This report is presented to demonstrate the image findings of a patient with a recent onset of progressive spinal cord compression caused by a giant arachnoid cyst of the thoracolumbar spine. The patient presented with gradual onset paraparesis and the diagnosis of arachnoid cyst was made on MRI. Surgery was successful with respect to in-toto removal of the cyst, following which there was reversal of cord compression and symptoms. Histological diagnosis was of an arachnoid cyst.

**Keywords**: arachnoid cyst; extradural; spinal; magnetic resonance imaging.

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**INTRODUCTION**

Spinal arachnoid cysts are relatively uncommon lesions which may be intradural or extradural, with the intradural variety being rare (7).

Spinal arachnoid cyst accounts for approximately 1% of primary spinal cord tumours; however, the widespread use of magnetic resonance imaging (MRI) has been associated with an increasing number of reported cases in recent years. Although magnetic resonance imaging (MRI) is currently considered the diagnostic procedure of choice (7), some arachnoid cysts have been reported to elude diagnosis by this modality because of the similar signal intensity of the cyst and the subarachnoid space (12).

**CASE REPORT**

The patient was an 18-year-old girl who had a chief complaint of gradual onset paraparesis over a period of 4 months. There was associated back pain and heaviness of her lower limbs on walking. Occasionally she had tingling and numbness in the lower limbs. There were no bowel and bladder complaints.

She had a normal birth history with no gross external congenital anomaly; there had been no known growth abnormalities. There was also no significant family history.

Neurological examination revealed power of 3/5 in lower limbs with hyper-reflexia and upgoing plantar reflexes. Position sense was impaired in both the lower limbs. Defecation and micturition were normal. Symptoms did not alter on postural changes. There was no history of trauma or spinal anaesthesia. She had never used medications which significantly affect haemostasis. Meticulous haematological assessment revealed no defects of haemostasis.

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**Correspondence**: Dr Muddassir Rashid, Department of Radiodiagnosis, Jawaharlal Nehru Medical College Hospital, AMU, Aligarh, India.

**E-mail**: beigmuddassir@yahoo.com or beigmuddassir@gmail.com

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Muddassir Rashid, MD, Resident.
Anjum Syed, MD, Consultant.
Ibne Ahmad, MD, Professor and Chairman.
Ekramullah, MD, Professor.

Department of Radiodiagnosis, Jawaharlal Nehru Medical College Hospital, AMU, Aligarh, India.
An anteroposterior radiograph of the thoracic and lumbar vertebrae showed increased interpedicular distance, while a lateral radiograph revealed increased anteroposterior diameter of the spinal canal. There was also posterior scalloping of the dorsal part of the vertebrae (fig 1). MRI of the thoraco-lumbar spine (1.5 T Siemens Avanto) disclosed a sharply-defined tumour of low signal intensity on T1-weighted images extending from D8-L3, compressing the distal spinal cord and conus. T2-weighted images demonstrated the same tumour with signal intensity equal to the cerebrospinal fluid (figs 2 & 3). MR myelography was done which revealed the extent of tumour sleeves through the neural foraminae along the nerve roots (fig 4). The diagnosis of an arachnoid cyst was made and surgery was advocated. Whole spine screening was done and there were no signs of intramedullary arachnoid cyst or coexistent syringomyelia, diastematomyelia, tethered cord, kyphoscoliosis, fibrolipoma, Klippel-Feil or other spinal anomalies.

Laminectomy was performed from the D11 to L3 vertebrae which revealed a colourless fluid-filled thin-walled extradural cyst compressing the dural sac. The cyst was seen extending into the adjacent neural foramina along the root sleeves of the spinal nerves at multiple levels. According to the classification of Nabors et al (9) the lesion was therefore diagnosed as a large extradural meningeal cyst Type II, previously termed as Tarlov’s ‘perineural cyst’ or ‘spinal nerve root diverticula’. The arachnoid cyst was identified and excised, the dura mater sutured, and the cyst removed. Histopathological examination of the cyst wall showed layered collagenous fibers and epithelial tissue with flat lining cells, confirming arachnoid cysts.
On post-operative MR-scanning a normal spinal cord was seen with a widening of the dural sac at the level of the operation. The patient improved gradually and at follow-up one year later, neurological examination was completely normal.

DISCUSSION

Arachnoid cysts are benign developmental cysts (16). The spinal arachnoid cysts may be intradural or extradural and are composed of normal or slightly thickened arachnoid, which is filled with a clear, colorless fluid that is most likely normal cerebro-spinal fluid (16). Arachnoid cysts may coexist with neural tube defects (14). Extradural cysts are believed to arise from defects in the dura through which arachnoid herniates; whereas the intradural cysts probably result from an alteration in the arachnoid trabeculae (14).

