SAS Journal of Medicine
Abbreviated Key Title: SAS J Med
ISSN 2454-5112
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology

Vertebral Echinococcosis Manifesting as Paraplegia: Insights from a Rare Case

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DOI: https://doi.org/10.36347/sasjm.2026.v12i01.001 | **Received:** 19.09.2025 | **Accepted:** 24.11.2025 | **Published:** 06.01.2026

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Abstract **Case Report**

Hydatidosis caused by echinococcus granulosus may affect any organ in the body, with the lungs and the liver as the most commonly affected organs. Vertebral compromise resulting from echinococcus granulosus has a low prevalence and accounts for less than 1% of bone compromise. We present the case of a 46-year-old woman from Morocco who presented with progressive back pain followed by acute paraplegia. Imaging revealed extensive osteolytic destruction of the T1-T3 vertebrae with associated paravertebral and pleural collections. Although initial suspicion was of spinal tuberculosis, subsequent biopsy confirmed the diagnosis of vertebral hydatidosis. Treatment consisted of surgical decompression, instrumented spinal stabilization, and adjuvant albendazole. Despite successful surgical intervention, the patient remained paraplegic at 18-month follow-up. This case highlights the diagnostic challenge and the need for early intervention to prevent severe neurological sequelae in patients with vertebral hydatidosis.

Keywords: Spinal hydatidosis; Cystic echinococcosis; Vertebral hydatid disease; Spinal cord compression.

INTRODUCTION

Hydatid disease, or cystic echinococcosis, is a zoonotic parasitic infection caused by the larval stage of *Echinococcus granulosus*. While it typically affects the liver and lungs, skeletal involvement is rare (0.5–4% of cases) [1–3], with the spine being the most commonly affected bone, though still comprising less than 1% of all cases[1].

In spinal hydatidosis, the parasite spreads hematogenously to vertebral bodies and proliferates without a fibrous capsule. This leads to slow, infiltrative bone destruction due to direct growth of daughter cysts [1,4], earning it the label "the malignant form of a benign disease" due to its aggressive nature, high recurrence, and poor prognosis[5].

Diagnosis is often delayed as the disease can remain asymptomatic for years, only becoming apparent after significant structural damage or neurological symptoms occur [6]. The reported case illustrates this, where chronic back pain progressed to acute paraplegia, highlighting both the diagnostic difficulty and potential severity of spinal hydatid disease.

CASE REPORT

A 46-year-old woman from a rural region of Morocco, with a known history of contact with domestic dogs and sheep, presented to our institution with a chief complaint of chronic debilitating upper back pain that had progressed over several months. Her illness began with an insidious course of upper thoracic pain over the preceding year. This initially manageable symptom progressed significantly in the final weeks before presentation, leading to bilateral leg weakness. Her condition then rapidly declined, resulting in complete paraplegia, urinary incontinence, and the development of a sensory deficit.

On physical examination, the patient was alert and oriented but in evident distress from pain. Palpation of the upper thoracic spine elicited significant tenderness. The neurological assessment confirmed a severe myelopathy, characterized by flaccid paralysis of the lower extremities (motor power grade 0/5) and a complete loss of sensation below the T6 dermatome, corresponding to the level of the xiphoid process. Deep tendon reflexes in the lower limbs were absent; however, bilateral Babinski signs were present, collectively indicating a severe upper motor neuron lesion.

Initial laboratory findings were largely unremarkable, with a normal white blood cell count. Inflammatory markers were only mildly elevated (C-reactive protein 15 mg/L, ESR 28 mm/hr).

Given the acute and severe neurological deficit, an emergency computed tomography scan was performed to identify the cause of the compression. The scan revealed extensive osteolytic destruction of the T1, T2, and T3 vertebrae, involving the bodies, pedicles, and posterior arches. This was associated with large paravertebral, mediastinal, and bilateral pleural soft tissue collections. The lesion was seen infiltrating the spinal canal, causing severe mass effect on the thoracic cord *(Fig1and 2).*

