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SPINAL HYDATID DISEASE: THREE CASE PRESENTATIONS

SPİNAL HİDATİK HASTALIK: ÜÇ VAKA SUNUMU

Ömür GÜNALDI[1],
Ender Ali OFLUOĞLU[2],
Hakan KINA[1],
Lütfi Ş. POSTALCI[1],
Hidayet AKDEMİR[2]

[1]Bakırköy Mental Health and Neurological Diseases Education and Research Hospital, Department of Neurosurgery, Bakırköy, İstanbul.
[2]Neurosurgery Medicana İnternational Hospital, Beylikdüzü, İstanbul.

SUMMARY

History: Hydatid disease of the spine is an extremely rare condition with a poor prognosis that presents diagnostic and therapeutic challenges.

Patients and Methods: In this study, we present three cases of spinal cyst hydatidosis. The mean age of the patients was 32.3 years, and the mean follow-up time was 4.6 years. The patients were evaluated by clinical, biochemical, and radiological follow-up. The location of the disease was limited to the lumbosacral region, except for one case that showed involvement of both the lumbosacral and the thoracic spine. The spine was exposed in the posterior for all cases. Decompression or curettage and resection of the infected bone were performed. For one patient, an additional fusion with instrumentation was performed after removing the involved posterior elements. During the postoperative period, combined anti-parasitic therapy with albendazole was also given to all patients.

Results: The outcome was excellent for one case, partial improvement was observed for another case, and no improvement was obtained for the other case. Repeated surgery was necessary for all cases, due to local recurrence or residual cysts.

Conclusions: Spinal hydatidosis is a locally malignant disease with a high reoccurrence rate. The preferred management strategy is spinal decompression, vertebrectomy and instrumentation in appropriate cases, and postoperative use of albendozole as a chemotherapeutic drug. Even when all treatment modalities are applied, local reoccurrence occurs very frequently. Due to the difficulties of treatment, ideally the disease would be prevented rather than treated, by eradication of the parasites from hosts in endemic areas.

Key words: Hydatid cyst, spinal infectıon, surgical treatment.

Level of evidence: Case report, Level IV

ÖZET

Geçmiş bilgiler: Spinal kist hidatik, çok nadir bir durum olup, mevcut tanı ve tedavi olanakları ile prognozu kötü bir hastalıktır.

Hastalar ve yöntem: Biz bu çalışmada, spinal kist hidatik hastalığı olan üç vaka sunduk. Ortalama yaş 32.3 yıl, ortalama takipsüresi ise 4.6 yıldı. Hastalar, klinik, biyomekanik ve radyolojik olarak değerlendirildi. Hem lumbosakral hem torakal tutulumu olan bir hasta dışında diğer hastalarda yerleşim, lomber bölge ile sınırlıydı. Bütün hastalarda omurga posteriordan dekomprese edildi. Enfekte kemik, dekomprese edilerek veya kürete edilerek çıkarıldı. Bir hastada posterior elemanlar çıkarıldıktan sonra ilave olarak enstrümantasyonla füzyon uygulandı. Bütün hastalara ameliyat sonrası albendazol ile anti-paraziter tedavi combine edildi.

Bulgular: Bir hastada sonuç mükemmeldi. Bir hastada kısmi iyileşme görülürken, diğer hastada iyileşme izlenmedi. Bütün hastalarda local nüks veya rezidiv nedeniyle tekrar cerrahi gerekti.

Sonuçlar: Spinal hidatik hastalık, yüksek nüks oranı olan malin bir hastalıktır. Tercih edilen tedavi yaklaşımı spinal dekompresyon, vertebrektomi, uygun vakalarda enstrümantasyon ve operasyon sonrası albendazol kullanılmasıdır. Tüm tedavi yaklaşımlarına rağmen local nüks çok sık görülür. En iyi ve ideal tedavi, hastalığa neden olan parazitin taşıyıcı endemic bölgelerde eradike edilerek korunmaktır.

