease is described in the literature.²⁰

Pathology

Primary infestation of the spine is thought to occur through the portovertebral shunts and the center of the vertebral body is the first site to be involved.^{1,21} In contrast to extraosseous involvement, larval penetration of the cancellous bone in the vertebrae causes a multivesicular and diffuse infiltration.²¹ Cortical weakening due to compression or avascular necrosis^{22,23} may eventually lead to pathological fractures^{7,13,23} and spread to the pedicles, laminae or the ribs may subsequently occur.^{7,13} Death of the cyst results in dystrophic calcification, which may be radiographically demonstrated. The intervertebral discs are spared in the early stages of the disease,² as is the case in tuberculosis. Finally, the infestation may extend beyond the vertebral body and periosteum with the development of a spherical cyst with space occupying symptomatology (Figure 1). Bone destruction and pus-like fluid may be encountered during operation, leading to the name 'ossifluent abscess'.²⁴

Incidence

Although rare in developed countries, the disease continues to be a significant health problem in endemic areas.^{13,16,25} The disease is especially prevalent in areas where livestock is raised in association with dogs: Mediterranean, eastern European, east African, central Asian and South American countries, Australia and New Zealand. The disease is rare in Western Europe and north America, but reviewing the cases in North America, Rao *et al*²⁵ noted that sporadic cases from California, Utah, Lower Mis-

issippi valley, Alaska and northwest Canada were reported. Sapkas *et al*⁷ stated that the infestation may be seen anywhere in the world due to frequent overseas travel.

In contrast to visceral disease, bone or central nervous system involvement occurs much less frequently. Only 0.5 to 3.1% of patients suffer from bone involvement, half of which occurs in the spine.^{1,12,13,16,17,26}

Spinal hydatidosis is a significant cause of spinal cord compression syndrome in endemic areas with incidences ranging from 1.1 in Australia to as high as 14% in Tunisia.^{1,2,7,12,16,17,21,23-25} Spinal hydatid disease in Turkey is responsible for 3.8% of all spinal cord compression syndromes.¹²

Spinal disease most frequently involves the thoracic segments (46% to 50%). Lumbar (20% to 29%) and sacral (20% to 23%) diseases also occur.^{1,7,10,12-17,21,25,27,28} Cervical spine is the least commonly involved area.

Clinical manifestations

Usually clinical manifestations appear long after the initial infestation as cysts grow slowly over a period of several years hampered by the resistance of the surrounding bone. There are no characteristic signs or symptoms. A long history of back pain and/or signs of spinal cord compression are the usual manifestations. Back pain was present in 85%, radicular pain in 25% to 95% and paraparesis in 25% to 77% of patients.^{12,21,25} Cauda equina syndrome also may be noted.^{2,15} It is noted that a history of trauma was present in up to 20% of the cases.¹ Özek *et al*²¹ reported that rapid onset of neurological dysfunction is due to vascular compromise and associated with a slow and incomplete recovery. Baysefer *et al*²⁵ noted that severe paraparesis is usually secondary to epidural disease and that this is associated with a more favorable prognosis.

Paraplegia due to recurrent disease is reported to be as high as 45%.^{12,23,26}

Rupture of a cyst with spillage of the content may provoke a variety of hypersensitivity reactions such as pruritus, urticaria, edema, dyspnoea, asthma, vomiting, diarrhea, colicky abdominal pain and even anaphylactic shock.^{3,5}

Secondary bacterial infection may occur and further complicates the diagnosis.^{13,21} This should be considered whenever fever and irritability are present²¹ (Figure 2).

Diagnosis

Spinal hydatid disease should be considered in the differential diagnosis of spinal cord compression syndrome in endemic countries. Due to the relative rarity of the entity most diagnoses are made intraoperatively which increases the risk of future recurrences. As the choice of the most suitable surgical approach relies on correct diagnosis and

because special measures are required to prevent intraoperative spillage, preoperative diagnosis is of high importance. The differential diagnosis should include tuberculosis, pyogenic infections, fibrous

dysplasia, enchondroma, metastatic malignancy, multiple myeloma and giant cell tumor.⁷

Serodiagnostic tests may be used for preoperative diagnosis in suspected cases.⁴ Detection of antibody

