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Two cases of chemical meningitis following spinal anesthesia

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Introduction

Meningitis that develops following spinal anesthesia includes both bacterial meningitis, which produces life-threatening symptoms, and aseptic meningitis, which is normally associated with less severe symptoms and a satisfactory prognosis. Chemically induced meningitis, which belongs to the latter category, is even less severe than most other types of meningitis, and patients recover in a short time. It is often overlooked as a simple case of headache and fever following spinal anesthesia. Very few physicians are aware that the condition can be provoked by sterilizing agents.

Two cases of meningitis were encountered in which the cause may have been a residual sterilizing agent in syringes that were used to inject a local anesthetic agent for spinal anesthesia.

Case reports

Case 1

A 30-year-old woman was scheduled to undergo evacuation of the uterine contents under spinal anesthesia for a hydatidiform mole. Following handwashing and disinfection, the anesthesiologist put on sterilized, disposable gloves. He then disinfected the patient's skin twice with 0.5% chlorhexidine ethanol, using a tray for spinal anesthesia that had been sterilized and disinfected within the hospital. A puncture was made without any difficulty between L3 and L4 by using a disposable 25G needle for spinal anesthesia, and 2 ml of 0.3% dibucaine HCl was injected by using a 5-ml glass syringe that had been sterilized in the hospital. At the completion of the surgical procedure, the patient experienced mild nausea. In the ward 3 h after spinal anesthesia, she developed nausea, headache, a slight fever, and chills. Five hours later, her temperature returned to 37.6°C.

A neurological examination was conducted the next day, and no abnormalities of the cranial nerves were recognized; however, headache, nuchal rigidity, and positive Kernig sign were noted. Hematological examination revealed an inflammatory state (white blood cell count, $9990 \cdot mm^{-3}$; C-reactive protein, $2.6\,mg \cdot dl^{-1}$). The results of a lumbar puncture indicated the following: cerebrospinal fluid, slightly turbid; initial pressure, $175\,mmH_20$; cell counts $664/3\,mm^3$ (poly, 400 and mono, 264); protein, $152\,mg \cdot dl^{-1}$; sugar, $57\,mg \cdot dl^{-1}$. The patient was treated with piperacillin sodium for 8 days under the diagnosis of meningitis. The cerebrospinal fluid culture yielded no bacterial growth. On the fifth day, she no longer suffered from neurological sequelae and was considered to have recovered completely.

Case 2

At 12 weeks and 6 days of pregnancy, a 24-year-old woman with cervical incompetence that had been caused by cervical conization was scheduled to undergo cervical cerclage under spinal anesthesia to prevent premature labor. Following handwashing and disinfection, the anesthetic procedure was performed in the same way as in Case 1 using the same drug and equipment set.

Throughout the surgical procedure and after the patient was returned to the ward, no changes in vital signs were noted and she reported no subjective symptoms. However, nausea and vomiting developed 90 min and headache 2 h after spinal anesthesia. Twelve hours after

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anesthesia, she experienced urinary incontinence, somnolence, and nuchal rigidity. The physical findings at this time were blood pressure, 109/63 mmHg; heart rate, 98 beats·min^{-1}; and body temperature, 37.1°C. Neurological examination found no abnormalities in the cranial nerves but did recognize nuchal rigidity and positive Kernig signs. A hematological examination showed signs of inflammation (white blood cell count, 12890·mm^{-3}; C-reactive protein, 0.94 mg·dl^{-1}). Cranial computed tomography indicated diffuse cerebral edema. On the following day, a lumbar puncture revealed the following: cerebrospinal fluid, slightly turbid; initial pressure, 155 mmH_2O; cell count, 7500/3 mm^3 (poly, 6000, mono, 1500); protein, 127 mg·dl^{-1}; sugar, 95 mg·dl^{-1}. Starting on the first day of the current illness, the patient was treated with ampicillin for 3 days and cefpirome sulfate for 2 days. The cerebrospinal fluid culture yielded no bacterial growth. On the fifth day after onset, the patient recovered without any neurological sequelae.