Anahtar Kelimeler: Kist hidatid, omurga enfeksiyonu, cerrahi tedavi

Kanıt Düzeyi: Olgu sunumu, Düzey IV

Address: Dr. Ender Ofluoğlu, Ayazağa Oyak Sitesi, 16-B Blok, D.13, Şişli, İSTANBUL.
Tel.: 0212 332 05 74
Gsm: 0532 441 09 28
Fax.: 0212 242 39 85
E-mail: enderofluoglu@yahoo.com
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INTRODUCTION:

Hydatid cysts are an infectious disease caused by *Echinococcus granulosus* and are most frequently seen in the Middle East, South America, New Zealand, and Mediterranean countries[2,19]. 60–70% of hydatid cysts are located in the liver, and 10–15% in the lung. Spinal hydatid cysts account for 1% of all cases of hydatid disease[21].

An osseous location is seen for 0.5–2% of cysts, and approximately half of those are vertebrally located[8]. While the majority of cases involve the thoracic and lumbar spine, sacral and pelvic involvements are rare[15]. Disease usually spreads over the spine by direct extension from pulmonary, abdominal, or pelvic infestation, and affects the dorsal region of the spine in about 50% of all cases[4]. Lumbar spine involvement is seen in about 20% of all cases of spinal hydatid disease. Over a period of ten years, three cases of spinal hydatid disease with neurological deficit were diagnosed and treated at our institution, and observed for an average follow-up of 4.6 years.

The aim of this retrospective study is to share our experiences of the pitfalls and challenges in the diagnosis and management of spinal hydatid disease, together with a review of the relevant literature.

CASE REPORTS:

A retrospective evaluation of three patients with spinal hydatid disease was done. The patients were all male, with a mean age of 32.3 (range: 14–52) years. The reoccurrence or residual time was 23.6 months, and the mean duration of follow-up was 4.6 (range: 1–9) years. Clinical presentations of the patients are shown in Table-1. All patients had a history of intractable back pain before the onset of other symptoms, and gradually increasing neurological deficit of the lower limbs. All the patients were investigated with routine hematological screening, plain radiographs of the spine, and ultrasonographic examination of the abdomen. The results of Casoni-Weinberg and indirect hemagglutination tests were positive for two cases and negative for the third. Computed tomography (CT) and magnetic resonance imaging (MRI) were used for diagnosis and delineating the extension of the lesions, evaluating the primary target organs, and determining any reoccurrences. The infected area was restricted to the lumbosacral spine, except for one case showing involvement of the lumbar and thoracic spine together. The disease involved more than one vertebra at the lumbosacral spine in all three patients. The diagnosis wasconfirmed with both histopathological and surgical specimens, due to the presence of multiple cysts within the vertebrae and extradural or intradural space.

The management strategy is shown in Table-2. All patients underwent surgery to excise the cysts, and laminectomy performed by a posterior approach at the level of spinal involvement. The cysts were removed carefully to avoid spillage, and chemical sterilizing agents (20% saline) were used to prevent reoccurrence. Anti-helminth therapy was prescribed of 15 mg/kg/day of albendozole for a year for all patients. The patients were followed up every three months in the first postoperative year and yearly thereafter. repeated surgeries was necessary for all cases due to local reoccurrence or residues. There was no mortality in the study.

Case-1:

A 14-year-old boy was admitted to our clinic with pain in the back and both legs, and disability in walking. He had received surgery for a pulmonary hydatid cyst one year previously at another institute. After pulmonary surgery, he used albendazole as adjuvant chemotherapy for six months at a dosage of 15 mg/kg/day. Before admittance to our clinic, he had back pain for two months and complained of difficulty in walking and pain in both legs for three weeks. On neurological examination, predominantly right asymmetrical paraparesis was observed. Bladder and bowel functions were normal. Computed tomography of the abdomen and lung and abdominal ultrasonography showed no abnormalities. Immunological tests for hydatid cysts (Casoni and indirect hemagglutination) were positive at a level of 1/80 titration. MRI of the lumbosacral region was performed, which revealed a large, complex, multiloculated/multiseptated, predominantly cystic lesion, extending from the L3 to the S2 levels (Fig.-1.a,b).

In addition, MRI of the thoracic region showed a lobulated 3×2 cm cystic lesion with regular contours at the T5 level (Fig.-1.c).

After intravenous gadolinium injection, there was no contrast enhancement on T1-weighted images.

The lesion compressed the dural sac mediolaterally, predominantly at the right side, at the T5 level (Fig.-1.d).

