

# Case Report: A Case of Meningitis of Undetermined Origin Following Spinal Anaesthesia

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## Abstract

Meningitis occurring after spinal anesthesia (SA) is a rare and much feared complication of this anesthesiologic technique. We report the case of an ASA I woman who underwent SA for saphenectomy and 24 hours later developed meningitis which was ultimately classified as 'aseptic'. Etiology of post-spinal meningitis is much debated and includes failure of aseptic

technique with direct inoculation of bacteria into cerebrospinal fluid, the presence of asymptomatic bacteremia and contamination during puncture through microscopic bleeding, and ultimately the possibility of chemical meningitis. We discuss each specific cause and the possible ways of prevention.

**Keywords:** post spinal anesthesia meningitis, aseptic meningitis.

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## Case Report

Meningitis is a rare, but life-threatening complication of spinal anesthesia. In most instances its presentation is acute, within 24-48 hours of the procedure, and does not differ from meningitis arising from more common sources. Its etiology, though, is much debated and not always clear.

We report the case of a 40 year old, ASA I woman undergoing right saphenectomy with selective spinal anesthesia in ambulatory surgery. Preoperative evaluation was carried out the day before surgery and revealed no health problems. The patient arrived at eight o'clock on the morning of surgery, in good general condition with no signs of systemic infection, and was prepared for selective spinal anesthesia in the recovery room. Venous access with 18G catheter was positioned in the left arm and infusion of 500 ml saline solution was started. The patient was then premedicated with Midazolam 5 mg i.m. and was placed in the right lateral decubitus fetal position. No signs of skin infection were present. A sterile technique (mask, hat, and sterile gloves) was used. The surgical field was prepared with iodine solution (polivinilpirrolidone iodine complex 7.5%). Local anesthesia with 2 ml of lidocaine 2% was performed and then a 25 G Sprotte spinal needle (Polymedic) was inserted at the L3-L4 spinal interspace, obtaining free flow of clear cerebrospinal fluid (CSF) on the second attempt. 7 mg of 0.5% hyperbaric bupivacaine were injected. A disposable spinal kit was used. No antibiotic surgical prophylaxis was administered, as is routine for this kind of surgery.

After surgery the patient remained monitored in the recovery room with dedicated nursing personnel. She was able to urinate spontaneously after 3 hours, and had no nausea and thus had a light lunch. She readily gained back motor and sensory right leg function and was thus 4 mobilized twice. After 6 hours from the end of surgery the patient was dismissed in general good condition.

On the next morning the patient was admitted to the emergency department with an altered mental status, a Glasgow Coma Scale score of 10, fever and nuchal rigidity. She had closed eyes, was

arousable to localized painful stimulus, but did not answer to commands. Vital parameters were normal. During the night the husband reported repeated episodes of vomiting.

Lumbar puncture showed a turbid CSF with a protein concentration of 626 mg/dl (normal range, 20-50 mg/dl), a glucose concentration of 8 mg/dl (normal range, 40-70 mg/dl), and numerous polymorphonuclear cells. Blood chemistry was significant for a WBC count of 22,000. Chest radiography showed a right lower lobe paracardiac infiltrate with initial pneumonia screening for anti-Legionella and anti-S. Pneumoniae antibodies both negative. The patient was started on IV antibiotic therapy with Ceftriaxone 2g BID and Vancomycin 500 mg QID, was given dexamethasone 10 mg and was admitted to the neurology department with the diagnosis of acute bacterial meningitis. The pulmonary infiltrate was treated as atypical pneumonia.

Clinical progression was favourable and the patient had no permanent neurologic deficit. Repeated CSF cultures from day one through day 7 showed no growth of bacteria, and blood cultures were negative as well.

## Discussion

The pathogenesis of meningitis following spinal anesthesia is debated and only few cases of the latter are described in literature. Three possible explanations are currently given. First and foremost, there is the failure to obtain complete asepsis during the procedure, either from equipment contamination or from not following a strict aseptic technique, and thus with direct introduction of bacteria into the CSF. A second possible pathogenesis is the presence of prior asymptomatic bacteremia and the contamination of CSF fluid through microscopic bleeding caused by needle insertion. Finally, there is the hypothesis of a physico-chemical meningitis from introduction of iodine solution or by needle trauma, that may be supposed in the absence of bacterial growth.

In the described case, all sequential CSF cultures were negative for bacterial growth. Nevertheless, the low glucose CSF concentration and the rapid response to wide-spectrum antibiotic treatment do not exclude a bacterial origin for the meningitis. Appropriate antibiotic prophylaxis could inhibit bacterial growth in cultures, but no antibiotic was administered to this patient as per routine for this surgery. DNA extraction and amplification of samples of CSF could help in determining whether a bacterial cause was present, and should always be done in the absence of evident bacterial growth in culture<sup>1</sup>. The most frequent bacterium involved in this complication appears to be *Streptococcus Salivarius* but without DNA sequencing, in fact, it is not always isolated [1]. A strict sterile technique was followed in our case although we did not use single-use containers of polyvidone iodine solution. Multiple-use containers are less effective in creating asepsis and are more susceptible to colonization by bacteria [2].

The pulmonary infiltrate noticed on chest radiography may let us suppose a misdiagnosed flogistic process present before the procedure and surgery, but the absence of bacteremia with the negative blood cultures actually exclude this hypothesis.

Finally, the hypothesis of chemical meningitis may not be excluded in the presence of more than a single puncture attempt and with the use of 10% polyvidone iodine. Incidence of chemical meningitis has greatly decreased since the introduction of autoclaving and, more recently, of disposable anesthesia trays, but cases are still being reported [3].

Our patient was discharged in good condition with the diagnosis of post-spinal aseptic meningitis, since the precise etiologic determinant could not be ascertained. We believe that the rarity of this complication warrants the necessity to share the experience for such cases so that discussion and learning may help other practitioners.

## References

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