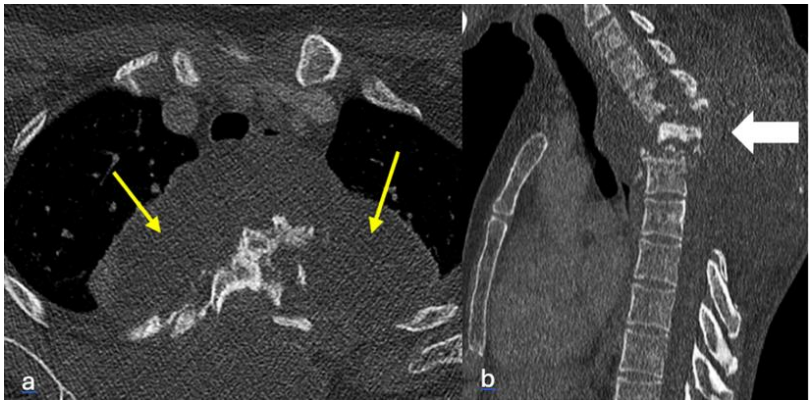

Figure 1 : Axial (a)and sagittal(b) CT reconstructions (bone window) demonstrating destruction of D1–D3 vertebrae, posterior wall recession, and the resulting angulation of the spine (white arrow).it shows also massive paravertebral and pleural collections (yellow arrows)

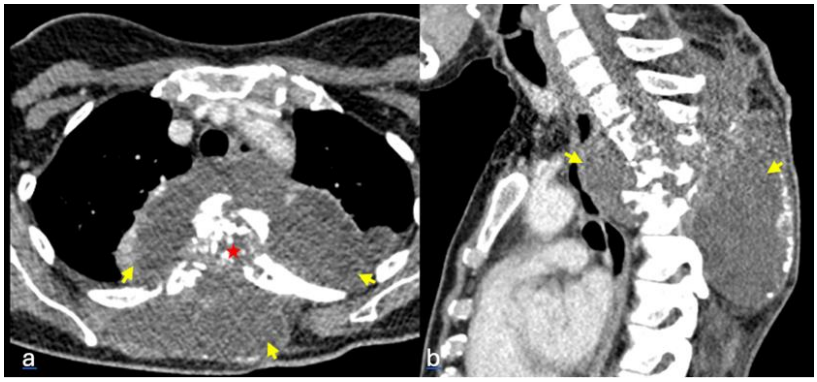

Figure 2 : Axial (a) and sagittal (b)contrast-enhanced CT (mediastinal window) showing extensive vertebral osteolysis, large paravertebral and pleural collections (yellow arrows), and intramedullary extension (red asterisk)

Considering the radiological findings of destructive spondylodiscitis in a patient from an endemic area, spinal tuberculosis (Pott's disease) was the first diagnosis to consider. Consequently, a workup for *Mycobacterium tuberculosis* was initiated. An interferon-gamma release assay (Quantiferon-TB Gold) was performed, which returned negative, making Pott's disease less probable but not definitively excluded.

A subsequent MRI was performed, which played a pivotal role in challenging the initial diagnosis of spinal tuberculosis (Pott's disease) and raising suspicion for vertebral hydatidosis. The imaging revealed a well-defined, multiloculated cystic lesion with intramedullary extension, showing the characteristic features of hydatid disease: T2 hyperintensity, T1 hypointensity, and peripheral enhancement following gadolinium administration. These findings were not compatible with typical vertebral tuberculosis.

Following this revised diagnosis, a multidisciplinary team comprising neurosurgeons, orthopedic surgeons, and infectious disease specialists convened to formulate a treatment strategy. The immediate priorities included urgent decompression of the spinal cord to prevent further neurological deterioration, maximum safe resection of the parasitic lesion, and stabilization of the structurally compromised spine.

The patient was started on high-dose Albendazole (15 mg/kg/day) preoperatively to reduce the viability of the hydatid cysts and minimize the risk of dissemination during surgery. She subsequently underwent emergency posterior decompression and stabilization via a wide C7 to T4 laminectomy.

Intraoperative findings revealed complete obliteration of the epidural space by numerous grape-like hydatid vesicles infiltrating surrounding tissues. Careful debulking of both epidural and paravertebral components was undertaken, with hypertonic (3%) saline irrigation used as a scolicidal agent to inactivate any residual parasitic material. Due to the extensive invasion and destruction, radical excision was not feasible.

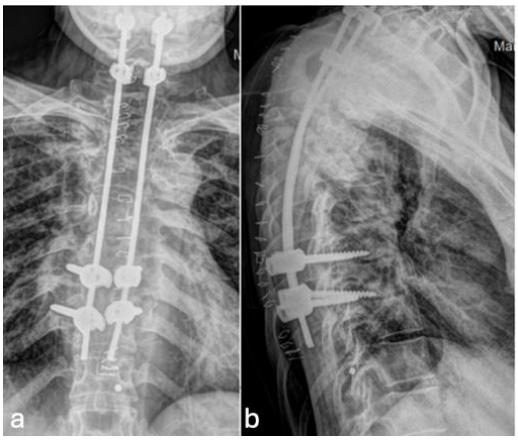

Figure 3: Anteroposterior(a) and lateral(b) radiographs of postoperative appearance of the cervicothoracic spine demonstrating posterior instrumentation in satisfactory alignment, with no radiographic evidence of hardware failure, displacement, or loosening on standard projections

To address the profound spinal instability, a long-segment posterior fixation was performed. Pedicle screws from C6 to T6 were placed and connected with rods to create a stable construct bridging the destroyed vertebral segments. Pathological analysis of the excised tissue confirmed the diagnosis of spinal echinococcosis.

