

## PREEXISTING NEUROLOGIC DISORDER TRIGGERED BY ANESTHESIA?

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### ABSTRACT

Neurologic deficits appearing after various anesthetic techniques are not always caused by these procedures, also some are attributed to them. Neuraxial blocks – spinal, more often than epidural – have been associated with the postoperative relapse of multiple sclerosis, although the evidence is not strong. The mechanism is unknown but it is speculated that the disease makes the spinal cord more susceptible to the neurotoxic effects of local anesthetics.

We present the case of 42-years-old female, scheduled for ureteroscopy for ureteric calculi under spinal anesthesia. Postoperatively she developed various neurological symptoms that together with the paraclinical findings led to the diagnosis of multiple sclerosis.

**Key words:** anesthesia, neurologic deficit, multiple sclerosis

### INTRODUCTION

Neurologic deficits appearing after various anesthetic techniques are not always caused by these procedures, also some are attributed to. Anesthesia, its complications or the medication used can be the trigger of a dormant, preexisting neurological disease because of various functional alterations produced. (1) The cause of postoperative deficits is sometimes difficult to evaluate, because neural injury may also occur as a result of surgical trauma, tourniquet pressure, improper patient positioning, prolonged labour.

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the CNS, characterized by multiple sites of demyelination in the brain and spinal cord. Peripheral nerves are not affected. The course of the disease consists of remitting and relapsing of symptoms at unpredictable intervals. Stress, fatigue and surgery are implicated in the relapsing of MS. Neuraxial blocks – spinal, more of-

ten, than epidural, have been associated with the postoperative relapse of MS, although the evidence is not strong. The mechanism is unknown but it is speculated that the disease (the demyelination, more precise) makes the spinal cord more susceptible to the neurotoxic effects of local anesthetics, which are used in greater concentration in spinal than in epidural anesthesia. (2,3)

### CASE REPORT

A 42-year-old, ASA physical status II women, with a history of a treated virus C hepatitis, was scheduled for ureteroscopy for ureteric calculi with spinal anesthesia. With the patient in the sitting position, a 25-gauge Quincke needle was inserted at the L2-3 level, clear CSF was obtained and 3 ml of hyperbaric 0,5% bupivacaine were administered, obtaining a T10 sensory level. The operation and immediate recovery were uneventful. The next day, the patient developed photophobia, severe postural

