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Recurrent Vertebral Hydatidosis with Locally Aggressive Tumor-Like Behavior and Progressive Compressive Myelopathy: A Case Report

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ABSTRACT

Background: *Hydatid disease, or cystic echinococcosis, is a zoonotic parasitic infection caused by the larval stage of Echinococcus granulosus. The liver and lungs are the most frequent sites of involvement, whereas osseous localization is uncommon. Among skeletal forms, vertebral hydatidosis is one of the most severe because of its infiltrative behavior, destructive bone involvement, high recurrence risk, and potential for irreversible neurological impairment.*

Case Presentation: *We report the case of a 46-year-old man from western Morocco with recurrent disseminated hydatid disease involving the lungs, liver, and spine. Over a 20-year period, he underwent multiple surgical procedures for recurrent hepatic, pulmonary, and vertebral hydatid cysts. He presented with progressive bilateral lumbosciatica, severe gait limitation, spastic paraparesis, pyramidal signs, sensory loss below the umbilicus, and sphincter dysfunction, consistent with chronic compressive myelopathy.*

Imaging and Management: *Spinal MRI showed multiloculated daughter cysts extending from T9 to T12, with paravertebral, epidural, and intraspinal involvement causing spinal cord compression. Axial sequences demonstrated a paravertebral hydatid abscess with posterior soft-tissue extension and fistulization toward the skin surface. CT showed vertebral lysis, postoperative instability, and mechanical failure of the osteosynthesis construct. Surgical decompression was performed with evacuation of the paravertebral abscess, removal of daughter cysts, debridement of involved tissues, and extraction of the broken osteosynthesis material.*

Outcome: At three months after surgery, the neurological status remained essentially unchanged, with persistent spasticity, severe residual sensorimotor deficit, and marked functional limitation.

Conclusion: *Recurrent vertebral hydatidosis should be regarded as a chronic, locally aggressive, destructive spinal disease with tumor-like behavior. Early recognition of recurrence, maximal safe decompression, prevention of intraoperative dissemination, stabilization when required, and prolonged radiological surveillance are essential to preserve neurological function.*

Keywords

Vertebral hydatidosis, Spinal echinococcosis, Hydatid cyst, Recurrence, Paravertebral abscess, Compressive myelopathy, Osteosynthesis failure, Albendazole.

Introduction

Hydatid disease, or cystic echinococcosis, is a globally distributed zoonotic parasitic infection that remains highly endemic in pastoral regions, including Mediterranean countries. It represents a major public health burden, affecting more than one million people worldwide and causing substantial morbidity, mortality, and socioeconomic losses [1-4].

Morocco is considered a highly endemic country, with persistent

transmission in both humans and livestock. This endemicity is mainly related to the traditional dog-livestock cycle, rural exposure, and slaughtering practices [2,3].

The liver and lungs are the most frequent sites of involvement, whereas osseous localization remains uncommon. Among skeletal forms, the vertebral column is the predominant site and one of the most severe localizations because of its infiltrative behavior, destructive bone involvement, recurrence risk, and potential for irreversible neurological impairment [5-9].

Case Report

A 46-year-old man living in a village near Essaouira, western Morocco, presented with progressively worsening bilateral lumbosciatica associated with increasing difficulty walking. He initially required a cane for ambulation and subsequently developed intermittent claudication of the lower limbs, with a walking perimeter progressively reduced to approximately 2 meters. The clinical course evolved toward severe neurological deterioration, characterized by spastic paraplegia and progressive sphincter dysfunction, including urinary and fecal incontinence.

The patient had a significant past medical and surgical history of recurrent hydatid disease over the preceding 20 years. He had undergone 14 surgical procedures for pulmonary, hepatic, and vertebral hydatid cysts. Each vertebral surgical intervention was followed by clinical improvement; however, the disease repeatedly recurred, leading to progressive functional impairment.

Table 1: Previous surgical history according to hydatid cyst location.

Hydatid cyst location	Years of surgery
Hepatic hydatid cyst	1994, 2002, 2010
Pulmonary hydatid cyst	1996, 2002
Vertebral hydatid cyst	2004, 2006, 2011, 2015, 2018, 2020, 2022, 2023, 2024

On admission, the patient was afebrile and hemodynamically stable. Neurological examination revealed spastic paraplegia of the lower limbs. Deep tendon reflexes were markedly exaggerated, with bilateral hyperreflexia of the patellar and Achilles tendons. Bilateral clonus was present, and plantar reflex testing showed an upgoing Babinski response on both sides, consistent with pyramidal tract involvement.

Motor examination demonstrated severe bilateral lower-limb weakness. Muscle strength was graded 1/5 globally according to the Medical Research Council scale. Sensory examination revealed a clear sensory level at approximately the umbilical region, with preserved sensation above this level and complete sensory loss below it. Both superficial and deep sensory modalities were absent in the lower limbs. Sphincter dysfunction was evident at the time of examination, with urinary and fecal incontinence.

