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Hemianopia: A complication of epidural injection in a patient with arachnoid cyst – case report

Venkata S Rendu[1] and Behzad Eftekhar[2,3]

Abstract

Epidural injections are routinely used for short-term management of radicular pain and chronic low back pain. Prescription of this intervention, in the presence of intracranial abnormalities, is a topic of debate. Intracranial arachnoid cysts are cerebrospinal fluid-filled spaces, which are usually asymptomatic despite being a formidable size. As far as the authors know, there have been no cases depicted in indexed literature regarding asymptomatic supratentorial arachnoid cysts becoming symptomatic post undertaking of spinal epidural injections. We depict this phenomenon in a 53-year-old woman, who ultimately required a craniotomy to address their symptoms. Asymptomatic supratentorial arachnoid cysts can become symptomatic post undertaking of spinal epidural injections. In cases of known cranial arachnoid cysts with mass effect, the small risk that the cranial arachnoid cyst may become symptomatic during or after epidural injections should be a consideration and the patients should be informed of the potential associated risks.

Keywords

Arachnoid cyst, cerebrospinal fluid, epidural injections, spinal injection

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Introduction

Arachnoid cysts (ACs) are cerebrospinal fluid-(CSF) filled spaces that occur in approximately 1.2% of the general population.[1] Intracranially, most are found in relation to an arachnoid cistern or a cerebral fissure, with the middle cranial fossa being the predominant location. Other common locations are the cerebellopontine angle, suprasellar region, and posterior fossa. The ratio of incidence for male:female is approximately 4:1, respectively, with a left-sided predilection. With the exception of suprasellar AC, most are asymptomatic, and are usually discovered incidentally on magnetic resonance imaging (MRI) investigation.[2]

Epidural injections are well known, common procedures for the short-term relief of radicular pain. They are used as adjuncts in the nonoperative management of patients with spondyloarthropathy, including degenerative disc disease, nonspecific pain, lumbar back pain, spinal, and foraminal stenoses. Notable complications include infection, epidural haematoma, intravascular injection, arachnoiditis, CSF fistula, and nerve trauma resulting in paraparesis.[2,3]

As far as the authors know, there have been no reported cases in the indexed literature thus far with respect to *cranial* ACs, including in the supratentorial compartments, becoming symptomatic following lumbar epidural injection in an adult.[4]

Case description

A 53-year-old right-handed woman, with a medical background of chronic lumbar spondyloarthropathy and associated sciatica but no known history of CSF circulation disorders or other previously known structural pathologies of the neuroaxis, presented to the emergency department (ED). She described a 1-week history of sudden onset generalized headache, blurred vision with associated vomiting, and

[1]Department of Neurosurgery, Royal Hobart Hospital, Hobart, TAS, Australia
[2]Department of Neurosurgery, Nepean Clinical School, The University of Sydney, Sydney, NSW, Australia
[3]Department of Neurosurgery, Australian School of Advanced Medicine, Macquarie University, Sydney, NSW, Australia

Corresponding Author:
Behzad Eftekhar, Nepean Clinical School, SMS, FMH, The University of Sydney, Sydney, NSW 2006, Australia.
Email: Behzad.Eftekhar@sydney.edu.au

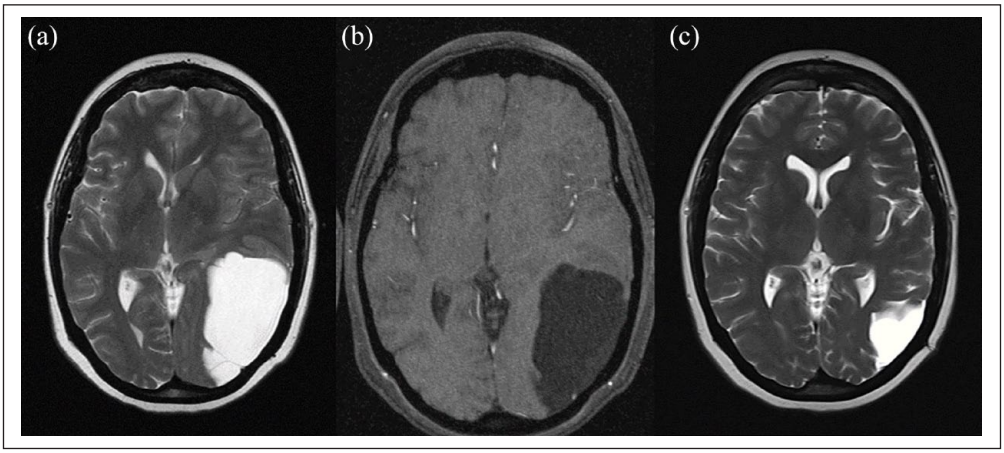

Figure 1. (a) Preoperative axial images of the patient with the left T2-weighted MRI brain image, (b) fat-suppressed T1-post contrast image, identifying the left parieto-occipital lesion, consistent in appearance with an arachnoid cyst. (c) A postoperative axial T2-weighted MRI brain sequence revealing the decompressed left parieto-occipital region, and improvement of the midline shift.