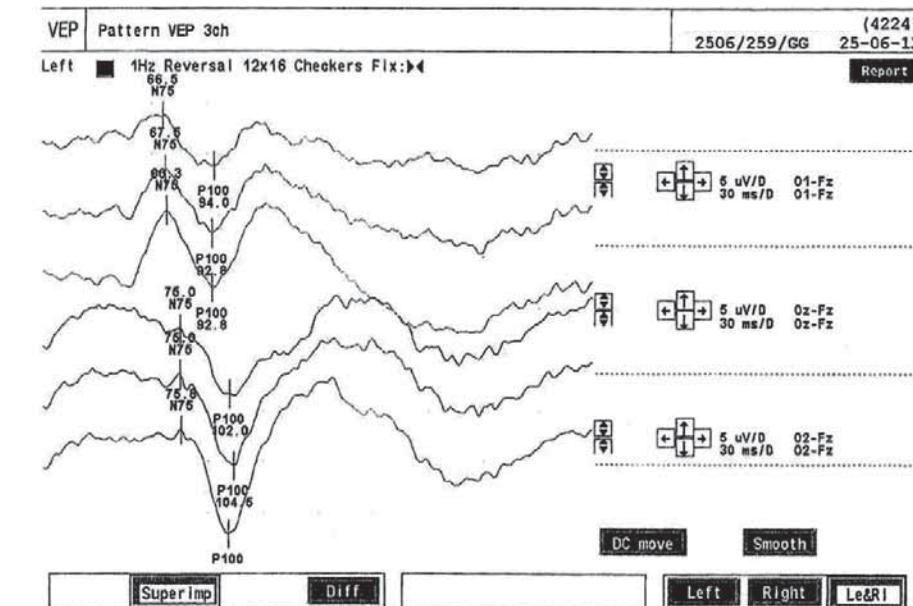
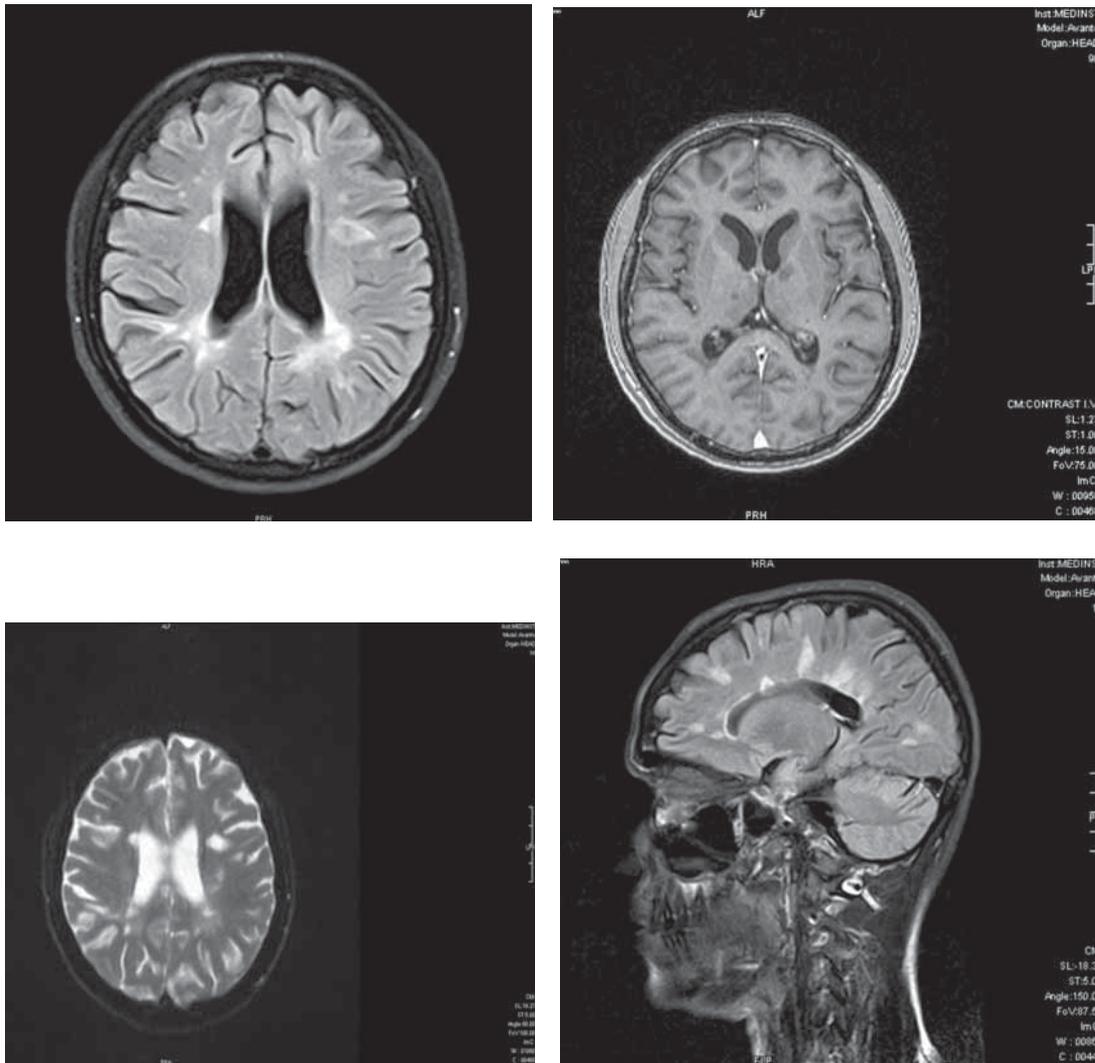
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headache relived by supine position, associated with back pain and weakness of the right lower extremity. Conservative therapy consisting of intrave-

nous fluids, caffeine benzoate, anti-inflammatory drugs and bed rest was initiated and slightly improved the symptoms. A neurologist was called for



