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Case report

Arachnoiditis Following Spinal Anesthesia for Cesarean Section

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ABSTRACT

We report a case of arachnoiditis following subarachnoidal anesthesia for Cesarean section, with paraparesis, lumbar pain and arterial hypertension. The patient was diagnosed with arachnoidits on the basis of the clinical and imaging findings eight days after receiving subarachnoidal anesthesia for a C-section. She presented with emergent hypertensive crisis, and post-partum pre-eclampsia is ruled out. This presentation might be related with the inhibition of spinal regulatory centers. She was treated with systemic dexamethasone. The patient was totally symptom-free within 10 days and went on to recover fully and has been well during more than one year of follow-up.

Aracnoiditis postanestesia raquídea para cesárea

Palabras clave:
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RESUMEN

Se reporta un caso de aracnoiditis tras anestesia subaracnoidea para cesárea que cursa con paraparesia, dolor lumbar e hipertensión arterial. Al paciente se le diagnostica aracnoiditis con criterios clínicos e imaginológicos 8 días después de recibir anestesia subaracnoidea para cesárea. Presenta emergencia hipertensiva, se descarta preeclampsia posparto. Posiblemete esta presentación se relacione con inhibición de centros reguladores medulares. Se trató con dexametasona sistémica. La paciente presenta recuperación completa de sus síntomas en el curso de 10 días, sin recaídas en más de 1 año de seguimiento.

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Introduction

Neurologic lesions in neuroaxial anesthesia are rare, with an estimated incidence of less than 0.04%, including direct neurological injury caused by the needle or by irritation from the agents delivered.[1] Arachnoiditis is the inflammation of the arachnoid membrane, one of the three that envelop the spinal cord and nerve roots. This inflammation produces a fibrinous exudate that promotes adhesion of the nerve roots to the dura mater. Its incidence is unknown, even in the obstetric population in which the use of neuroaxial anesthesia has increased in recent years.[2] The importance of these reports lies in the characterization of the disease in relation to the broad spectrum of clinical presentations, in order to develop prospective studies designed to identify risk factors and vulnerable populations, and prevent potential catastrophic sequelae.[3,4]

Case presentation

Twenty-eight year-old black female patient, gravida 4, para 2, with a history of one stillbirth and one miscarriage, referred by a local health center because of suspected fetal macrosomia. She is in her 40th week of gestation, with uterine height of 37 cm, and is admitted to the obstetrics service on August 22nd, 2008.

On ultrasound, the fetal weight is estimated at 3900 g and, consequently, she is taken to C-section on the same day. The procedure was performed under subarachnoidal anesthesia placed at the level of L3-L4 using two quincke 25 and 26 gauge needles, and a mass of 8 mg of heavy bupivacaine and 20 #mg of fentanyl, both preservative-free and approved for intrathecal administration. There is no more information in the clinical record, but the patient reported having felt pain and more than 3 attempted punctures, and no complications. She was discharged the next day.

The patient was readmitted on August 29th due to back pain, intense headache and hypertensive crisis. The initial diagnostic impression was post-puncture headache versus post-partum pre-eclampsia. Despite oral management with amlodipine, clonidine, hycrochlorothiazide, and enalapril, the patient required a sodium nitroprusside for control of her blood pressure. She was assessed by internal medicine and, given the suspicion of subarachnoid hemorrhage, a brain CT scan was requested, but as found to be normal. Three lumbar taps were performed, recovering hemolyzed blood, although they were made at three different points in time, in three different sites and by three different operators.

Three days later, the patient was assessed by the anesthesia service because of the possibility of post-puncture headache, but this diagnosis was ruled out. Upon examination, there was high blood pressure at 160/110 and neurological deficit in the form of lower limb paresis and a strength score of 3/5. The case was discussed with neurosurgery and they considered ruling out spinal arteriovenous malformation or arachnoiditis. In view of a suspected arachnoiditis, steroid treatment was started using 16 mg of dexamethasone.

A cerebral 6-vessel angiography was performed on September 3rd, with normal findings. The patient evolved satisfactorily until the clinical picture resolved, except for the persistence of hypertension.