T5 and L4, L5, and S1 laminectomies were performed in the same session, showing many pearly-white intradural extramedullary hydatid cysts. One was removed intact, while the others ruptured intraoperatively, and so the surgical field was irrigated with 20% hypertonic saline Histopathological examination confirmed the diagnosis of hydatid cysts. Albendazole treatment at a dosage of 15 mg/kg/day was started in the preoperative period. The postoperative stage was uneventful, and the patient was discharged on the tenth day postoperatively. He was readmitted to our clinic with a history of urinary retention after a three month symptom-free period. In an MRI examination three months postoperatively, it was shown that the hydatid cysts had totally disappeared in the thoracic region (Fig-1.e), but there was a residualcyst in the lumbar region (Fig-1.f).

The patient was operated on again, and after laminectomy was performed, multiple intradural extramedullary hydatid cysts were completely removed, and the cavity was irrigated with 20% hypertonic saline. Residual hydatid cysts were not observed in an MRI examination on the first day postoperatively (Fig-1.g). The patient had a good recovery with no reoccurrence in a one year follow-up.

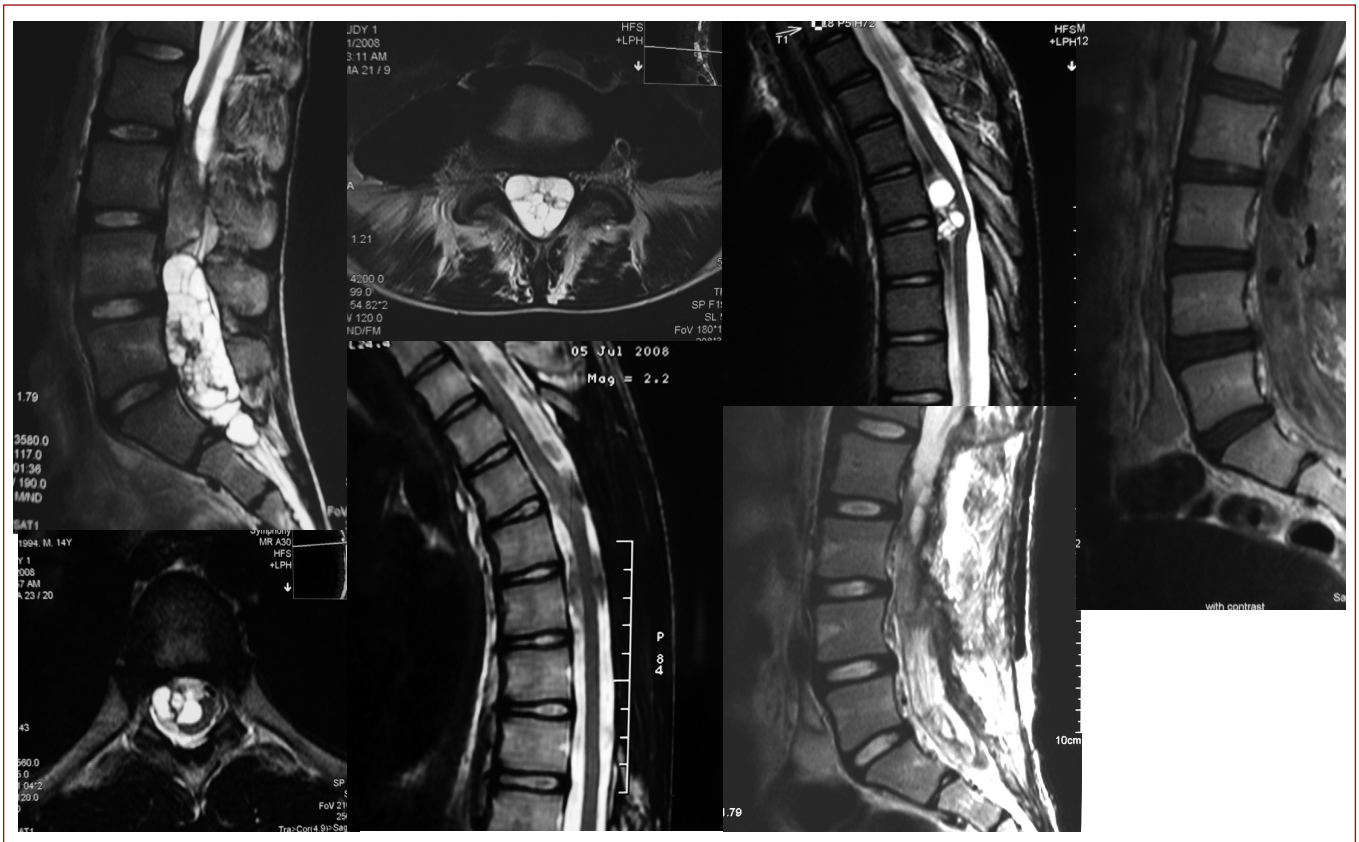

Figure-1. a. Sagittal T2-weighted MRI showing cyst formation from L3 to S2. **b.** Axial T2-weighted MRI showing many intradural extramedullary multiseptate cysts at the L4 level. **c.** Sagittal T2-weighted MRI showing cystic lesions that are almost isodense with cerebrospinal fluid, causing intradural extension and spinal cord compression at the T5 level. **d.** Axial T2-weighted MRI showing multi-cystic lesions compressing the dural sac mediolaterally, predominantly at the right side, at the T5 level. **e.** Three month follow-up thoracic sagittal T2-weighted MRI showing total removal of the cysts. **f.** Three month follow-up lumbar sagittal T2-weighted MRI showing a residual cyst. **g.** Postoperative MRI showing the residual hydatid cyst was totally removed.

Case-2:

A 31-year-old male patient was admitted to our clinic in 2001 because of three months of pain in his ankle and left leg in the form of sciatalgia. The neurological examination revealed a positive Lasegue sign at 45°, ankle and toe dorsal flexion weakness, hypoesthesia at the L5–S3 dermatomes, and a hypoactive Achilles reflex on the left side. The anal reflex and sphincter tonus were normal. Lumbar MRI showed a non-enhancing mass lesion that was compressing the left L5, S1, S2 and S3 roots. The lesion was hypointense in T1-weighted images and hyperintense in T2-weighted images. The lesion was located from the L4 corpus to the S3 level and also had components in the perisacral and sacroiliac articulations (Fig.-2.a).

