

Accidental Rupture Of A Spinal Extradural Arachnoid Cyst During Central Neuraxial Blockade

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Abstract

Intraspinal extradural arachnoid cysts are uncommon expanding lesions in the spinal canal and may communicate with the subarachnoid space. Most arachnoid cysts are asymptomatic and detected incidentally on MRI or myelography. They commonly occur in the middle or lower thoracic spine, less commonly in the lumbar region and lie posteriorly or posterolaterally in the spinal canal. Extradural arachnoid cysts pose a serious challenge to the anaesthetist especially during central neuraxial blockade. I would like to report a case of a young man posted for an emergency open reduction for fracture tibia. Combined spinal epidural anaesthesia was planned. Two attempts of epidural needle insertion through the midline led to leak of clear colourless viscous fluid. Later, spinal anaesthesia was performed through the paramedian approach. The patient had a stable post operative course and recovered normally. The patient was later diagnosed with an extradural arachnoid cyst in the midline at the lumbar level.

INTRODUCTION

Intraspinal extradural cystic lesions are rare and may include arachnoid cyst, synovial cyst, ganglion cyst, Tarlov's perineural cyst, meningocoele, dermoid cyst, hydatid cyst, neuroma with cystic changes, post-traumatic meningeal diverticuli and mesothelial cyst¹. The classification of these cysts is difficult clinically but radiological and histopathological studies may aid in the diagnosis.

Asymptomatic cysts can be encountered during central neuraxial blockade and may lead to serious postoperative complications due to their close proximity to the spinal cord and communication with the subarachnoid space.

Here, we describe a case of a man who was incidentally diagnosed with an extradural arachnoid cyst during central neuraxial blockade.

CASE REPORT

A 38 year old young healthy man presented to the emergency department with a history of road traffic accident leading to an open fracture of left tibia. The patient had no past surgical or medical history. He also denied any history of trauma to the back, backache or neurological symptoms.

Vitals: Height-170cm, Weight- 70 kg, Pulse- 90/min regular, BP- 130/80 mmHg. Airway: Mouth opening adequate,

Malampatti class I. General and systemic examination did not reveal any abnormality.

After initial resuscitation, the patient was shifted to the operation theatre for open reduction of tibial fracture. A combined spinal epidural anaesthesia was planned.

The procedure was explained and an informed consent was taken. An 18G IV cannula secured and ECG, BP and SpO₂ monitors were attached.

With the patient in sitting position, L3-L4 interspace was infiltrated with 2% lignocaine in the midline. Under all aseptic precautions, a 16G Tuohy's needle was gradually advanced using loss of resistance to air technique. Due to sudden gush of clear, colourless and viscous fluid, the Tuohy's needle was withdrawn. During the procedure, no give way sensation to dura was encountered and the Tuohy's needle was inserted upto a depth of just 2.5 cm. Later, the procedure was repeated in the L2-L3 interspace through the midline after local anaesthetic infiltration. Again, as the Tuohy's needle was inserted just beyond 2.5cm, colourless viscous fluid flowed out through the needle. The procedure was abandoned and the patient's vitals were carefully observed. The decision was made to perform the case under spinal anaesthesia. After local anaesthetic infiltration, a 25G Quincke's needle was inserted through the paramedian position in the L2-L3 space. An initial loss of resistance to

ligamentum flavum, followed by loss of resistance to dura was distinctly felt. After confirming a free flow of CSF, 3ml of 0.5% isobaric bupivacaine with 25 micrograms of fentanyl was injected in the subarachnoid space. The subarachnoid space was reached at a depth of approximately 4.5cms from the skin. It was also noticed that the fluid obtained through the first two midline attempts was of a different consistency as compared to the cerebrospinal fluid obtained in the third attempt. The patient was immediately made supine and his heart rate, blood pressure and respiration were carefully observed. The patient achieved a complete motor block and the level of sensory block was T10.

Intraoperatively, the patient was comfortable and haemodynamically stable. The surgery was completed successfully. The postoperative period was uneventful and the patient recovered normally without any headache or neurological complications.

Later, the patient was referred to neurosurgery for further evaluation. MRI confirmed an extradural arachnoid cyst in the midline extending from T12 to L3. The cyst had no communication with the subarachnoid space. There was no evidence of cord compression.

As the patient was asymptomatic, it was decided to review the patient annually with a repeat MRI scans. The patient was informed about this condition and was advised to seek urgent neurosurgical opinion in case of any neurological symptoms.

DISCUSSION

Extradural arachnoid cysts usually occur in the middle or lower thoracic spine, less frequently in the lumbar region². These lesions are twice as common in males and usually present in the second decade. Lumbar and sacral cysts usually present in the third and fifth decades. These lesions often arise dorsally and can partially protrude into the adjacent neural foramen. A single cyst can extend over several spinal segments, or multiple cysts with separate dural defects and communicating pedicles can compose one lesion. They usually communicate with the subarachnoid space through a small dural defect and stalk^{3,8}.

