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Isolated Extradural Hydatid Cyst of the Dorsal Spine: A Case Report

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Abstract | **Case Report**

Vertebro-medullary hydatidosis is a rare disease, and isolated extradural spinal involvement is exceptional, particularly in children, making diagnosis and management challenging. We report the case of a 12-year-old child from a rural area, with no significant medical history, who presented with progressive dorsolumbar pain for three months, followed by heaviness of both lower limbs and sphincter dysfunction. Clinical examination revealed a dorsal spinal cord compression syndrome. Magnetic resonance imaging (MRI) of the dorsolumbar spine demonstrated a well-defined, bilobed anterior extradural cystic lesion, predominantly right-sided, centered at T12, appearing hypointense on T1-weighted images and hyperintense on T2-weighted images, causing posterior displacement and compression of the spinal cord without associated bone involvement. Computed tomography confirmed an intraspinal epidural lesion without vertebral destruction. Laboratory tests were unremarkable except for mild eosinophilia, and no other hydatid localizations were identified. An emergency surgical decompression was performed via posterior laminectomy with complete excision of the cyst. Histopathological examination confirmed the diagnosis of a hydatid cyst. Postoperatively, the patient received albendazole therapy according to the national protocol. The postoperative course was favorable, with complete neurological recovery after 11 months of follow-up. This case highlights the rarity of primary isolated extradural spinal hydatid cysts, the crucial role of MRI in diagnosis, and the importance of early combined surgical and medical treatment, which offers a good neurological prognosis.

Keywords: Vertebral-Medullary Hydatidosis, Extradural Hydatid Cyst, Spinal Cord Compression, Spinal MRI, Decompressive Surgery.

INTRODUCTION

Vertebro-medullary hydatidosis is a rare condition, characterized by the diversity of its clinical symptoms and the diagnostic and therapeutic challenges it presents. The primarily extradural location is exceptional, posing differential diagnostic problems even in hydatid endemic areas.

CASE REPORT

Child A.A., aged 12, living in a rural area, with no significant medical history, presented with progressive dorsolumbar pain three months prior to hospitalization. A few days later, this was followed by heaviness in both lower limbs and sphincter dysfunction. Clinical examination revealed a dorsal spinal cord compression syndrome. A dorsolumbar MRI performed with sagittal T1- and T2-weighted sequences (Fig. 1), coronal T2-weighted sequences (Fig. 2), and axial T2-weighted gradient echo sequences (Fig. 3) showed an anterior extradural lesion, predominantly on the right, displacing the flattened spinal cord posteriorly. The lesion was bilobed, well-defined, hypointense on T1-weighted images, hyperintense on T2-weighted images, and centered on T12. It was causing compression of the dural sac with perilesional spinal cord compression. A CT scan of the thoracolumbar spine, performed to investigate for bone involvement, revealed a bilobed, intraspinal lesion centered on T12, measuring 25 x 13 mm, filling the epidural space, displacing the dural sac without invading it, and showing fine peripheral enhancement with contrast (Figs. 4, 5, and 6). Standard radiographic examination of the spine showed no bone lesions. The chest X-ray was unremarkable. Laboratory tests were normal except for mild eosinophilia. Abdominal ultrasound was normal. The differential diagnoses included an extradural spinal abscess, a dysembryoplastic lesion, or hydatid disease. An emergency surgical approach was performed, consisting of a posterior decompression laminectomy centered on T12, which allowed for complete resection of the cyst

that was displacing and stretching the right nerve root posteriorly and the dural sheath to the left. The cyst's hydatid nature was presumed and confirmed by final histological examination. The immediate postoperative course was uneventful. Medical treatment with albendazole was prescribed according to the national standard protocol, at a dose of 400 mg per day for 28 days, repeated three times with 10-day breaks in between, with monthly monitoring of liver function. Complete recovery of the deficit was observed after 11 months of follow-up.