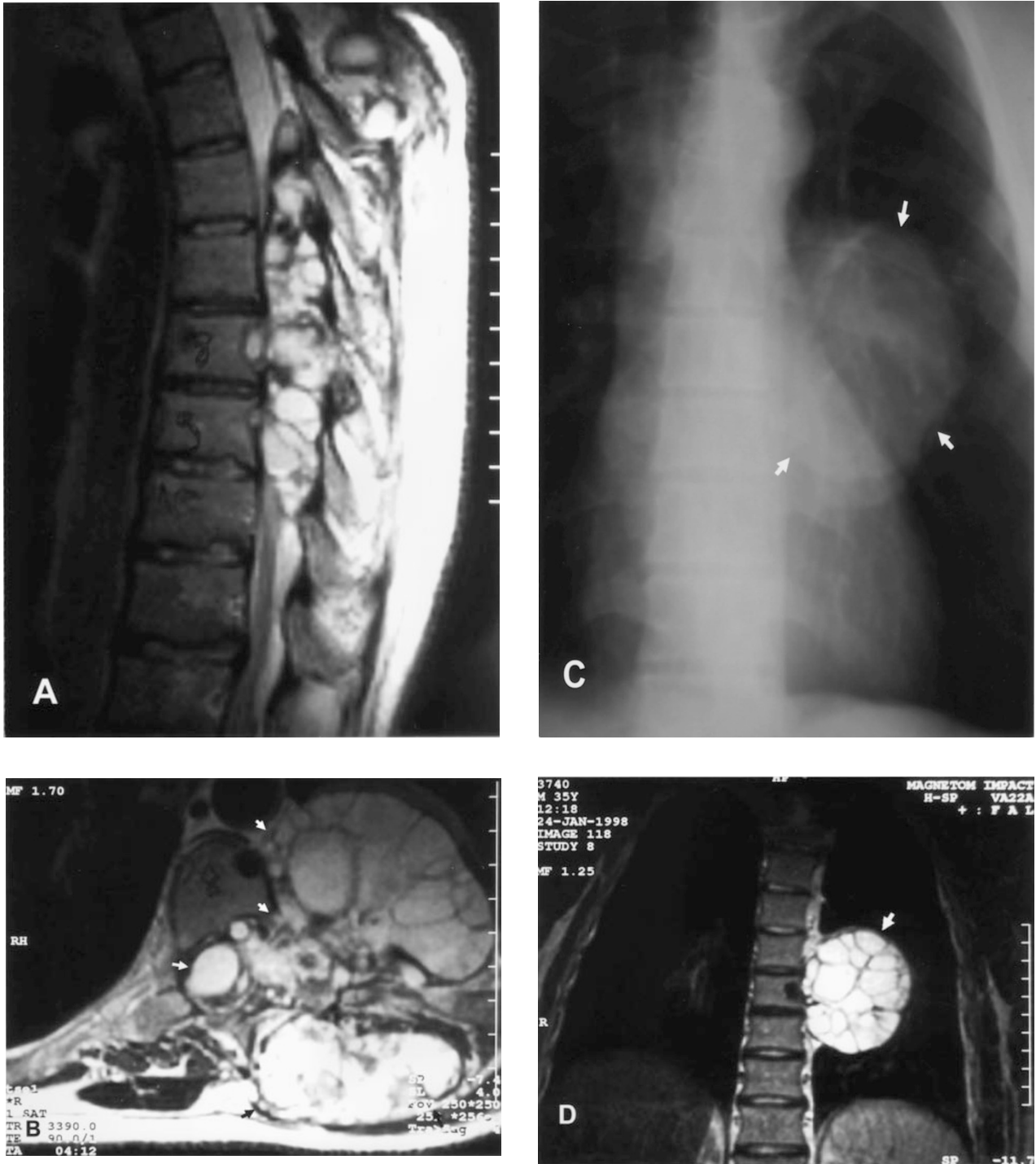


Figure 1 (A–D). A 35-year-old male patient presented to our clinic with recurrence two years after operation for epidurally located spinal hydatid disease. (A, B) T2W sagittal and axial images showing multiloculated cysts extending into the thoracic cavity and subcutaneous tissue (arrows). (C) AP pulmonary plain x-ray showing the posteriorly located mass lateral to the vertebral body (arrows). (D) T2W coronal MR image shows pulmonary extension of the multiloculated cyst. (arrow)