ACs occur as single or multiple cysts, more frequently extradurally than intradurally, are located in 80% in the thoracic, in 15% in the cervical and in 5% in the lumbar spine, affect men and women equally and are most commonly encountered at an age between 30 and 50 years (11).

ACs are located posterior to the thecal sac and only rarely anteriorly (20). The cause of these cysts remains obscure; some are thought to be congenital malformations, especially in young children without evidence of healed meningitis or back injury (4). An association between spinal arachnoid cysts and neural tube defects has also been reported. Rabb et al (14) described 11 paediatric patients with spinal arachnoid cysts, among whom six had meningomyelocele. These patients with meningomyelocele seem particularly prone to having anteriorly situated arachnoid cysts. The authors pointed out that the cysts developed as a result of the abnormal CSF flow and extensive arachnoid adhesions found in their patients. Unless congenital in origin, ACs may be secondary to trauma, infection, subarachnoidal bleeding or they may be idiopathic (20).

It seems that the rare anterior cysts are of traumatic or inflammatory origin, whereas posterior ones probably originate from arachnoid trabeculae or the septum posticum of Schwalbe (20).

The clinical picture depends on the level of the compression of the cord. Symptoms caused by ACs may fluctuate, mimic other disorders such as multiple sclerosis (11), change upon the patient’s position (valve mechanism of the cyst (11,20) which was most probably the cause of acute deterioration in our patient) and increase during the Valsalva manoeuvre (20). Most frequently, patients complain of spinal pain, radicular and/or myelopathic syndromes. Neurological signs include disturbances of sensation, paresis, paraparesis, disturbed defecation and micturition and gait impairment due to ataxia (11,20).

A plain roentgenogram is usually normal, but in some cases enlargement of the spinal canal at the cyst level and occasionally scoliosis or kyphosis coexist (17). Prior to the advent of MRI, correct diagnosis was obtained by myelography and CT myelography, invasively (3). However, some authors pointed out that the most important preoperative diagnostic procedure is myelography performed with the patient in both the supine and prone positions (1,6). MRI demonstrated the extent, size and nature of the cysts. The arachnoid cysts have been
described as isointense with CSF on T1-weighted images. Cysts with higher protein content result in slightly higher signal intensity than CSF on T1-weighted images. T2-weighted MRI demonstrates a heterogeneous signal intensity, depending on the flow effect in the cyst fluid. MRI is useful to assess the size, nature and extent of the cystic lesion as well as the mass effect on the cord and associated signs of meningeal inflammation. Moderate to severe arachnoiditis can be diagnosed with even non-enhanced MRI. Increased CSF signal intensity on T1 weighted images leading to loss of CSF-cord interface is strongly suggestive of the arachnoiditis.

The differential diagnosis on imaging includes other intradural cystic lesions like dermoids, epidermoids, hydatidosis and cysticercosis. Dermoids can be differentiated as they are most commonly seen in the midline and reveal few fat signal intensity areas; however the cystic lesions seen in our patient revealed CSF intensity. Epidermoids are usually seen in children and are mostly located in the lumbar spine. They are lobulated lesions with minimally different signal intensity as compared with adjacent CSF. In a rare case reported by Ciftci et al., multiple intradural cysticercosis was found in the basal cistern, cisterna magna, and cervical subarachnoid space which was isointense with cerebrospinal fluid both on T2 and T1 weighted images. Intradural hydatid cysts have been reported very sporadically in the English literature. The case reported by Pushparaj et al. of an intradural hydatid cyst, in the dorsal spine revealed a cystic lesion with signal intensity similar to CSF on MRI.

Complete excision is the treatment of choice and in those cases where scarring, adherence to the cord, or ventral cyst location makes total excision impossible, wide fenestration of the cyst to allow communication with the subarachnoid space is the next best treatment. Other methods are stenting or cyst-to-peritoneum/-atrium shunting. Simple needle aspiration is inadequate unless there is prolonged cord compression with subsequent atrophy or myelomalacia.

To our knowledge, this is the most extensive arachnoid cyst reported in the lumbar region. MRI is the diagnostic procedure of choice for detecting the intraspinal and especially the intramedullary cysts. The exact location, extent, and severity of cord compression are easily visualised, and other diagnostic possibilities, such as a tumour, demyelinating disorders, and intrinsic spinal cord lesions, are excluded. In this patient, MRI clearly revealed the cyst, and immediate operative treatment was possible.

REFERENCES