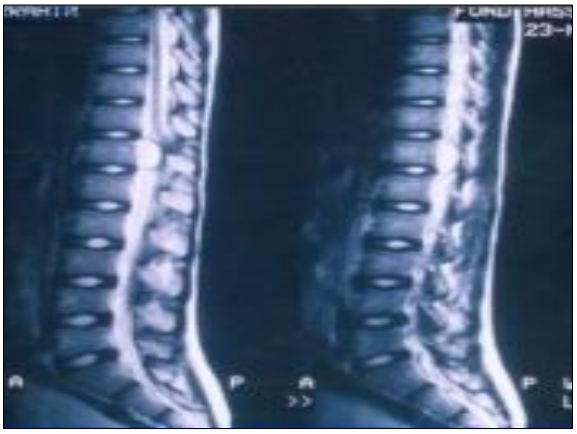

Figure 1: Sagittal T2-weighted sequence images showing an anterior, hyperintense extradural process centered on T12, causing compression of the dural sac with peri-lesional spinal cord injury

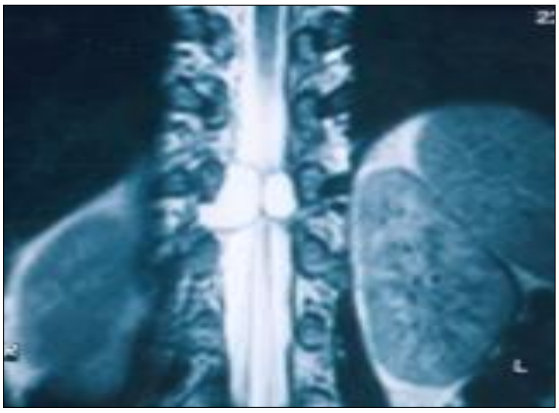

Figure 2: Coronal T2-weighted sequence shows a predominantly right-sided, bilobed, well-defined, hyperintense extradural process centered on D12

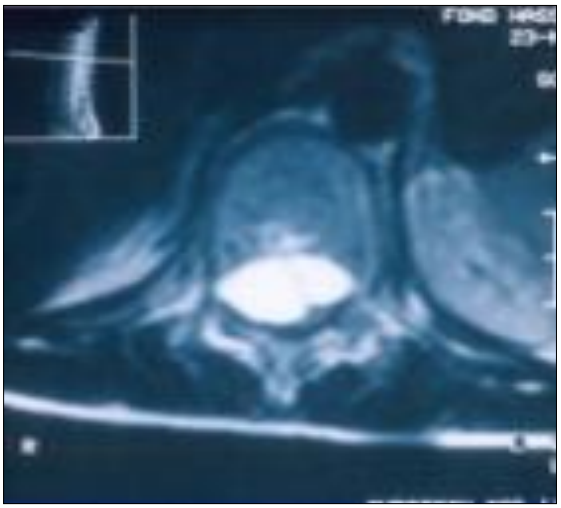

Figure 3: Axial gradient echo scan shows an anterior extradural process predominantly on the right, displacing the flattened spinal cord posteriorly

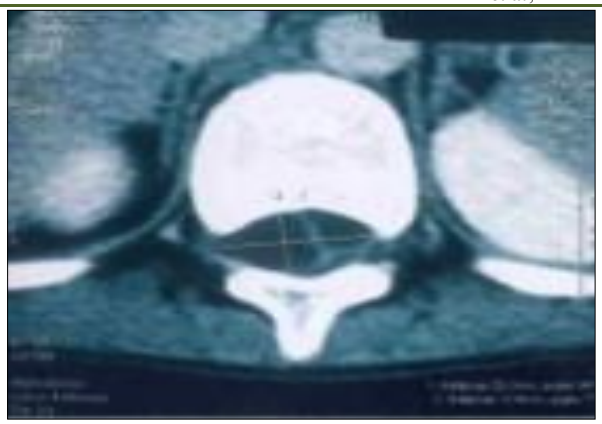

Figure 4: Axial section in parenchymal window after injection of contrast product shows an intracanalicular lesion process centered on D12, bilobed, pushing back the dural sheath without invading it, taking up the contrast product thinly at the periphery