photophobia. One day prior to the onset of her symptoms, she underwent an outpatient epidural injection for her chronic pain symptoms. The procedure had gone uneventfully. On examination in the ED, her initial vitals were within normal limits. The patient had difficulty with speech repetition, there was presence of a right homonymous hemianopia, with a degree of right-sided visuospatial neglect. There were no clinical or biochemical features to suggest infection.

An initial computerized tomography (CT) scan of the brain revealed a hypodense lesion in the left parieto-occipital region consistent with the neurological findings.[5] To further evaluate, an MRI scan of the brain was performed, which confirmed a T1 homogenously hypointense, T2 hyperintense, well-demarcated lesion measuring approximately 8 cm × 6 cm × 5 cm in the left parieto-occipital region, with associated mass effect, midline shift, and effacement of the occipital horn of the left lateral ventricle. There were no radiological or clinical features to suggest obstructive hydrocephalus (Figure 1(a) and (b)).

The patient underwent a left parieto-occipital craniotomy for decompression of the lesion. The mass effect was addressed adequately through fenestration of the cyst and connection to the subarachnoid space. Postoperative MRI imaging revealed significant improvement in relation to midline shift (Figure 1(c)). Her postoperative recovery was uncomplicated. Serial ophthalmological assessments identified improvement of the visual deficits. A 6-monthly progression brain MRI showed stability with respect to the collapsed AC cavity. The histopathological and immunohistochemical analyses confirmed the diagnosis of AC.[2]

Discussion

We reported a case of a patient with an asymptomatic AC becoming symptomatic requiring neurosurgical intervention after epidural spinal procedure. ACs are classified as either congenital or acquired. Congenital (true) ACs are believed to occur during fetal development from splitting of arachnoid membrane, and contain fluid that is similar to CSF. Acquired (secondary) ACs can occur secondary to trauma, hemorrhage, or infection and communicate with the subarachnoid space. ACs may also be classified as either symptomatic or asymptomatic, and also by location.[6,7] Literature evaluation reveals common relative distributions for these lesions as sylvian (49%), cerebellopontine angle (11%), supracollicular (10%), cerebral convexity (4%), vermian (9%), interhemispheric fissure (%5), sellar/suprasellar (9%), and interpeduncular (3%). Radiological differentials for the identified intracranial lesion include AC, epidermoid/dermoid lesions, glioneuronal tumors, ependymal cyst, and porencephalic cysts.[2]

Although the pathogenesis of AC is a poorly understood topic, various theories have been proposed, including developmental alterations in arachnoid membrane resulting in splitting or duplication and subsequent fluid accumulation, defect or abnormalities in relation to CSF flow, or possible agenesis of part of the brain during development.[2,6]

In relation to the expansion of ACs and possible correlation to lesions becoming symptomatic due to raised intracranial pressure, several explanations have been put forward, which include active fluid secretion from the cyst wall of the AC with the involvement of the Na^+—K^+—$2Cl^-$ co-transporter, the osmotic gradient theory, which results in water inflow into the cyst cavity, and furthermore, ball and valve mechanisms for larger cysts, which promote unidirectional flow of CSF—which are further supported by intraoperative findings of valve-like foldings on AC walls or intra-cystic membranes.[2,6]

The clinical presentation of symptomatic ACs can vary based on location and presence of other factors such as ventriculomegaly, mass effect +/− associated midline shift, and types of structures involved. Surgical treatment options for ACs, when symptomatic include cysto-peritoneal shunt placement, craniotomy, or endoscopic fenestration. ACs with considerable mass effect in many aspects can behave

like any other mass lesions in the brain. There are publications that have suggested that epidural anesthesia is dangerous in patients with cerebral space-occupying lesions not only because of the risks associated with accidental dural puncture, but also because epidural drug and fluid injection contributing to increasing intracranial pressure.[8,9] There are case reports of cerebral herniation and major neurological complications following epidural anesthesia.[10,11]