There was no pathology in cranial, cervical and thoracic MRIs. Tomography of the abdomen and lung and abdominal ultrasonography showed no abnormalities. Immunological tests for hydatid cysts (Casoni and indirect hemagglutination) were negative. During the operation, L4–5 total laminectomy and left L3–S1–S2 partial hemilaminectomy were performed by a posterior approach. Thinning and erosion were observed in the posterior elements of the extradural space. After the excision of multiple pearl-like cysts with white capsules, which had a tendency to rupture spontaneously, the operation area was washed with hydrogen peroxide and hypertonic saline. The diagnosis of hydatid cysts was confirmed histopathologically.

Albendazole treatment (15mg/kg/day) was administered for one year postoperatively. In the first week postoperatively, a lumbosacral MRI was taken and it was observed that the lumbar cysts had disappeared, but there were residual cysts in the sacroiliac area. The patient's postoperative pain decreased and no meaningful changes were observed in muscle strength. The patient was discharged and clinically followed up. He was readmitted to our clinic with left foot drop and urinary retention complaints that had begun ten days previously, after three years symptom-free. On MRI examination of the patient, the presence of hydatid cysts was observed in the sacral and pelvic area (Fig.-2.b).

The patient was operated on again. Perioperatively, bone destruction was observed at the posterior structures of the sacrum. Left S2 and S3 partial hemilaminectomies were performed and the left S1 defect was widened. It was seen that the sacrum was eroded and cyst formation had spread into the pelvis. Multiple pearl-shaped cysts of various sizes, which tended to rupture spontaneously, were removed. The operation site was irrigated with hydrogen peroxide and hypertonic saline. Postoperative albendazole treatment was begun. It was observed that the foot drop and urinary retention improved in the early postoperative stage. In an MRI examination on the second day postoperatively, it could be seen that the hydatid cysts had totally disappeared (Fig.-2.c).

After one year of follow-up, the patient was neurologically intact, but local reoccurrence was observed on a lumbosacral MRI. The patient refused the advised repeat surgery.

Case-3:

A 52-year-old man was admitted to our hospital with progressive pain in his back and both legs, lower extremity weakness, respiratory distress, impotence, and incontinence of the bladder and the bowel. He had received surgery seven times with a diagnosis of lumbar extradural hydatid disease at another institute, and had received long-term oral albendazole. Abdominal ultrasonography showed multiple cysts in the liver and intrapelvic area. Laboratory test results were within normal limits.