Discussion

For the present cases, bacterial or viral meningitis was excluded became of the onset of illness and the appearance and findings of the cerebrospinal fluid. There had been a relatively large number of reports on aseptic meningitis following spinal anesthesia until the 1940s. The incidence was 0.26% [1,2]. Since then, improvements in sterilization and antiseptic procedures and the distribution of disposable equipment have reduced the incidence of aseptic meningitis dramatically. In the last 3 years, only a single case of aseptic meningitis in a child has been reported [3]. For the cases of aseptic meningitis, previous reports [1,4–8] incriminated cleansing agents and antiseptics that adhere to the syringes and puncture needles for spinal anesthesia and called the resultant condition chemical meningitis. We examined the glass syringe that was cleansed and sterilized at our hospital and used for spinal anesthesia by ninhydrin color reaction using thin-layer chromatography. A minute quantity of Tego-51 solution used as an antiseptic was detected. In the present two cases, meningitis developed shortly after a spinal puncture. Because no bacterial growth was detected in the cerebrospinal fluid culture, the sugar content of the cerebrospinal fluid was normal, and the prognosis was satisfactory, the most likely diagnosis was chemical meningitis. As a disease resembling chemical meningitis in their clinical course, aseptic meningitis induced by drugs administrated systematically has been recognized. These meningitides are caused by NSAIDs, H_2 blockers, carbamazepine, azathioprine, and antibacterial agents [9]. From the symptoms and immunological data, the cause of these meningitides has been presumed to be an acute hypersensitivity reaction to drugs. Neither of the patients reported here had been exposed to these drugs prior to onset, which substantiated the diagnosis of chemical meningitis.

During the cleaning process for glass syringes at our hospital, it is suspected that insufficient rinsing under running water to remove the antiseptic agents accounts for the cause of meningitis. It is very difficult to remove antiseptics in syringes completely. Therefore, the use of disposable syringes is more efficient to prevent meningitis after spinal anesthesia. Yokoyama et al. [10] stated that disposable spinal needles should be used, and that during the cleaning process, the anesthetic equipment set should be thoroughly washed in water or alcohol without antiseptics, followed by sterilizing by autoclaving.

Until these cases occurred, we had been using glass syringes that had been sterilized by the method mentioned above for about 300 patients annually. The incidence was limited to the above-cited two cases, and both patients had been treated in the Department of Gynecology and Obstetrics. During pregnancy, the immune and endocrine systems change [11,12]. The level of human chorionic gonadotropin (hCG), which is reputedly associated with an immunosuppressive action, dramatically increases [13], especially in the early stage of pregnancy. The pregnant state may alter the response to a minute quantity of any chemical substance that contaminates the cerebrospinal fluid.

In conclusion, we report here two obstetric cases of chemical meningitis following spinal anesthesia. The cause may have been a residual sterilizing agent in the syringes.

References

1. Goldmann WW, Sandford JP (1960) An "Epidemic" of chemical meningitis. Am J Med 29:94–101
2. Thorsen G (1947) Neurological complications after spinal anesthesia and results from 2493 follow-up cases. Act Chir Scand (suppl 121) 95:1–272
3. Easley RB, George R, Connor D, Tobias JD (1999) Aseptic meningitis after spinal anesthesia in an infant. Anesthesiology 91:305–307
4. Harding SA, Collis RE, Morgan BM (1994) Meningitis after combined spinal—epidural anesthesia in obstetrics. Br J Anaesth 73:545–547
5. Gibbons RB (1969) Chemical meningitis following spinal anesthesia. JAMA 210:900–902
6. Bert AA, Laasberg LH (1985) Aseptic meningitis following spinal anesthesia: a complication of the past? Anesthesiology 62:674–677
7. Austin DA, Sokolowski JW (1968) Postlumbar puncture chemical meningitis. NY J Med 68:2444–2446
8. Kashiwagi S, Inoya Y, Asahi N, Oota K, Katou K, Tomita Y (1985) A case of aseptic meningitis following spinal anesthesia (in Japanese). Rinsho Masui (J Clin Anesth) 9:429–432

9. Burke D, Wildsmith JAW (1997) Meningitis after spinal anesthesia. Br J Anaesth 78:635–636
10. Yokoyama K, Masuda R, Tamura T, Simai N, Oumi S, Kobayashi T (1991) Spinal anesthesia for medical clinics (in Japanese). HBJ Syuppannkyoku, Tokyo, pp 176–177
11. Clemens LE, Siiteri PK, Stites DP (1979) Mechanisms of immunosuppression of progesterone on maternal lymphocyte activation during pregnancy. J Immunol 122:1978–1985
12. Gaugas JM, Curzen P (1978) Polyamine interaction with pregnancy serum in suppression of lymphocyte transformation. Lancet 1:18–20
13. Takeuchi S, Takakuwa K (1987) Maternal immunological adaptation for pregnancy. In: Sakamoto S, Taki I, Murooka H (eds) Immunological guide for obstetrics (in Japanese). Kaneharasyuppann, Tokyo, pp 50–55