

Hydatid Disease of the Lumbar Spine: Combined Surgical and Medical Treatment—A Case Report

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Larval forms of the cestode worm *Echinococcus* cause hydatid disease. The life cycle of the parasite often involves dogs as final hosts and human beings and sheep as intermediate hosts; therefore, the disease is prevalent in sheep-raising countries (eg, the Middle East and Turkey). Human beings become infected by ingesting water or food contaminated by the parasite eggs. After the eggs hatch in the intestine, they migrate through the portal venous system to settle in the liver and lungs in most cases.¹ Rarely, the parasite reaches the systemic circulation. Spinal involvement represents 0.5% to 1% of all cases of echinococcosis. Spinal infection represents about 44% of skeletal echinococcal cysts.² Cysts are common in the dorsal spine, followed by lumbar and sacral sites.³

Hydatid disease of the spine could occur by direct extension from pulmonary or pelvic infestation; less commonly, it starts primarily in the vertebral body. In spinal involvement, the compression of neural tissue with resulting neurological deficit is relatively common.⁴

The treatment recommended is decompression of the neural tissue, excision of the cyst with or without stabilization, and postoperative antihelminthic drug therapy for a long duration to prevent late recurrence.⁵

Conclusions as to the best postoperative regimen with minimal side effects and easy patient compliance are not available yet because hydatid disease is a rare condition and too few cases occur to obtain proper statistics.⁵ Medical treatment could be in the form of albendazole or mebendazole, with or without praziquantel.⁶ Early discontinuation of medical treatment—with risk of recurrence—could be due to the gastrointestinal disturbances or hepatic side effects or the long duration of treatment needed.⁵⁻⁸

We present a case of primary hydatid disease of the lumbar spine with no other organ involvement. The patient was treated by surgical excision followed by medical treatment in the form of albendazole for 1½ years postoperatively.

The patient now has been followed up for 6 years postoperatively with no recurrence.

CASE REPORT

The patient was a 32-year-old nonsmoking man who worked as a carpenter and had no history of major illness. The patient presented to the outpatient clinic with a history of persistent low back pain during the preceding 5 months that was treated by nonsteroidal anti-inflammatory drugs. The low back pain partially improved with medical treatment, but pain recurred after stoppage of medication. The patient started to experience weakness in both lower limbs and in sphincter control during the 2 weeks before presentation.

Clinical examination showed diminished sensation in both lower limbs to the level of the third lumbar nerve root. Motor examination showed weakness of both lower limbs, which was more obvious in the right lower limb. This weakness affected ankle dorsal and plantar flexors and knee extensors (level of third lumbar nerve root).

Plain radiographs showed an osteolytic lesion affecting mainly the posterior half of the vertebral body of the third lumbar vertebra. Computerized tomography and nuclear magnetic resonance imaging showed bone destruction of the posterior two thirds of the vertebral body and invasion of the spinal canal with encroachment on the spinal cord (Figure 1). The osteolytic lesion was extending through

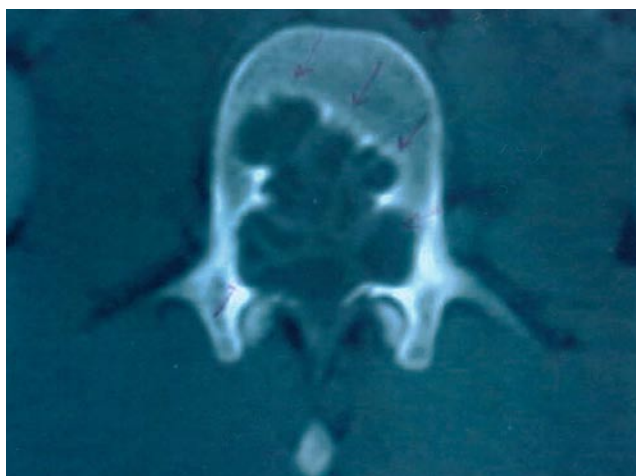


Figure 1. Computerized tomography at the level of third lumbar vertebra shows multiloculated osteolytic lesion involving the posterior half of the vertebral body and extending to the spinal canal with cord compression.

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Figure 2. Magnetic resonance T₂-weighted image shows fluid-containing osteolytic lesion (hydatid cyst) destroying the posterior half of the third lumbar vertebral body and extending posteriorly, compressing the spinal cord.

the intervertebral foramen to the paraspinal muscles. This bone-destroying lesion was multilocular with positive signal on a magnetic resonance T₂-weighted image. This positive signal gave an indication that this lesion was a fluid-containing cyst, which could be serous fluid or blood (Figure 2).

The decision was made to decompress the neural tissue surgically with incision or excision biopsy of the osteolytic lesion. A posterior midline surgical approach was used. Wide laminectomy of the third lumbar vertebra was performed. The lesion was removed using the same technique of posterior lumbar interbody fusion.⁹ After curettage, an autogenous bone graft was put in to replace the removed and destroyed bone of the posterior vertebral body. Posterior fixation using pedicular screws was associated with posterolateral fusion (Figure 3).

Postoperative pathological examination of the cyst revealed that it was of hydatid nature, with detection of hydatid scoleces. Serological studies showed a positive indirect hemagglutination test for hydatid disease with a titer of 1/1280. Chest radiological assessment, abdominal ultrasound, and bone scan were done to exclude any other lesion, and these proved to be negative.