Spinal MRI showed multiple daughter cysts arranged in a multiloculated pattern, suggestive of recurrent spinal hydatidosis. The cystic lesions extended along the thoracic spine from T9 to T12, with compression and displacement of the spinal cord. The distribution of the lesions suggested transosseous and foraminal spread from vertebral involvement toward the paravertebral and epidural compartments, reflecting the infiltrative and recurrent nature of vertebral hydatid disease.

Axial sequences revealed posterior paravertebral extension with a large multiloculated cystic collection extending toward the subcutaneous tissues and skin surface, consistent with fistulization of a paravertebral hydatid abscess. CT scan showed vertebral lysis, postoperative instability, and mechanical failure of the osteosynthesis construct.

Given the severity of the neurological deficit and the radiological evidence of spinal canal compromise, the patient underwent surgical management aimed at neural decompression. Intraoperatively, evacuation of the paravertebral abscess was performed, together with careful removal of the daughter cysts and debridement of the involved tissues. The broken osteosynthesis material was removed because of mechanical failure and its potential contribution to local instability and persistent inflammatory reaction.

After cyst evacuation and debridement, the operative cavity was irrigated with hypertonic saline, used in this case as a scolicidal agent. Postoperative systemic antiparasitic therapy based on albendazole was administered to reduce cyst viability, treat microscopic residual disease, and decrease the probability of further recurrence.

At the three-month postoperative assessment, the neurological status remained essentially unchanged. The patient exhibited persistent spastic paraparesis with marked pyramidal hypertonia of the lower limbs, associated with residual motor weakness and sensory impairment. Functional autonomy remained severely limited, with no meaningful recovery of gait capacity.

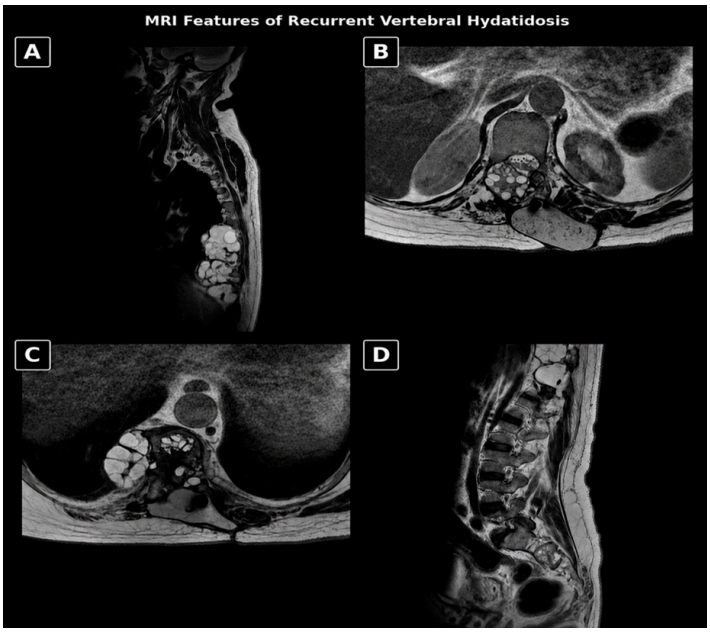


Figure 1: Multiplanar T2-weighted spinal MRI in recurrent vertebral hydatidosis. A: Sagittal image showing multiloculated paravertebral and intraspinal cystic lesions extending from T9 to T12. B,C: Axial T2-weighted MRI showing a multiloculated paravertebral hydatid abscess with posterior soft-tissue extension and fistulization toward the skin surface. The lesion is associated with epidural/intraspinal involvement, resulting in spinal canal narrowing and compression of neural structures. D: Sagittal image showing vertebral involvement with postoperative changes and canal compromise.



Figure 2: Sagittal CT scan showing postoperative osteosynthesis material with mechanical failure, vertebral lysis, posterior element involvement, and spinal canal compromise.

Discussion

Vertebral hydatid disease represents the most frequent form of osseous echinococcosis, accounting for approximately 50% of skeletal hydatid localizations. The thoracic spine is most commonly involved, followed by the lumbar and cervical regions. The disease usually begins within the cancellous bone of the vertebral body, where the parasite progressively proliferates in an infiltrative manner. In the absence of a true limiting pericyst, cystic extension may occur along trabecular bone and subsequently involve the pedicles, laminae, spinous processes, and adjacent paravertebral soft tissues [6-9].

As the disease progresses, cortical bone destruction may allow extension into the epidural space. Epidural dissemination is clinically significant because it can result in compression of the spinal cord, cauda equina, or nerve roots. Neurological manifestations depend on the level and severity of compression and may include radiculopathy, myelopathy, sensory disturbances, motor weakness, paraplegia, and sphincter dysfunction. Neural compression may result from the direct mass effect of cystic lesions, vertebral collapse secondary to bone destruction, or associated epidural inflammatory tissue [7,8].