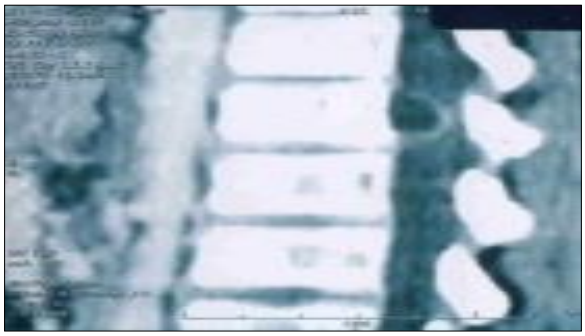

Figure 5: Sagittal reconstruction shows an anterior, hypodense, intracanalicular lesion process, centered on D12

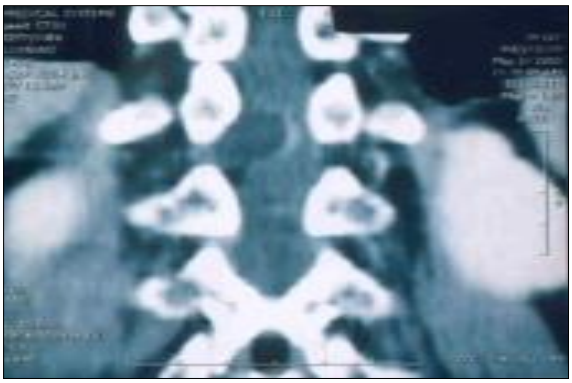

Figure 6: Coronal reconstruction shows an intracanalicular lesion process lateralized to the right

DISCUSSION

Vertebrospinal hydatidosis remains the most frequent and serious manifestation of osseous hydatidosis [1-3]. It accounts for approximately 40 to 50% of the latter and 1% of all hydatid disease locations [4-6]. The lesions are primarily somatospinal and epidural, extending secondarily into the spinal canal [4-7]. Primary intraspinal hydatidosis remains rare, even exceptional, and is confined to children and young adults [4-8], as in our case. The location is generally thoracic, rarely lumbar, and exceptionally cervical.

From a pathophysiological standpoint, the route of inoculation is hematogenous, as has been demonstrated for thoracic locations [4-6]. The presence of a portovertebral shunt may also explain the pathogenesis of its forms [9-11]. Clinically, the condition presents as a rapidly progressing neurological deficit syndrome, which allows for the distinction between primary intraspinal hydatidosis and common vertebral hydatidosis [4-12]. The spinal cord is a poorly vascularized area with no collateral vascular supply; it is therefore highly susceptible to ischemia. The development of the cyst and the resulting tension in the cerebrospinal fluid lead to early clinical manifestations with rapid onset and progression [13, 14]. Radiological diagnosis remains crucial given the nonspecificity of the clinical signs. Standard radiographs may be normal, as in our case, or show indirect signs of an intracanalicular mass, sometimes septated, compressing or displacing the

spinal cord, unaffected by contrast injection, located extradurally, without associated bone involvement [4-12]. MRI is increasingly replacing myelography-CT due to its multiplanar approach and excellent tissue contrast resolution. It now allows for careful examination of the canal contents and enables assessment of the relationship of hydatid cysts to the dural sheath and the degree of spinal cord injury. A solitary extradural hydatid cyst appears as a cystic mass, most often oval, hypointense on T1-weighted sequences, and hyperintense on T2-weighted sequences, depending on the intensity of the cerebrospinal fluid [9-16]. This appearance is related to the viability of the parasite, while its death is accompanied by a relative decrease in signal intensity [9-16]. On T1-weighted sequences after gadolinium injection, the cyst does not enhance, except sometimes peripherally [4-18]. In cases of multivesicular cysts, the septa appear intermediate on T1 and hypointense on T2 [4, 5]. A systematic search for other hydatid cysts is necessary, based on non-invasive investigations: ultrasound, CT, and MRI. Laboratory tests using immunological techniques specific to hydatid disease confirm the diagnosis [4-18]. Treatment is both medical and surgical. The surgical procedure consists of en bloc resection of the cyst via a posterior approach and decompressive laminectomy of the spinal cord or dural sheath and nerve roots. Dissection must be meticulous to avoid rupture or fissuring of the cyst. The prognosis for primary intraspinal forms is better than for vertebrospinal cord involvement, provided that treatment is initiated early.

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