MRI showed massive destruction of the L2–3 segment and spondyloptosis. Furthermore, multiple cystic lesions in the extradural space extending from the T12 to S1 segments, in the paravertebral muscles, and in the intrapelvic area were determined (Fig.-3.a,b).

A diagnosis of recurrent hydatid disease was considered. T12, L1, L4, L5, and S1 laminectomies were performed in the same session. During these, many pearly-white hydatid cysts located extradurally were removed completely with their capsule, the cavity was irrigated with 20% hypertonic saline, and posterior decompression and fusion were performed (Fig.-3.c).

After surgery, the back and other pains and respiratory distress improved, but the neurological deficits did not change during the postoperative period. The patient remained non-ambulatory with moderate flaccid paraplegia and a neurogenic bladder after six months of follow-up.

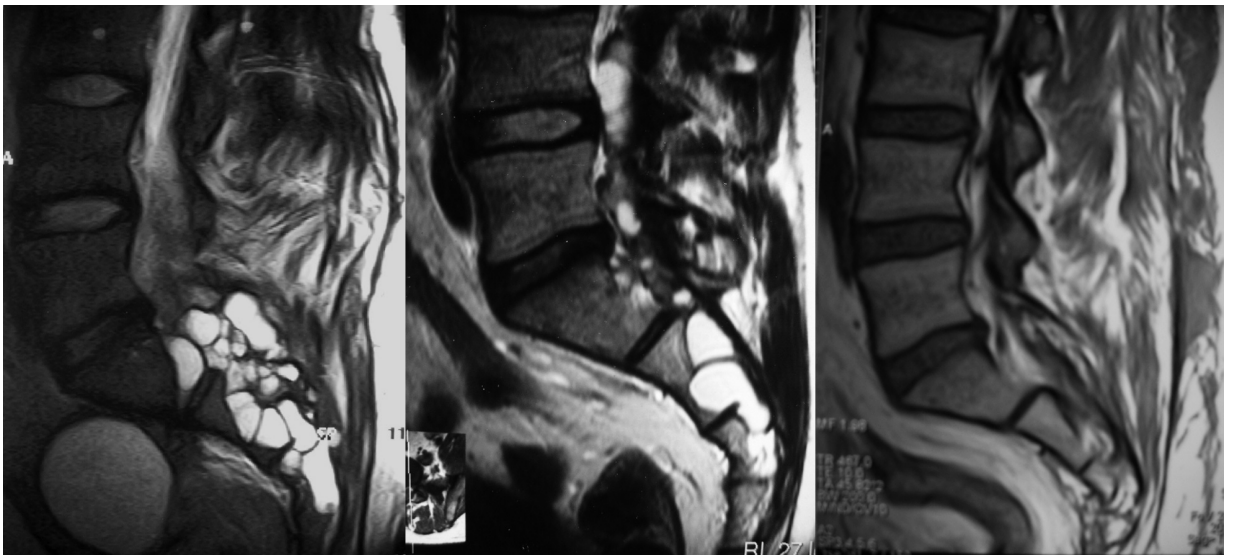

Figure-2.a. The lesion located from the L4 corpus to the S3 level was hyperintense on sagittal T2-weighted images. **b.** A sagittal T2-weighted MRI obtained after three years symptom-free demonstrates dense, multiple cysts in the sacral canal. **c.** After the second operation, the cysts were totally removed.

DISCUSSION:

The vertebral column is the site of primary hydatid disease in 0.5–2% of all echinococcal lesions of the body, but accounts for 50% of all hydatid disease of the bone[1]. The thoracic and lumbar spine is involved in 75% of cases, and neurologic deficit is said to occur in 25–84% of cases[17]. Abbassioun and Amirjamshidi[1] reported the involvement of thoracic vertebrae in 60% of cases, lumbosacral vertebrae in 35% of cases, and cervical vertebrae in 5% of cases in their study. Herrera et al.[14] reported the involvement of thoracic and lumbar vertebrae in 20 cases in 2005. Here, we report two cases with lumbosacral involvement, and one case with both thoracic and lumbosacral involvement.