Medical treatment in the form of prophylactic antibiotic for 5 days was combined with nonsteroidal anti-inflammatory drugs. To avoid a recurrence, medical treatment for hydatidosis was given in the form of albendazole, 2 tablets a day (each 200 mg) for 4 weeks. This course was given 3 times 1 month apart. This was followed by 6 weeks of

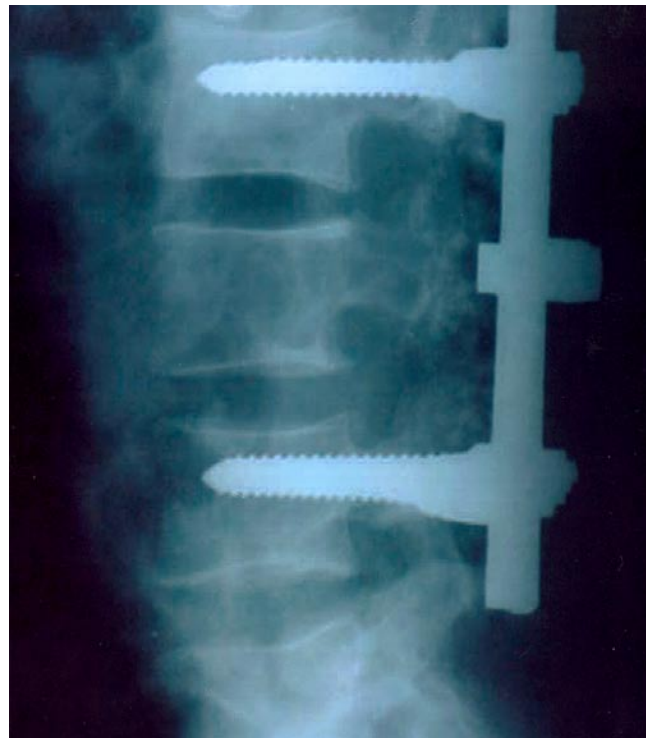


Figure 3. Six-year follow-up plain radiograph lateral view of the lumbar spine shows completely healed lesion in the third lumbar vertebra. Internal fixation was achieved with the pedicular screws placed between the second and fourth lumbar vertebrae, with proper alignment of the spine.

treatment that was repeated 3 times, with 6-month resting periods between each course. At 1½ years postoperatively, medical treatment was stopped completely because of elevated liver enzymes, and since then the patient has refused to take any more antihelminthic drugs and has preferred to continue with serological follow-up without medical treatment.

The patient was followed-up for 6 years postoperatively with clinical examination and plain radiographs (Figure 3), liver function tests in the form of serum aspartate aminotransferase and alanine aminotransferase, and hydatid serological examination (indirect hemagglutination test) every 6 months. The indirect hemagglutination test for hydatid disease titer started to return to normal after 1 year postoperatively, with no recurrence. Liver function was back to normal soon after discontinuation of medical treatment. The patient had full neurological recovery 3 months postoperatively.

The patient now has a full-time job, is active, and occasionally complains of low back pain that does not affect his level of activity. The patient does not take any medication for the occasional back pain.

DISCUSSION

Hydatid disease of the spine is not a common problem but is associated with great morbidity.² The prognosis is considered poor because of disease recurrence rates. For this reason, many authors recommend maintaining medical treatment postoperatively for several years to avoid recurrence.⁵

The patient's compliance plays an important role in postoperative medical treatment. Drugs used in this type of disease are associated with some gastrointestinal problems that affect patients' compliance.⁷ Also, the hepatic toxic effect of these drugs requires close monitoring of liver function during and after treatment.⁵ Nevertheless, surgical removal alone is not enough, given that radical resection is not always possible because of the sensitive anatomical structures surrounding the cyst.¹⁰

We think surgical and medical treatment are complementary and that this needs to be considered by the treating physician to achieve the goal of treatment, which is eradication of the disease. The shorter the duration of medical treatment, the better the patient compliance and the fewer the side effects.

In this case report, the combination of surgical resection and postoperative chemotherapy of relatively short duration achieved the goal of treatment. In the future this program of chemotherapy needs to be tried in more clinical cases of hydatid disease of the spine to get more conclusive data on the treatment results.

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The authors report no actual or potential conflict of interest in relation to this article.

REFERENCES

1. Kammerer WS, Schantz PM. Echinococcal disease. *Infect Dis Clin North Am.* 1993;7:605-618.
2. Charles RW, Govender S, Naidoo O. Echinococcal infection of the spine with neural involvement. *Spine.* 1988;13:47-49.
3. Pamir MN, Akalan N, Ozgent T, Erben A. Spinal hydatid cyst. *Surg Neurol.* 1984;21:53-57.
4. Vengsarkar US, Abraham J. Hydatid disease of the spine. *J Postgrad Med.* 1965;11:133-136.
5. Vicuna RG, Carvajal I, Garcia AO, et al. Primary solitary echinococcosis in cervical spine. *Spine.* 2000;25:520-523.
6. Lam KS, Faraj A, Mulholland RC, Finch RG. Medical decompression of vertebral hydatidosis. *Spine.* 1997;22:2050-2055.
7. Keller TM, Schweitzer JS, Helfend LK, Chappell T. Treatment of progressive cervical spinal instability secondary to hydatid disease. *Spine.* 1997;22:915-919.
8. Basak M, Ozel A, Yildirim O, Erturk M. Relapsing hydatid disease involving the vertebral body and paravertebral soft tissues. *Acta Radiol.* 2002;43(2):192-193.
9. Brislin B, Vaccaro AR. Advances in posterior lumbar interbody fusion. *Orthop Clin North Am.* 2002;33(2):367-374.
10. Baykaner MK, Dogulu F, Ozturk KG, Edali N. A viable residual spinal hydatid cyst cured with albendazole. *J Neurosurg.* 2000;93:142-144.