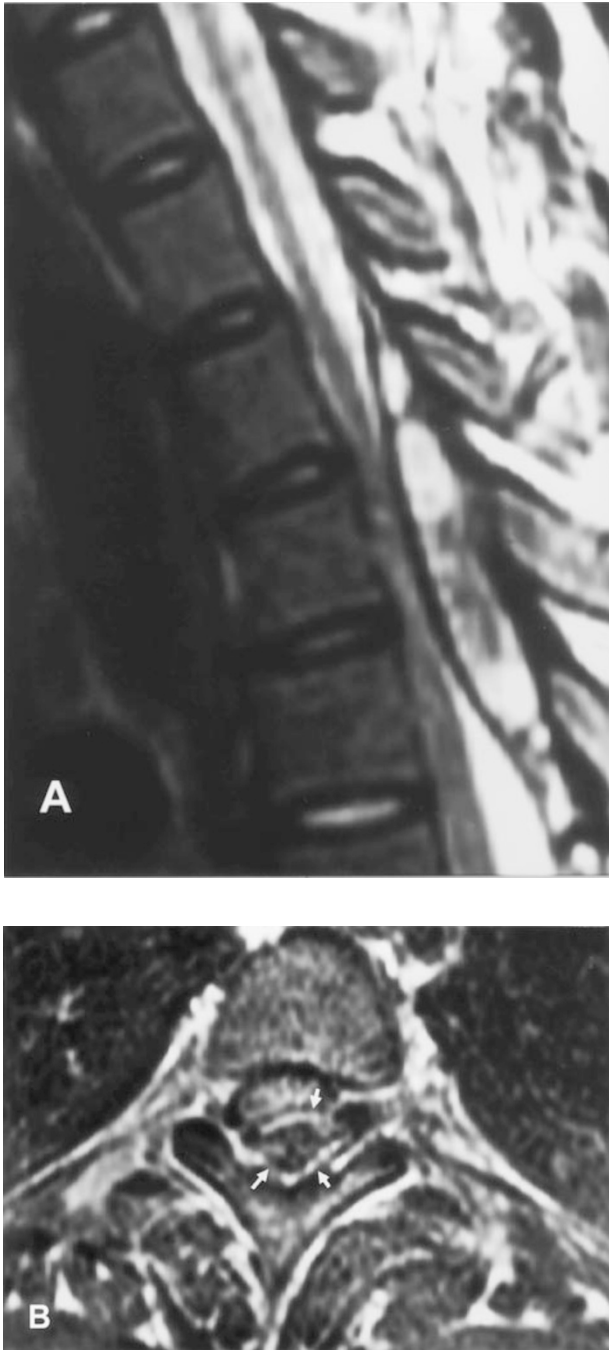


Figure 2 (A, B). A 21-year-old female patient presented to our clinic with motor weakness and paresthesias in the lower extremities and a dermal fistula in the back with purulent discharge. The patient was shown to have secondary pyogenic infection of spinal hydatid cysts. (A): T2W NMR image showing epidural spinal cysts in dorsal spine. (B) Contrast enhanced T1W axial MR image shows epidurally located hydatid cyst (arrows). Note that the spinal cord is displaced anteriorly and laterally

directed to echinococcal polypeptide antigens has the highest degree of specificity.^{4,5} ELISA, indirect hemagglutination and complement fixation tests are

reported to be 80% to 100% sensitive and 88% to 96% specific in abdominal disease.⁵ However, the sensitivity decreases abruptly to 25% to 56% in extrahepatic disease which limits their use in the diagnosis or follow up for primary bone disease.⁵ Second line tests like immunoblotting (Antigen 5 precipitation: Arc-5 Test) may be used for confirmation after ELISA and hemagglutination.⁴ Imaging is more sensitive than serodiagnosis in spinal hydatid disease.⁶ A negative serology should not exclude the diagnosis of hydatid disease in the presence of suggestive imaging results.^{5,6} Diagnostic fine needle aspiration is usually avoided because of the risk of spillage or anaphylaxis due to cyst rupture. Not uncommonly, the final diagnosis is delayed until the surgery and direct visualization of the lesions.^{1,3,7,25}