As the primary site of the infestation and the precise extension of spinal hydatid disease are very difficult to verify, this led Braithwaite and Less[8] to classify the spinal disease into five types: 1) Primary intramedullary hydatid cyst; 2) intradural extramedullary hydatid cyst; 3) extradural intraspinal hydatid cyst; 4) hydatid disease of the vertebrae; and 5) paravertebral hydatid disease.

While hydatid cysts are usually reported in extradural locations, intradural extramedullary hydatid cysts are extremely rare in the literature[4].

Kalkan et al.[16] reviewed 25 cases of intradural extramedullary spinal cysts reported in the literature worldwide in 2007. In the present study, cysts were located extradurally in two cases and intradural extramedullary in the other case.

The clinical presentation depends on the level of the affected vertebrae and the stage of the disease. There are no pathognomonic signs or symptoms of spinal hydatid disease other than symptoms related to compression[19]. During the intraosseous phase, no symptoms are produced. When the bone is breached by the cysts and the extradural space is violated, neurological deficit with unremitting pain ensues[18]. Most symptoms relate to the spinal cord or spinal nerve root because of compression effects or ischemic changes. The patients usually present with a long history of back and radicular pain. Weakness of the limbs occurs later, and paraplegia is reported in 25–84% of cases[9]. In our study, all patients suffered from back and radicular pain. While paraparesis and paraplegia were present in one case, cauda equina syndrome developed at the second admission of another, who had had a normal examination at the preceding hospitalization.

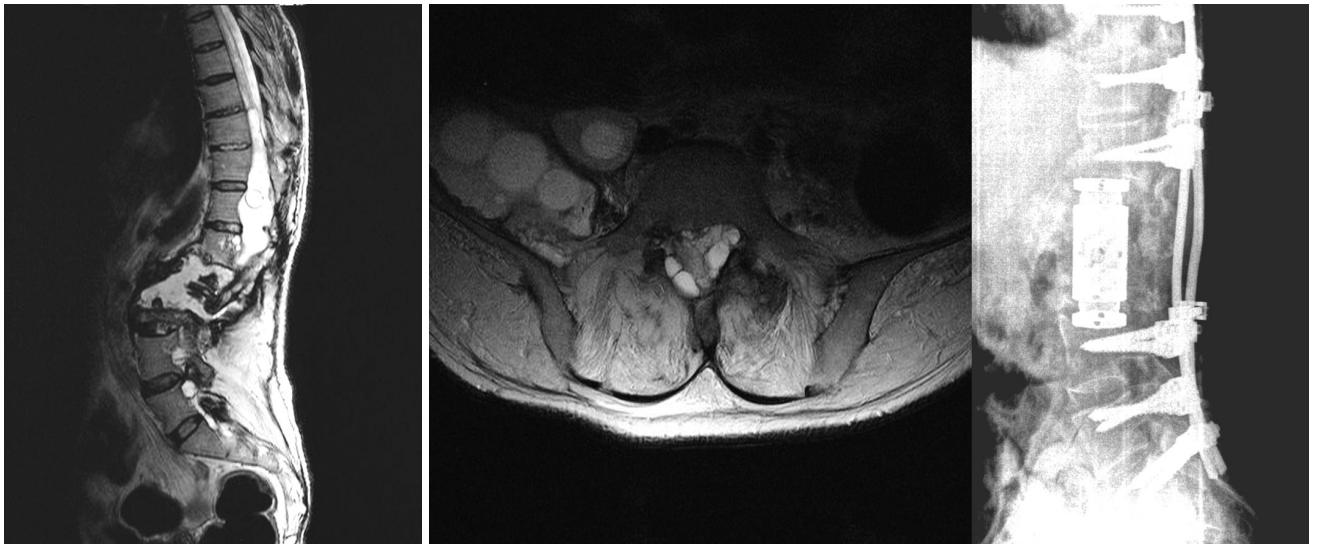

Figure-3.a. MRI showing massive destruction of the L2–3 segment, spondyloptosis and multiple cystic lesions in the extradural space extending from the T12 to S1 segments. **b.** Axial T2 MRI images showing many cysts in the paravertebral muscles and in the intrapelvic area. **c.** Posterior decompression and fusion were performed after removal of invaded vertebral bodies and cysts.