Following surgery, the patient underwent a postoperative spinal radiograph (Fig 3), which demonstrated that the vertebral arthrodesis was correctly positioned, with all screws and rods in proper alignment. There were no signs of hardware loosening, malposition, or secondary fractures. The vertebral alignment was maintained, and no immediate postoperative complications, such as implant failure or local infection, were evident on imaging.

The postoperative course was uneventful. The patient continued long-term Albendazole therapy, with regular liver function monitoring. Unfortunately, despite effective decompression and stabilization, the severe preoperative neurological deficit persisted, and the patient remained permanently paraplegic at the T6 sensory level.

After hospital discharge, she was referred to a specialized rehabilitation center, where she began an intensive program focusing on wheelchair mobility and activities of daily living.

For follow-up, one year after treatment, the patient first underwent a spinal CT scan (Fig4), which provided detailed evaluation of the bone structures. The CT confirmed a reduction in the size of the original spondylocystic lesion and allowed assessment of the vertebral arthrodesis and its stability, with no signs of loosening or hardware complications.

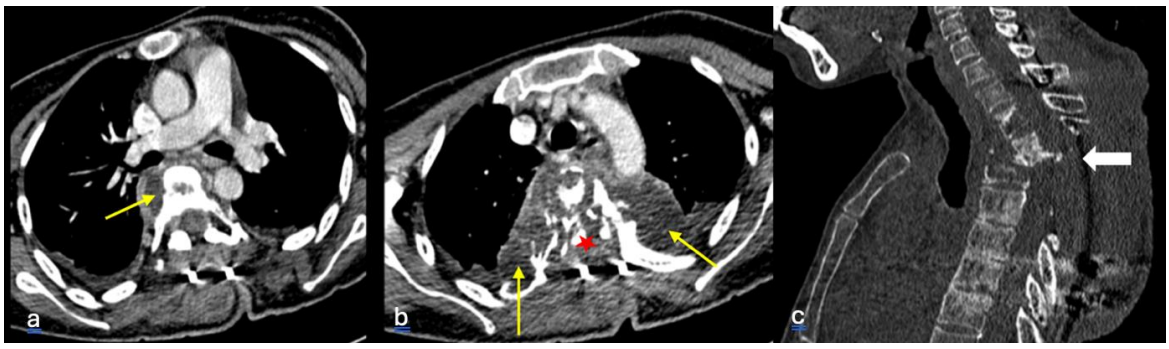

Figure 4: Follow-up CT scan of vertebral hydatidosis demonstrating persistence of a cystic-solid lesion with intramedullary extension (red asterisk in b, axial soft tissue window). The scan also reveals pleural and paravertebral cystic formations (yellow arrows in a and b, axial soft tissue window) and vertebral body collapse (white arrow in c, sagittal bone window)

Subsequently, a medullary MRI was performed to better assess the soft tissue and intramedullary extension of the disease. MRI revealed persistence of the vertebral hydatid process, with multiple cystic formations extending into the mediastinum and pleura, as well as residual intramedullary involvement (Fig5). These findings provided a comprehensive evaluation of both osseous and soft tissue disease progression while confirming the stability of the spinal fixation.

