Cord Herniation through the Site of Undiagnosed Thoracic Dermoid Tumour during Spinal Anaesthesia; Report of a Case and Describing Ways to Avoid

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Abstract: Spinal anaesthesia (SA) is one of the most prevalent types of anaesthetic procedures. There are very few reports of cord herniation through the site of spinal canal stenosis due to tumour. A 33-year-old female presented with acute paraparesis after spinal anaesthesia for caesarean section. Magnetic resonance imaging (MRI) revealed an intradural mass from posterior of T6 to T8-T9 interface. We operated the patient and after laminectomy of T6 to T9, dermoid tumour containing hairs was totally resected and cord was completely decompressed. After 6 months, the patient is without any neurological deficit. Puncturing the dura with cerebrospinal fluid (CSF) in the presence of an extramedullary mass could cause cord herniation through the blockade. In these cases, awareness about related signs even in absence of symptoms or complaints could help us to prevent post-SA neurological deficit.

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Introduction

Spinal anaesthesia (SA), so called spinal block, is one of the most prevalent types of anaesthetic procedures, sometimes being superior to the general anaesthesia (Yüksek et al., 2020). In several comparable studies, SA had some superiority to the general anaesthesia (GA) in safety and in possible procedure-related complications (Visser et al., 2009; Mortazavi et al., 2022). Because of prevalent usage of this technique, knowing possible complications and pitfalls are crucial. In the literature, there are many reports of neurological deficits after SA and all of them discussed etiologies, direct toxic effect of injected drug to the spinal column, compressive hematoma in site of puncture, etc. (Nicholson and Eversole, 1946). However, very few reports of cord herniation through the site of spinal canal stenosis due to tumour could be found (Doh et al., 2001; Krishnan and Roychowdhury, 2013). Considering etiologies of these complications, it is important to know what pre-procedural data and exams could be helpful to prevent them.

Here, we present a rare case of thoracic spine dermoid cyst without any previous alarming symptoms that after SA, caused significant neurological deficits.

Case report

A 33-year-old female presented with acute paraparesis after spinal anaesthesia for caesarean section (C/S) without any previous complaints or problems, in October 2021. The patient was a primipara and because of obstetric problems she underwent C/S one week ago. She was unable to walk alone. She had also some degree of sphincter disturbance as difficulty in urination and constipation concomitant with distal paraesthesia of lower limbs. On neurological examination, she had symmetrical proximal and distal weakness of both lower limbs, 3 from 5 based on muscle strength grading (Naqvi and Sherman, 2020). We found decreased sensation below umbilicus, based on pinprick test (two point discrimination). Sphincter tone was normal. Examination revealed bilateral hyperreflexia with positive Babinski sign. After diagnosis of paraparesis in previous center, for excluding complications related to site of the intervention (lumbar puncture – LP), the patient underwent an emergency lumbosacral magnetic resonance imaging (MRI) that was normal (Figure 1A). Then after performing thoracic imaging, it revealed an intradural mass from posterior of T6 to T8-T9 interface (Figure 1B–G).

We decided to operate the patient in prone position. During T6 to T9 laminectomy, we found a stalk was invaginated into T9 lamina and then a bundle could be seen from it to the midline skin at level of T10. The stalk was connected to a dural defect at T9 level (Figure 2A). After opening of the dura, we could see a large yellow intradural mass containing of hairs that compressed thoracic cord to the anterior and left side (Figure 2B and C). The mass was debulked internally and its thin capsule was resected, as much as possible, excepting the pial interface with cord due to adherence (Figure 2D). We found also some osseous elements in the mass. Finally, dura was sutured in watertight manner and tract to skin was completely

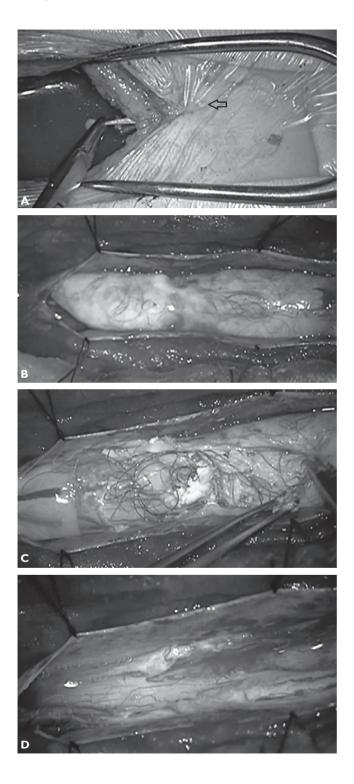


Figure 1 – Lumbosacral magnetic resonance imaging (MRI) (A) of patient showed no pathology but a complete cerebrospinal fluid (CSF) block can be seen in thoracic magnetic resonance myelogram (B, C). An hyperintense intradural extramedullary mass is present at T2, accompanied with severe cord compression and displacement (D, E). The lesion is also hypointense in T1 (F) with peripheral hyperintensity that after Gadolinium injection, is enhanced (G).

resected (Figure 2E). After 6 months, the patient is without any neurological deficit. Informed consent was obtained from the patient for this report.

Discussion

Throughout the literature, SA is safer and has less complications, in comparison with other anaesthetic options e.g., general anaesthesia (GA) (Yüksek et al., 2020). In spite this promising recommendations, occurrence of related neurological complications could be dangerous and knowing the ways to prevent them is crucial.



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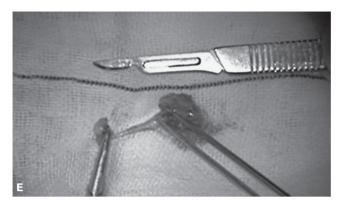


Figure 2 – Dermal sinus tract with its associated dimple is marked with arrow (A). After dural opening, we faced with a long, huge intradural mass with thin capsule containing fluid yellow matrix with hairs (B, C). The capsule was resected as much as possible (D) and finally, dermal sinus tract with its dimple were removed (E).