Other laboratory tests are usually not informative except that eosinophilia may be noted in the peripheral blood.²⁹

Although plain X-rays are not diagnostic of the disease, 'moth eaten' lesions with surrounding sclerosis and calcifications extending into paravertebral soft tissues, when present, are highly suggestive. Bone changes are reported to be present in only 27% of cases.¹² Compression fractures with loss of the corpus height or erosion of the pedicles may be noted.^{7,12}

Myelography, which is of little diagnostic value, carries the risk of cyst puncture with intradural dissemination or anaphylaxis and therefore should be avoided.⁶

CT has excellent bone resolution and usually shows irregular erosions of the cancellous bone devoid of any subperiosteal reaction or enlargement of the vertebra.^{1,12,21,25,27} Tekkök and Benli¹¹ concluded that CT myelography, although inferior to MRI in showing paravertebral soft tissue extension is of great diagnostic value in showing cysts in bone and their intraspinal extension.

The MRI with its superior soft tissue resolution is the most sensitive diagnostic method as well as the method of choice for determination of the extent of the disease.^{6,16,27,30} Berk *et al*³⁰ reviewed MRI characteristics of the lesions and concluded that they had a unique appearance: A sausage-like shape with two dome shaped ends, thin and regular walls and no septation or debris in the lumen. The lesions are occasionally spherical. Signal characteristics of the cyst content are usually similar to that of CSF.^{27,30,31} On T1W images the cyst wall appears iso- or slightly more hypo-intense than the cyst content and enhances slightly after contrast injection. T2W images demonstrate a low intensity rim which correlates to the pathological findings of reactive fibrosis and degeneration surrounding the parasitic membrane. It has also been reported that viability of the cysts could be determined by their MRI characteristics.³⁰ Namely, on T2W images cysts tend to lose their hyperintensity and the walls tend to lose their hypointense signal upon inactivation.^{13,30,31} The authors noted that the differential diagnosis included dorsal arachnoid diverticula,

dorsal intrathoracic and lateral thoracic meningo-coeles.

The CT and MRI provide complementary data both in diagnosis and follow up of patients with spinal echinococcosis.^{21,27,30} Early postoperative MRI is valuable in demonstrating lesions missed during the operation.¹⁸ However, the diagnosis of recurrent disease should not rely solely on imaging but should also be based on symptomatology.⁶

Head, thorax and abdomen should be scanned for extra-spinal cysts upon the diagnosis of hydatid disease.^{6,32}

Treatment

Surgery

Surgery is currently considered the treatment of choice for spinal hydatid disease.^{1,12,21,25} Provided that most of the cases are admitted with signs and symptoms of spinal cord compression syndrome, urgent surgery is validated and most surgeons act accordingly.^{12,26,27}

Preoperative diagnosis of spinal hydatid disease, as well as the determination of its extent, is of crucial importance for planning the operative approach. Posterior or anterior approaches may be considered.

Pure intradurally or pure epidurally located lesions in all spinal levels are approached posteriorly. In these cases surgical excision without intraoperative rupture results in cure.^{12,17,18}

Bone involvement is a major challenge for the treating surgeon. Due to the infiltrative nature of the lesion, spillage of scolices is unavoidable during the surgery for lesions involving the bone.^{1,12,21,25} Even laminectomy may result in rupture of the cysts and spillage.¹ It must be kept in mind that the disease is highly prone to recurrence, and plans for the surgical approach and stabilization must be made accordingly.³⁸

Simple decompression with laminectomy was by far the most commonly reported procedure.^{1,7,10–12,14,15,18,22,25–27,29,33–37} Except for one case,¹² all cases in the literature were first managed by laminectomy. This approach allows simple and easy decompression with little morbidity and mortality. However, access to the vertebral body is limited which may be a problem especially in recurrent disease. Stability of the anterior column must be ascertained to prevent postoperative kyphosis. Some authors advocate a more radical approach with as much bone removal as possible.¹ Radical removal is not shown to preclude disease progression. However, extensive resection with stabilization and grafting is shown to prolong survival in selected cases.³⁸

Several procedures were reported in the cervical region. Posterior approaches including laminectomy, laminectomy with lateral mass plating were reported to be effective with low or no morbidity.^{22,38} Reported anterior approaches were corpectomy with

simple bony fusion and halo bracing and corpectomy with anterior plating.²² In the only reported case of anterior corpectomy with simple bony fusion and external halo bracing²² the authors reported development of postoperative kyphosis with progressive myelopathy. The patient was successfully managed by lateral mass plating.