Arachnoid cysts are usually an incidental finding on imaging. Arachnoid cysts may cause symptoms by compressing the spinal cord or nerve roots. The mechanism through which the spinal cord can be compressed by such cysts is unknown, but many authors have speculated that a

one-way valve mechanism causes intermittent increased pressure within the extradural cyst, leading to expansion and compression⁸. The location of the cyst within the spine and the severity of spinal cord and root compression affect the clinical presentation. Spastic tetraparesis and impaired sensory levels are indicative of cervical cysts, whereas Horner syndrome is a common presentation in patients with cysts that occur lower in the cervical spine. Patients with thoracic cysts tend to present with progressive spastic paraparesis, but back pain is generally uncommon; conversely, patients with lumbar and lumbosacral cysts classically present with low-back pain, radiculopathy, and bowel and bladder dysfunction². Overall, motor weakness is usually more predominant than sensory loss. Symptoms can be intermittent and exacerbated by Valsalva manoeuvres or gravitational positional forces².

Arachnoid cysts may be classified as primary or secondary. Primary cysts most likely arise during development and may expand throughout life and progressively displace spinal tissue. They can develop due to arachnoid herniation through a congenital defect in the dura at or near a nerve root or in the midline. Secondary cysts follow a variety of spinal insults including head injury, haemorrhage, chemical irritation, meningitis, tumors and iatrogenic such as injected contrast media or anaesthetics or from the intraoperative contaminants of fibrin glue⁶. Nabors et al. classified spinal cysts simply into three major categories on the basis of surgical, radiological, and histological criteria. Type I are spinal extradural meningeal cysts without spinal nerve root fibers (two subclassifications are defined as Type IA-extradural meningeal cyst and Type IB-sacral meningocele). Type II are spinal extradural meningeal cysts with spinal nerve root fibers (Tarlov cyst), and Type III are spinal intradural cysts, which are intradural spinal arachnoid cysts.^{2,3,4} Intradural spinal arachnoid cysts should be differentiated from Tarlov cysts, which are extradural meningeal dilatations of the posterior spinal nerve root sheath. They have potential but not actual communication with the spinal subarachnoid space and usually occur distal to the junction of the posterior nerve root and dorsal root ganglion⁴.

Radiographs of the spine usually show bone erosion with widening of the canal, erosion of pedicles, foraminal enlargement, and scalloping of the vertebral bodies or the sacrum. MRI is the diagnostic procedure of choice as it is noninvasive and can demonstrate the cyst nature, exact size, and anatomic relationship with the spinal cord. Cinematic

MRI and CT myelography are used to evaluate the presence of communication between the cyst and subarachnoid space⁴.

For incidentally discovered spinal arachnoid cysts that cause no symptoms, surgery is not recommended. No correlation exists between the size of a cyst and the need for treatment⁶. Yearly imaging should be done to detect any new abnormality and to

determine whether the cyst is truly benign. If symptoms arise, reevaluation of the cyst with MRI should be immediately undertaken.

Symptomatic cysts should be surgically resected. If complete resection is impossible, fenestration of the cyst wall, drainage, or shunting may relieve symptoms⁶.

DIFFERENTIAL DIAGNOSIS OF CYSTS IN THE SPINE

Cystic loculations in adhesive arachnoiditis (often multiple; other features of arachnoiditis may be evident).

Meningoceles (most common in the lumbosacral region; readily identified in association with spina bifida, rare in adults).

Cysts of dorsal nerve root ganglia (observed in 8-24% of autopsies)

Extradural ganglion and synovial cysts (posterolateral location, communication with the facet joint, presence of synovial lining in synovial cysts)⁵.

Tarlov cysts (perineural cysts of sacral or coccygeal nerve roots arising between the layers of connective tissue covering the nerve roots; communication between the cyst and subarachnoid space may be present).

ANAESTHETIC IMPLICATIONS

Arachnoid cysts may be associated with neural tube defects like syringomyelia, dorsal kyphosis and ankylosing spondylitis. Arachnoid cysts have also been described in

patients with Marfan syndrome, neurofibromatosis, autosomal dominant polycystic kidney disease, and generalized connective tissue disorders of uncertain type⁴.

Trauma may lead to intracystic haemorrhage or hematoma leading to increased intracystic pressure causing neurological symptoms. The patients may present with a backache following central neuraxial blockade and the radiological findings may be confused with an epidural haematoma⁷.

CONCLUSION

Extradural arachnoid cyst is an uncommon expanding lesion in the spinal canal which may communicate with the subarachnoid space. This may be accidentally encountered during central neuraxial blocks.

We describe one case of an asymptomatic extradural arachnoid cyst of thoracolumbar region.

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