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## *Streptococcus mitis*-induced Bacteremia and Meningitis after Spinal Anesthesia

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BACTERIAL meningitis is a rarely reported complication of spinal anesthesia.<sup>1,2</sup> We report a case of a viridans streptococcal bacteremia and meningitis developing in a man within 24 h of an uncomplicated inguinal herniorrhaphy performed under spinal anesthesia.

### Case Report

A 76-yr-old man was admitted for elective repair of an easily reducible right inguinal hernia, which had been slowly enlarging over an 18-yr period. His medical history included bipolar affective disorder, Parkinson's disease, congestive cardiac failure, and mild obstructive airway disease. The patient was known to have had a murmur of mitral regurgitation for at least 2 yr but no other history of cardiac disease.

Preoperative assessment the day before surgery indicated that these conditions were medically well controlled. The patient's sublingual temperature was 36.5° C. There was no evidence of ear, nose, or throat infection, but the patient had poor oral hygiene. Baseline hematologic and biochemical investigations, which had been performed 2 weeks earlier, had normal results, including a leukocyte count of  $8.7 \times 10^9/L$ .

He was taken to the operating room, and the lumbar area was prepared with a 1-min application of 10% povidone-iodine. The skin and deeper tissues were infiltrated with 1% lidocaine, and a 26-gauge needle was passed through a 19-gauge introducer needle. After clear cerebrospinal fluid (CSF) was seen, 4 ml of 0.5% bupivacaine was injected, producing satisfactory spinal block up to the T10 dermatome. The anesthesiologist wore a mask, a sterile gown and gloves,

and used an autoclave-sterilized dressing pack and sterile disposable needles and syringes. No other anesthetic drugs were given, nor was an oropharyngeal airway used. Inguinal herniorrhaphy was performed without complication. The patient was well immediately postoperatively, but 16 h later, vomiting, a fever of 39.0° C, and progressive obtundation occurred. Physical examination revealed generalized rigidity and upgoing plantar responses but no other focal neurologic signs. There were no stigmata of infective endocarditis. Laboratory investigations revealed a leukocytosis of  $18.6 \times 10^9/L$  (90% neutrophils) and normal biochemical results. A specimen was taken for blood culture, and intravenous ampicillin, gentamicin, and metronidazole were administered. The results of a computed tomographic scan of the head without an intravenous contrast agent were initially reported to be normal for a 76-yr-old patient. The next day a viridans type of streptococci, subsequently identified as *Streptococcus mitis*, grew in both anaerobic and aerobic blood culture bottles. Lumbar puncture was performed 24 h after the commencement of antibiotic drugs and revealed turbid CSF with a cell count of 15,000 polymorphonuclear leukocyte cells/ $\mu l$ , a protein concentration of 1.1 g/L (normal range, 0.1–0.4 g/L), and a glucose concentration of 1.9 mM (normal range, 2.5–4.5 mM). Gram staining revealed occasional intracellular gram-positive cocci, but the CSF culture was negative.

Acute streptococcal meningitis was diagnosed and treated with intravenous penicillin 2.4 g every 6 h and gentamicin 80 mg twice daily continued for 14 days. A review of the original computed tomographic scan revealed material in the posterior horns of the lateral ventricles, highly suggestive of purulent material consistent with ventriculitis (fig. 1). Transthoracic two-dimensional echocardiography performed on the second postoperative day suggested vegetations on a redundant mildly incompetent mitral valve, but transesophageal echocardiography 5 days later did not confirm the presence of vegetations.

The response to therapy was dramatic. There was complete clearing of the sensorium with resolution of the fever over 4–5 days. These signs were accompanied by a decrease in the peripheral leukocyte count and C-reactive protein. The patient was discharged home on the 16th postoperative day and was well at follow-up.

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### Discussion

The role of spinal anesthesia in the pathogenesis of meningitis remains controversial, with the few reported cases open to criticism. Berman *et al.*<sup>1</sup> reported a case

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cation of spinal anesthesia. Those providing postoperative care should be aware of this complication because early diagnosis and treatment may be life saving.

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## Acute Arterial Insufficiency of the Upper Extremity after Central Venous Cannulation

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SINCE the introduction of Seldinger's<sup>1</sup> ingenious technique of vascular cannulation in 1952, the medical literature has been replete with descriptions of multiple iatrogenic complications associated with intravascular catheters and their insertion. We present a case in which there was a complication secondary to the use of a central venous catheter.

### Case Report

A 6.4-kg 10-month-old girl with a medical history significant only for craniofacial asymmetry was admitted to the New York University

Medical Center for elective surgical correction of her anomaly. Because this procedure is associated with massive blood loss, it is our policy to monitor arterial and central venous pressures *via* invasive catheters.

After uneventful inhalation induction of general anesthesia, peripheral intravenous access was established, and her trachea was intubated. The left radial artery was cannulated easily. The surgical fellow then attempted cannulation of the left subclavian vein using the Seldinger technique and an Arrow Pediatric Jugular Puncture Kit (product no. AK-04150-E, Arrow International, Reading, Pennsylvania), which contains a 35-cm long, 0.64-mm diameter spring guide wire with J tip and a 3.8-cm 22-gauge introducer needle. Difficulty maneuvering the guide wire was encountered, and it was withdrawn through the introducer needle while both were still in the patient's chest. The guide wire was kinked and reduced in length. The arterial pressure wave form decreased in amplitude then became flat. Physical examination of the left upper extremity revealed pallor and pulselessness, which was confirmed by oscillometric blood pressure cuff, pulse oximetry, and poor capillary refill. Normal perfusion of the right upper extremity was verified by similar methods. The patient's breath sounds were equal, her vital signs were stable, and there was no evidence of a hematoma.

At this point, we realized that the guide wire had fractured, and the distal segment remained *in vivo*, resulting in arterial insufficiency of the left upper extremity. The underlying causes for this injury were hypothesized to be one of the following: external compression of the subclavian artery by either the retained guide wire or a hematoma; perforation of the subclavian artery, and raising of an ob-

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