Some authors reported many neurological consequences after SA, with maximum prevalence of 1/1000; from post-dural puncture headache and drug related toxicity to cardiac arrest due to autonomic imbalance (Agarwal and Kishore, 2009). In general, the neurologic complications related to SA could be summarized to local intervention-site related (backache, hematoma, abscess, arachnoiditis, meningitis, etc.), direct neural elements injury (drug toxicity, autonomic cardiac arrest and direct cord tapping in cases of undiagnosed tethered cord) and cerebrospinal fluid (CSF) related (intracranial hypotension related headache, etc.) (Agarwal and Kishore, 2009; Kim et al., 2015).

History taking and clinical presentation	Positive history for CNS disease (repeated meningitis, etc.); firstly, needs neurologist or neurosurgeon consultation			
	Positive familial history of midline CNS anomalies (dysraphism, etc.)			
	Presence of any neurological symptoms (cranial nerves palsies, paraparesis and other neurological deficits, severe ICP raising headache, disturbed vision, sphincter disturbance, etc.)			
Physical examination	Inspection	Search the midline from nasion to coccyx for dimple or any other surface anomalies; clues for dysraphism		
	Examination	Presence of any neurological deficits; limbs and cranial nerves		
		Presence of any neurological signs; altered DTRs, positive UMN signs (Hofmann's and Babinski's)		
During SA	Reduce CSF volume taking as low as possible			
	In presence of acute paraparesis	Trendelenburg position must be considered		
		Emergent neurologist or neurosurgeon consultation and imaging		

Table 1 – Some clinical hints to avoid CNS herniation during SA	Table 1	1 – Some	clinical hints to	avoid CNS	herniation	during SA
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CNS – central nervous system; SA – spinal anaesthesia; ICP – intracranial pressure; DTRs – deep tendon reflexes; UMN – upper motor neuron; CSF – cerebrospinal fluid

For the understanding of all possible neurologic consequences after SA, the CSF dynamic should be revealed. It is accepted that LP in concomitant with any intracranial mass could cause a lethal cerebral herniation through the foramen magnum (Su et al., 2002). Interestingly, this "sinking effect" after puncturing the dura accompanying CSF taking can develop in any location throughout the spinal canal, from foramen magnum to the sacrum (Doh et al., 2001; Krishnan and Roychowdhury, 2013; Mokri, 2013). Same as our case, dynamic of CSF circulation was changed and CSF circulated in two relatively separated compartments, above and below the canal stenosis due to the tumour. Now, tumour location resembles the foramen magnum and any puncturing of dura and taking CSF below the stenosis could cause intracanal hypotension below the stenosis and as a result, cord herniation through the tumour site and expected neurological deficits are inevitable.

Theoretically, any extramedullary lesions that cause stenosis and CSF block could represent the possible etiology for cord herniation during SA, especially if high amount of CSF is obtained; throughout the literature we can see very few cases in this subject (Doh et al., 2001; Krishnan and Roychowdhury, 2013). For intramedullary lesions, the mass must be as large as to completely block the circulation of CSF and put the spinal cord in close contact with spinal canal wall; that makes any vertical displacement of cord difficult and finally, dural puncture causes neurological deficit, as a result of pressure gradient between separated compartments above and below the stenosis after LP and resulting herniation of the cord through there. In fact, LP can deplete just the compartment below the stenosis. Some other possible causes could explain resulting neurological impairment after dural tap; emerged pressure differences in epidural venous plexus and possible new compression or swelling of the spinal cord adjacent to level of pathology, etc.

In general, such event is more common with concomitant large extramedullar mass (extradural or intradural), e.g., previously reported neurofibroma or large extruded cervical disc, than an expanded intramedullary mass (Doh et al., 2001; Krishnan and Roychowdhury, 2013). In some cases, when the hematoma is not causative and there is acute deficit after LP, we could put the patient in Trendelenburg position to relief herniation and if it is possible inject a sterile solution in the same amount as was taken CSF. In case of SA, this strategy is not appropriate because more prevalent causes of post procedural neurological deficits are other etiologies than cord herniation, this will not work or even be dangerous, e.g., in case of epidural hematoma (Nicholson and Eversole, 1946; Agarwal and Kishore, 2009).

A drawback in our case report is determining status of the neurological exam before SA that we do not know anything about that. Warning neurological status of the patient could possibly be helpful to prevent this complication. It is true that the patient had no complains or problems based on obtained history, but during the neurological exam it is possible to find some signs of involvement in central nervous system (CNS); increases deep tendon reflexes (DTRs) and other upper motor neuron (UMN) signs, e.g., Hoffman's and Babinski signs even without any neurological deficits. Also in this case, if we were more accurate, we could see the dimple of the dermal sinus tract in midline of the skin over the spinal column that could be the alarming sign of a developing anomaly. As a result, anaesthesiologists has to be aware of signs and symptoms that revealed UMN disease such as compressing mass, hyperreflexia, positive Hoffmann's test and Babinski sign, history taking related to it, etc. It is possible these lesions, especially in developmental types or with insidious growth, do not have any symptoms even in presence of a large compressing mass. Table 1 explains some helpful hints to avoid possible complication during SA.

Conclusion

SA is safe and commonly used procedure for anaesthesia. Puncturing the dura associated with CSF taking and in presence of an extramedullary mass with significant compression of the cord could cause cord herniation through the blockade and resulting acute neurological deficits. In these cases, awareness about related manifestations even in absence of symptoms or complaints could help in preventing the post-SA neurological deficit.

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