Progressive osteolysis weakens the structural integrity of the affected vertebrae and may lead to spinal instability. Advanced disease can be complicated by pathological fractures, vertebral collapse, kyphotic deformity, and mechanical instability. Because of its destructive and infiltrative pattern, vertebral hydatidosis may closely mimic malignant spinal tumors or infectious spondylodiscitis, particularly tuberculous spondylitis. Early recognition is therefore essential to prevent irreversible neurological impairment and to guide appropriate surgical and antiparasitic management [7,8].

The present case illustrates the chronic and aggressive course of recurrent vertebral hydatidosis over a 20-year period [5]. Repeated recurrence was associated with progressive vertebral lysis, spinal canal compromise, and stepwise neurological deterioration. The patient's clinical presentation, dominated by spastic paraparesis, pyramidal signs, and sphincter dysfunction, reflects advanced spinal cord involvement [5-8].

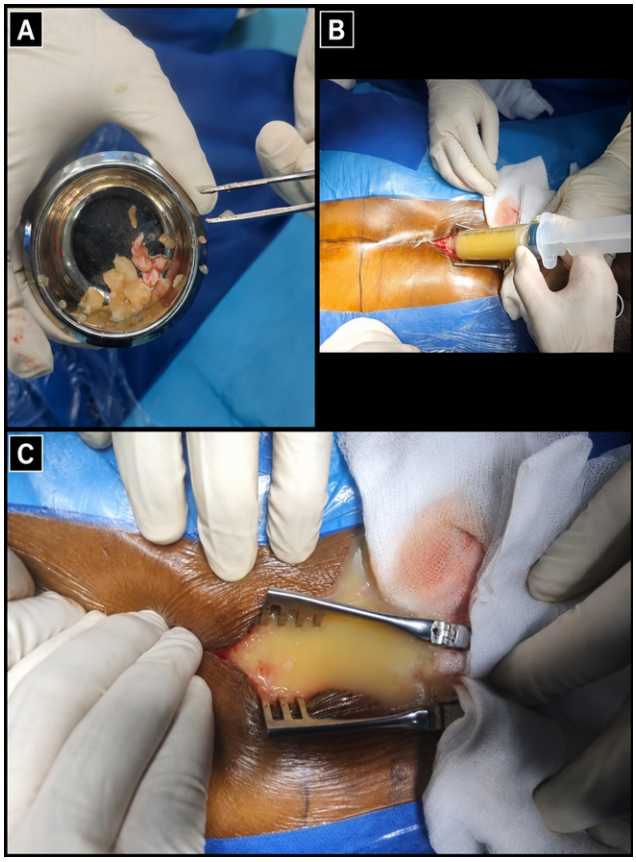


Figure 3: Intraoperative views of recurrent vertebral hydatidosis. A: Evacuated daughter cysts and hydatid fragments. B: Aspiration of the paravertebral abscess. C: Surgical exposure and drainage of the paravertebral abscess cavity, showing abundant purulent/cystic material.

These findings suggest chronic compressive myelopathy, likely aggravated by recurrent cystic expansion, inflammatory reaction, postoperative fibrosis, and progressive structural instability secondary to vertebral destruction [5-8]. A notable feature of this case is the progressively reduced neurological recovery after successive surgical interventions, a phenomenon that may be explained by cumulative spinal cord injury [5-8].

Recurrent compression may initially produce reversible dysfunction; however, prolonged or repeated compression can lead to irreversible myelopathic changes, including ischemia, demyelination, gliosis, and neuronal loss. In addition, repeated operations may contribute to epidural scarring and adhesions, making subsequent decompression technically more difficult and increasing the risk of incomplete resection or neurological injury. The presence of sphincter dysfunction is particularly concerning, as it usually indicates severe involvement of long spinal pathways and is often associated with poorer functional prognosis [5-8].

From a therapeutic perspective, recurrent vertebral hydatidosis remains challenging. Surgical treatment is primarily aimed at neural decompression, maximal safe cyst removal, prevention of intraoperative rupture, and restoration of spinal stability when necessary. However, radical excision is rarely achievable in extensive vertebral disease because cystic elements may infiltrate bone and surround neural or vascular structures.

Consequently, surgery alone is frequently insufficient, and recurrence may occur even after apparently successful decompression [5,7-9].

Based on published reviews, albendazole may not cure or reliably prevent recurrence in spinal cystic echinococcosis, although it is still generally used as an adjunct because it may reduce viability, delay recurrence, or limit seeding after cyst rupture. More recent postoperative spinal echinococcosis data also suggest that longer albendazole therapy may not prevent local recurrence, although it might delay relapse compared with shorter treatment [10].

Conclusion

Recurrent vertebral hydatidosis should be regarded as a chronic, destructive spinal disease with tumor-like behavior rather than a simple benign parasitic lesion. Recurrence remains one of the major therapeutic challenges in vertebral hydatid disease. Its occurrence is influenced by cyst rupture, incomplete excision, multiplicity of lesions, residual intraosseous or epidural parasitic elements, and the duration and effectiveness of postoperative antiparasitic therapy.

Optimal management should rely on early recognition of recurrence, maximal safe decompression, meticulous prevention of intraoperative dissemination, spinal stabilization when required, and prolonged postoperative clinical and radiological surveillance.

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