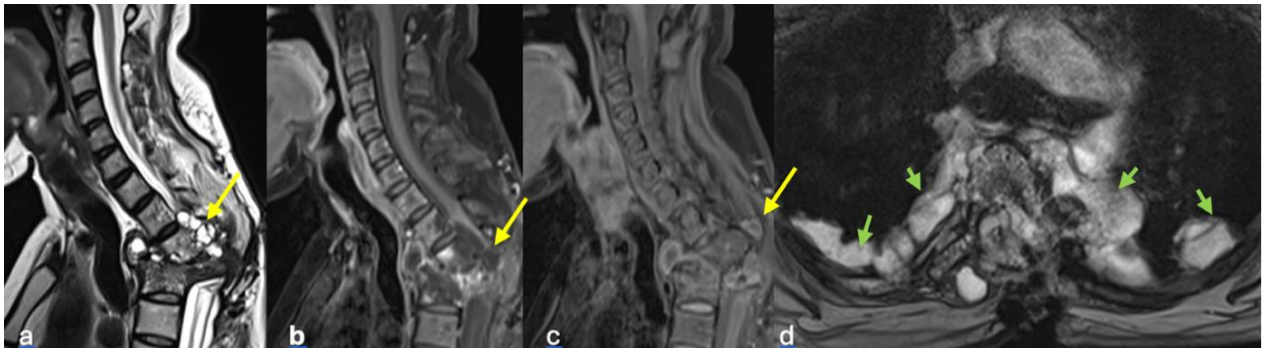

Figure 5: Follow-up MRI showing the persistence of a multiloculated solid-cystic lesion (yellow arrows) centered at D2–D3 with heterogeneous enhancement. Sagittal T1 (b, c) and T2 (a) weighted images, pre- and post-gadolinium, and axial post-contrast T1 sequence (d) reveal vertebral body and disc lysis, posterior wall retropulsion, spinal cord compression, and mediastinal-pulmonary extension (green arrows)

In summary, although incomplete excision the reduction in size of the hydatid lesion and the stability observed after one year of follow-up are encouraging signs. Rigorous monitoring, including regular clinical and radiological examinations, is essential to detect any unfavorable progression.

DISCUSSION

Hydatid disease is relatively common in the Mediterranean, the Middle East, Central Asia, East Africa, and some areas of South America. Sheep are the intermediate host of the tapeworm Echinococcus granulosus. The definitive hosts are dogs, foxes, and other carnivores. The tapeworm lives in the small bowel and infected ova are shed in the feces. When ingested by humans or sheep, the larvae enter the circulation, reaching the liver and lungs as a first filter, but some occasionally pass through those organs to reach the bones[5].

Consistent with the literature, our patient presented with features typical of advanced spinal hydatidosis [2]. The thoracic spine is the most commonly affected segment, accounting for approximately 50% of cases, and the development of neurological deficits[6] is frequently the event that precipitates medical consultation [1,7] . The pathophysiology of the disease explains the extensive destruction seen on imaging. Unlike visceral hydatid cysts, osseous lesions lack a limiting pericyst, allowing for uninhibited, infiltrative growth that mimics a malignant tumor—a behavior that has earned it the name "the white cancer"[8,9]. This results in the characteristic radiological findings of extensive osteolysis without reactive sclerosis, as was clearly demonstrated in our patient's CT scans.

A crucial aspect of this case was the initial diagnostic uncertainty. The radiological presentation of multi-level vertebral destruction with large paravertebral collections made spinal tuberculosis (Pott's disease) a primary differential diagnosis[4], a common and important pitfall in endemic regions. This highlights a critical teaching point: while advanced imaging like CT and MRI is invaluable, the features can overlap. In such scenarios, obtaining a tissue diagnosis is paramount. Although percutaneous biopsy of a hydatid cyst carries a theoretical risk of anaphylaxis and dissemination, its role becomes indispensable when the diagnosis is unclear and alternative pathologies must be excluded [5]. The negative Quantiferon test followed by a conclusive biopsy in our case exemplifies this necessary diagnostic pathway.

The management strategy employed aligns with the three fundamental goals of treatment for vertebral hydatidosis: urgent decompression of neural elements [10], maximal safe cytoreduction of the parasite, and stabilization of the spine [8]. The high rate of recurrence, reported to be as high as 30-40%, necessitates a combined medical and surgical approach [1]. Surgery alone is insufficient. The administration of pre- and post-operative Albendazole, combined with the intraoperative use of scolicidal agents like hypertonic saline [2], is the current standard of care aimed at sterilizing the surgical field and preventing future growth. Given the extensive destruction across three vertebral levels, a wide laminectomy and long-segment posterior fixation was the most appropriate strategy to achieve both decompression and immediate, rigid stability.

Ultimately, the outcome for our patient is sobering and speaks to the disease's grim prognosis once significant neurological injury has occurred. While the surgical and medical treatment successfully controlled the local disease and stabilized her spine, it could not reverse the spinal cord damage that had already taken

place. This case powerfully reinforces the concept that for vertebral hydatidosis, the window for effective intervention is often before the onset of major neurological deficits. The key to improving outcomes lies not just in refining treatment techniques, but in fostering a high index of suspicion that leads to earlier diagnosis in at-risk populations.

CONCLUSION

Spinal hydatidosis is a rare but severe form of echinococcosis. Early diagnosis is crucial, as delayed detection often results in irreversible neurological damage. MRI plays a key role in diagnosis, and management requires combined surgical decompression, spinal stabilization, and long-term antihelminthic therapy. Prognosis depends mainly on the degree of neurological impairment at presentation.

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