PEV - P100 cu latenta  $\leq$  100 ms pe 4 g - 10 Hz (O1-Fz)

FIGURE 1. A diagnosis of multiple sclerosis was made.

an examination, that revealed a conscious patient, with no signs of neck stiffness, brisk deep tendon reflexes on the right lower limb, depression of the abdominal reflexes and moderate headache. The rest of the neurological examination was unremarkable. A CT scan of the lumbo-thoracic spine was performed to rule out a possible spinal hematoma. On the sixth postoperative day, the headache was “clinically improved”, no symptoms on the lower limbs, but the patient reported weakness of both superior limbs. She also informed the medical team that approximately 2 years earlier, while suffering a dental extraction under local anesthesia, she had a temporary episode of weakness of the right upper extremity that lasted for 48 hours accompanied by vertigo. She claimed to have been asymptomatic since then.

The second neurological examination revealed in addition to the first one: eyes with convergent axis, with movement limitation of the right eye to the right, brisk reflexes, paresis tests positive on the upper limbs and extensor plantar responses.

The patient was subject to further investigation: a cerebral MRI that showed “hyperintense lesions on T2-weighted images and FLAIR situated on the middle cerebellar peduncle, on the pons, thalamus and the periventricular white matter; there are also hypointense “black holes” with diameter of maximum 1,2 cm; the lesions are not Gadolinium-enhance”; a visual evoked potential (VEP) with P100 wave latency prolonged, right eye 104 ms; a CSF analysis with oligoclonal banding of IgG present and the IgG index elevated.

## DISCUSSION

This case is relevant because it points out the importance of identifying a neurological pathology preexistent, even minor in patients undergoing anesthesia and surgery. A classic study, conducted by Dripss and Vandam more than 50 years ago reviewing neurological complication after spinal anesthesia, concludes that anesthesia may predispose to the exacerbation of neurological symptoms and to the revelation of dormant neurological disease. (4) Although local anesthetics are used safely worldwide, studies have shown multiple mechanisms by which the drugs may cause injury to nerve and nerve roots; it is obvious that under certain circumstances, excessive dosage and concentration may produce neurotoxicity. (3, 5)

The patient’s headache was misinterpreted as postdural puncture headache (PDPH) - the most common complication of spinal anesthesia, with a reported incidence as high as 25% in some studies. It is more frequent in young people, when using large diameter needles and it is suggested by some that women are at greater risk. The headache is mild or absent when the patient is supine, but head elevation leads to a severe fronto-occipital headache that is improved when returning in supine position. Sometimes, it is associated with cranial nerve symptoms, nausea and vomiting. (6)

The need for prompt diagnosis and intervention (the first 8 hours) in the event of a spinal hematoma is crucial because any delay reduces the chances of a proper recovery. That’s why if a neurological deficit (numbness or weakness) is suspected after neuraxial block an appropriate imaging study must be obtained. So, in our case the CT spine scan was a correct decision although the weakness was present just in the right lower limb.

If the patient’s symptoms haven’t evolved (upper extremity weakness), she would have been discharged from the hospital with a mild headache thus omitting the diagnosis of multiple sclerosis and being convinced that she had suffered an anesthetic complication- PDPH. We can’t incriminate either the anesthetic technique for worsening a progressive disease like MS, it can be just a coincidence.

The actual risk of a neurological complication in a patient suffering from a preexisting neurological disease is hard to be evaluated, especially when no controlled studies have been conducted. Surveys of the neuraxial complications have been published in literature, but these fail to reflect the actual incidence. (7, 8)

## CONCLUSION

Spinal and epidural anesthesia have long been associated with neurological complications that can be divided in two: those unrelated with the neuraxial block but coincide, temporally, and those direct resulting from the anesthetic technique. (9)

It is difficult to identify every patient scheduled for surgery with occult neurological symptoms; however, some of the cases can be identified by a more detailed history (like headache, fatigue, low back pain, paresthesia). (10)

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