A magnetic imaging test was performed on September 11th, showing an inflammatory process at the level of the cauda equina, consistent with arachnoiditis. She was discharged from the hospital for outpatient follow-up by the neurosurgery service. Three months later, the patient was normotensive and was not receiving any form of pharmacological treatment. To this date, she is symptom-free and has no neurological deficit.

Discussion

This case occurred in a Colombian hospital. A higher incidence of complications of neuroaxial anesthesia has been reported in developing countries.[5] However, this case was not associated with an inadequate technique or with the administration of a substance different from the protocols used in the rest of the world. There was difficulty in achieving blockade because multiple punctures and the use of two types of needles were required, constituting a risk factor, as was already mentioned.

Arachnoiditis may result from multiple causes ranging from infections such as syphilis, gonorrhea or tuberculosis; conditions secondary to chemical irritation due to steroids, anesthetics and contrast media used in radiology. It may be secondary to traumatic or surgical processes, in which case it usually manifest late with back pain of very difficult management and imaging studies showing cystic degeneration of the nerve roots.[6] Risk factors for the development of arachnoiditis after neuroaxial anesthesia include: multiple puncture attempts, traumatic punctures and patient-reported paresthesia at the time of using the technique, and others such as the components and the concentration of the substance administered, contamination with disinfectant solutions, the use of drugs with preservatives, the addition of vasopressors and adjuvants such as opioids, and patient-related factors such as the type of immune response and predisposition to developing neural tissue fibrosis.[4,7-10]

Clinical manifestations are complex, with unique duration in each individual case. In our patient, symptoms occurred eight days after the administration of anesthesia, but there are case reports where the problem is found even after 17 years.[11]

The clinical diagnosis is based on a triad consisting of back pain, neurologic deficit and magnetic resonance imaging consistent with the condition,[12] all of which were present in our patient. Additionally, an increased transmission through the dorsal horns and a hyperactive sympathetic system might explain the difficult hypertensive state which was eventually resolved at the same time as the neurological problems.[6]

A characteristic finding is low back pain, which may be accompanied by weakness of lower limbs, sensory changes in strength with differing degrees of reflex compromise. This clinical picture may be confused with spinal cord tethering, spinal tumors or compressive lesions,[12,13] and there are reports

of alterations of skin temperature corresponding with the affected dermatomes.[14] Neuropathy and hypertension may both be associated with obstetric pathology, with pregnancy or labor associated neuropathies having a higher incidence of around 1%, and pre-eclampsia-associated hypertension appearing up to eight weeks post-partum.[15]

For the differential diagnosis with obstetric pathology, MRI has a 92% sensitivity and a 100% specificity, characterizing an image of pseudocysts with narrowing, clustering of nerve roots towards the center of the dural sac, no visualization of nerve root shadows and an empty image in the dural sac, and replacement of the subarachnoid space by soft tissue.[7,16] Pre-eclampsia was ruled out on the basis of negative proteinuria, normal renal and hepatic function markers, and no hematologic abnormalities.

The goal of management is to intervene during the initial phase of the arachnoiditis in order to avoid permanent damage. Consequently, patients should receive this protocol as soon as possible, and it is important to do clinical follow-up of the patients who present paresthesias or some form of associated neurological deficit at the time of undergoing neuroaxial anesthesia. This could be done when the patient is checked after the intervention.

There are multiple management options, including non-steroidal anti-inflammatory agents, pulses of steroids, antidepressants, local anesthetics, neural stimulation and gabapentin. There are reports of management with intrathecal hyaluronidase in patients with etiologies other than anesthetic-related implications.[17] The management protocol is based on steroid pulses, more specifically methylprednisolone, anti-inflammatory agents, dipirone, anticonvulsants such as gabapentin or pergabalin, tri-cyclic antidepressants, alpha-2receptor agonists.[4,8,18] In our patient, we used high-dose systemic steriods, as have been used in other cases of arachnoiditis, achieving good response.[19]

The spectrum of presentation is wide, and so is the possibility of permanent sequelae, including paraplegia, cauda equina syndrome, sensory deficits and paresthesias, among others.[4] In this case, the patient recovered completely in a relatively short period of time and suffered no sequelae.

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Conflict of interests

None declared.

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