Operations in thoracic cases were mainly posterior.^{1,7,10,12,14,25,26,29} More extensive lesions with involvement of the vertebral body can be approached via a transpedicular route.²⁶ Three cases of anterior approach to thoracic lesions were reported.^{1,7,12} The first patient had presented with a pathologic compression fracture of the T9 vertebral body. The patient underwent transthoracic corpectomy with anterior fusion.¹² An autologous costal graft was well tolerated by the patient who was asymptomatic at the third year follow-up. The second patient⁷ was first approached posteriorly with laminectomy and transpedicular curettage. However, cysts were noted to extend into the thoracic cavity via neural foraminae and a second anterior procedure was carried out after posterior fusion. Reportedly the patient did well after the operation but suffered local recurrence with complete paraplegia 1 year after the operation and succumbed to the disease 2 years later having denied further therapy. In the third case,¹ cysts residing anterior to the vertebral body were simply evacuated with costotransversectomy.

The lumbar region was the second most commonly reported disease location and simple laminectomy was the most common surgery performed.^{1,11,12,18,29,38} Retroperitoneal abdominal corpectomy with posterior fusion with subsequent surgery for total destruction of the vertebral body is reported in lumbar disease.² No long-term outcome was included in the report regarding disease spread to retroperitoneal tissues.

Five cases of bone fusion were reported in cases with spinal hydatid disease.^{7,12,17,22,25} Autologous pelvic and costal bone grafts were reported. Bhojraj *et al*²⁶ speculated that extensive fusion with decortication might result in spread to uninvolved adjacent bone and reported a successful stabilization with a Luque system and sublaminar wiring after laminectomy and transpedicular curettage. However, the authors provided follow-up data of only 3 weeks which makes the evaluation of their hypothesis impossible. Sapkas *et al*⁷ reported his concerns about the possible infestation of graft material and advocated the use of acrylic cement for osteosynthesis. However, no cases are reported. Keller *et al*²² reported successful graft fusion and preservation of spinal stability even in the presence of verified active disease. Recurrence was noted in three of these cases. In one case, long-term outcome was not reported.⁴ In another, recurrence was seen 1 year⁷ and in the third, 3 years²² after bone fusion. Spinal stabilization was regained after revision in one of

the cases.²² The other case⁷ reportedly refused therapy and died of disease progression 2 years after recurrence.

Complications

Reported surgical complications are perioperative death, misdiagnosis with local spread, recurrence, increasing neurological deficit, secondary infection, CSF fistula and anaphylaxis.^{12,13} A mortality rate of 0 to 3% is reported which progressively increases to 15% in subsequent reoperations.^{1,12,13,21} Extreme caution should be taken not to rupture intradural or epidural solitary cysts as complete removal might potentially result in cure. İşlekel¹ reported a case misdiagnosed as a dermoid cyst. In this case, dural opening was reported to result in intradural invasion and recurrences. Although the effectivity is not proven^{13,38} most surgeons advocate intraoperative scolicalidal agents as well as adjuvant pharmacotherapy. Scolicalidal agents like 3% hypertonic saline, chlorhexidine, 80% alcohol, 0.5% silver nitrate or 10% formalin are used in abdominal disease. Use of Povidone iodide¹⁵ as a scolicalidal agent is reported in spinal cases but long-term results are lacking. Hypertonic saline is the most frequently used agent in spinal disease. İşlekel *et al*¹ reported an operative mortality due to toxic myelitis caused by formalin after a dural tear. Formalin irrigation is also reported to add to the morbidity by increasing tissue necrosis¹³ and should be avoided in spinal cases.