The diagnosis is usually suspected on the basis of clinical or radiological findings, in addition to a history of residence in endemic areas. An accurate diagnosis may be aided in some individuals by eosinophilia and positive results of a complement fixation test, a Casoni skin test, and an enzyme–linked immunoassay[7]. Although the positive predictive value of the Casoni test is 95%, it shows 40% false-positive results[13].

The radiographic characteristics of spinal hydatid disease are similar to those of primary and secondary tumors, Pott disease, and pyogenic infection of the spine[20]. MRI is the best imaging tool for a diagnosis of spinal hydatid disease, because it can show the relationship to the normal structures and the spinal cord, and the extension into the soft tissue[16]. A hydatid cyst is usually a single thin-walled cyst, with the contents showing a similar intensity to that of the cerebrospinal fluid[10]. On T2-weighted imaging, the cyst wall shows a low-intensity rim around a cyst content showing homogenously high signal.

Recently, one study denoted that MR spectroscopy can be useful to distinguish hydatid cysts from cysticercosis[11].

Successful treatment of vertebral hydatidosis represents a challenge, because of its local invasive features. Surgery is the treatment of choice for spinal hydatid disease. The surgical approach should be guided by clinical evaluation and the findings on CT and MRI. Laminectomy is used most frequently for the surgical management of spinal hydatidosis. Total removal of cysts without rupture should be the surgical goal[15]; however, radical excision is almost impossible in hydatid disease of the spine, because of the absence of distinct anatomical planes and the existence of neural structures. Some authors have used an anterior approach to perform total corpectomy and bone grafting, and a posterior approach to remove the involved posterior elements and to perform posterior fusion with instrumentation[12]. Apt et al. stated that posterior spinal cord decompression alone was incomplete, due to a failure to remove all cysts[3]. However, early surgical decompression, radical resection of the affected vertebrae and stabilization, irrigation of the surgical field with various solutions, and the usage of postoperative adjuvant chemotherapy is the preferred treatment. Although the efficacy of particular solutions remains unproven, they have been widely used in reported studies and include hypertonic saline (3%, 10%, and 20%), 0.5% betadine, 0.5% silver nitrate, and 2% formalin[1]. Poor outcomes may be related to the localization (intradural and extramedullary) of the cysts, and the weak penetration of anti-helminth drugs, albendazole or mebendazole, and metabolites into the intradural space by a passive diffusion transport mechanism[22].

Albendazole shows good oral absorption and higher intracystic levels. To prevent reoccurrence, therapy with albendazole is recommended for one year after adequate neural decompression. In the long-term, the results of reports on anti-helminth treatment for spinal hydatid disease in the literature are disappointing[5].

Reoccurrence is a dilemma for patients and surgeons. A reoccurrence rate of 30–100% has been reported in the literature[14]. Most reoccurrences occur within four years[1]. The average remission time between reoccurrences has been reported to be 29.6 months in one study, emphasizing the need for regular and prolonged follow up[6]. The high reoccurrence rate is due to extensive spread of the disease at the time of diagnosis, and rupture, spillage and implantation of cysts during excision and surgical intervention. Turtas et al. reported a 50% reoccurrence rate after posterior decompression[23]. They recommended extensive resection of the vertebral bodies and fusion to control hydatidosis. Incomplete removal of the affected vertebrae through an extensive posterior approach without spinal stabilization may exacerbate cord compression as a result of the increasing deformity.

In spinal hydatidosis, cure is possible in only a few instances, for patients with a single cyst that can be removed with an intact capsule[1]. We observed local reoccurrences in all three patients. All patients underwent repeated surgery for local reoccurrences or residual cysts.

In conclusion, spinal hydatidosis should be considered in differential diagnosis of any cystic lesions of the spinal cord, and also destructive lesions of the vertebrae, in endemic areas. Eradication of the disease cannot be achieved even after radical surgical intervention and chemotherapy. Although the role of chemotherapy in the prevention of the disease is not specifically determined, it is recommended by nearly all authors. The preferred management is spinal cord decompression by a posterior or anterior approach, vertebrectomy and instrumentation in appropriate cases, followed by adjuvant chemotherapy. Despite use of all these treatment modalities, this disease has a malignant course due to the high rate of reoccurrence. Results are seldom satisfactory and the prognosis is usually poor. We conclude that new anti-helminth drugs are required.

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