In our patient, the preceding spinal epidural injection, and the subsequent onset of the symptomatology consistent with the anatomical location of the AC, strongly suggests that change in the CSF dynamics or intracranial pressure as a result of spinal epidural procedure, would have been the cause of the neurological symptoms. In our case, an unnoticed dural puncture during the spinal procedure or changed intracranial pressure as the result of the procedure could have contributed to the subsequent symptomatology.[12,13]

There is a recent report of a case of symptomatic intracranial hypertension in an adult patient with spinal muscular atrophy and ACs receiving intrathecal nusinersen. While the authors could not clearly elucidate the cause of intracranial hypertension in the patient, they believed that the ACs might have contributed to a higher susceptibility to developing increased intracranial pressure in their patient.[14]

The current case report raises awareness regarding the risk of an AC with mass effect, becoming symptomatic after a spinal epidural injection procedure. An AC in such regard, should be treated as a cranial mass lesion when we are considering spinal epidural injection procedures. The presence of radiological mass effect is an important factor, as majority of untreated ACs do not have mass effect on MRI or CT of the brain, and we do not otherwise have evidence that their presence poses a higher risk with spinal epidural procedures.

Conclusions

Asymptomatic supratentorial ACs can become symptomatic post undertaking of spinal epidural injections. In cases of known cranial ACs with mass effect, the small risk that the cranial AC may become symptomatic during or after epidural injections should be a consideration and the patients should be informed of the potential associated risks.

Authors' contributions

Conception and design of study: B.E., Drafting the article: V.S.R., literature review: V.S.R., revising the article critically for important intellectual content: B.E., Approval of the version of the article to be published: V.S.R. and B.E.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

ORCID iDs

Venkata S Rendu https://orcid.org/0000-0002-3056-8022

Behzad Eftekhar https://orcid.org/0000-0002-5644-7384

References

1. Weber F. The prevalence of intracranial arachnoid cysts. In: Wester K (ed.) *Arachnoid cysts*. Cambridge, MA: Academic Press, 2018, pp. 95–100.
2. Kirollos R, Helmy A, Thomson S, et al. (eds.) Chapter 96 – Arachnoid cysts. In: *Oxford textbook of neurological surgery*. Oxford, UK: Oxford University Press, 2019, pp. 1101–1106.
3. Goodman BS, Posecion LW, Mallempati S, et al. Complications and pitfalls of lumbar interlaminar and transforaminal epidural injections. *Curr Rev Musculoskelet Med* 2008; 1: 212–222.
4. Umerani MS, Mostafa GA, Nada MAF, et al. Postepidural spinal intradural arachnoid cyst: a rare case report. *J Neurosci Rural Pract* 2017; 8(4): 677–679.
5. Fridriksson J, Kjartansson O, Morgan PS, et al. Impaired speech repetition and left parietal lobe damage. *J Neurosci* 2010; 30(33): 11057–11061.
6. Rengachary SS and Watanabe I. Ultrastructure and pathogenesis of intracranial arachnoid cysts. *J Neuropathol Exp Neurol* 1981; 40(1): 61–83.
7. Boutarbouch M, El Ouahabi A, Rifi L, et al. Management of intracranial arachnoid cysts: institutional experience with initial 32 cases and review of the literature. *Clin Neurol Neurosurg* 2008; 110(1): 1–7.
8. Hilt H, Gramm HJ and Link J. Changes in intracranial pressure associated with extradural anaesthesia. *Br J Anaesth* 1986; 58(6): 676–680.
9. Grocott HP and Mutch WA. Epidural anesthesia and acutely increased intracranial pressure. Lumbar epidural space hydrodynamics in a porcine model. *Anesthesiology* 1996; 85: 1086–1091.
10. Tsai HJ, Tsou MY, Ho CM, et al. Epidural analgesia associated with a fatal outcome in a patient with an unrecognized brain tumour. *Anaesth Intensive Care* 2004; 32(6): 832–834.
11. Su TM, Lan CM, Yang LC, et al. Brain tumor presenting with fatal herniation following delivery under epidural anesthesia. *Anesthesiology* 2002; 96(2): 508–509.
12. Grant R, Condon B, Hart I, et al. Changes in intracranial CSF volume after lumbar puncture and their relationship to post-LP headache. *J Neurol Neurosurg Psychiatry* 1991; 54(5): 440–442.
13. Bromage PR. Physiology and pharmacology of epidural anesthesia. *Anesthesiology* 1967; 28: 592.
14. Machetanz G, Grziwotz M, Semmler L, et al. Symptomatic intracranial hypertension in an adult patient with spinal muscular atrophy and arachnoid cysts receiving nusinersen. *J Neuromusc Dis* 2023; 10: 719–725.