Recurrence

Unfortunately even with extensive surgery results are far from being curative and recurrences are the rule.^{1,2,12,21,29,38,39,40} Up to 40% of patients are reported to have recurrent symptomatology within 2 years of initial operation.^{2,17,21,29} In recurrent lesions the major goal is restoration and preservation of neurological status and spinal stability. Several authors advocate the reinstitution of anti-helminthic pharmacotherapy which should be continued indefinitely.^{21,22} Presence of disease spread to paravertebral tissues further warrants the use of adjuvant therapy.²¹ However, most patients experience a downhill course with worsening neurological status and progressive destruction of the vertebral column with multiple recurrences.^{21,38} Incidence of paraplegia in recurrent disease is reported to be as high as 45%.^{12,34} Recurrence is considered a bad prognostic factor in spinal hydatid disease.²¹ Operative mortalities of up to 14.4% were reported in recurrent cases.¹² Keller *et al*²² reported a cervical case operated on six times with preservation of spinal stability and neurological status. The authors concluded that despite multiple recurrences, spinal stability could be achieved and should be the goal.

Minimally invasive techniques

Diagnostic or therapeutic CT guided fine needle aspiration of hydatid cysts is frequently used in abdominal hydatid disease.^{39,41} This technique however is usually avoided in spinal disease due to the risk of cyst rupture and severe anaphylaxis.¹³ In the single reported case, Spektor *et al*⁴¹ described the successful decompression of an extradural cervical spinal hydatid cyst by CT guided fine needle aspiration with complete resolution of quadriplegia. The authors advocate that with adequate prophylaxis against anaphylaxis and dissemination, CT guided aspiration of spinal hydatid cysts with hypertonic saline irrigation and subsequent chemotherapy may be an alternative to surgical therapy in selected cases.^{39,41} Other minimally invasive techniques such as endoscopic evacuation of cyst material has been reported in the literature.³¹ We think that minimally invasive methods may be of value in selected patients with localized spinal disease, in whom surgical therapy is contraindicated. However, these methods are still experimental and the efficacy and safety have not been determined.

Simple drainage of the infested cystic area is reported to result in pyogenic osteomyelitis and is strongly discouraged.¹³

Pharmacotherapy

Drug therapy with benzimidazole derivatives is proven to be effective in abdominal disease.⁸ A course of Albendazole (800 mg daily in two divided doses) is continued for 1 to 6 months (usually 3 months). Albendazole is preferred over Mebendazole due to better pharmacokinetic properties and superior efficacy against helminths.^{2,9} While these agents do not cure the bone disease they are used in conjunction with surgery to decrease avoid recurrences due to intraoperative spillage.^{7,9,12} Their effectiveness in preventing recurrence is also debatable. There are reports, however, that they may be effective in inoperable patients, prolonging the length of survival.² It should be kept in mind that both of these drugs are teratogenic and embryotoxic, and both may cause alterations in liver function and hematological adverse reactions.⁵ It is reported that the drug Praziquantel is also effective against Echinococci alone or in combination with Albendazole. Lam *et al*² reported an inoperable case of recurrent spinal hydatidosis treated with a combination of Albendazole plus Praziquantel having survived for 34 years.

Prognosis

Even with the use of scolicalidal agents like hypertonic saline or formalin, intraoperative seeding is commonly leading to recurrence rates up to 40%.^{12,13} Recurrences occur 2 to 28 months (mean: 25.2 months) after the initial operation.^{1,21} The recurrence rate for intradural extramedullary disease is reported to be low.¹²

The high recurrence rate is primarily responsible for the poor survival of only 5 years after the onset of symptoms.^{2,7} Lam *et al*² reported that the average age at death is 41 years. Operative mortality rates up to 15% are noted in the literature and are known to increase with subsequent operations. Paraplegia due to recurrent disease is reported to be as high as 45%.^{12,26}

Conclusion

Spinal hydatid disease, although rare in developed countries, must be considered in the differential diagnosis of spinal cord compression syndrome especially in endemic areas. Surgical decompression is the treatment of choice. Due to the invasive nature of the disease, surgery alone is seldom curative. Adjuvant drug therapy as well as intraoperative prophylaxis are indicated. Despite all measures, the disease has a high propensity for recurrence and leads to progressive destruction of the vertebral column and neurological deterioration. Instrumentation with bony fusion may be required to preserve spinal stability. Despite aggressive treatment, overall outcome is still poor leading to a prognosis measured by years.

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