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Case Report
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Yellow cerebrospinal fluid flow during spinal anesthesia: a case report

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When a spinal needle enters the subarachnoid space after dural puncture, a clear cerebrospinal fluid (CSF) emerges. If the color of the CSF is not clear, it is necessary to rule out the cause of such a change including subarachnoid hemorrhage (SAH). Here, we report a case of a seventy-nine-year-old female, to whom we initially attempted spinal anesthesia but converted to general anesthesia due to yellow CSF. Unlike normal colorless CSF, yellowish CSF is called xanthochromia. Xanthochromia may result from various causes such as blood, bilirubin, and protein in CSF. We suggest against to proceeding with spinal anesthesia in patients with xanthochromia because it is difficult to differentiate the exact cause without appropriate CSF analysis at the moment.

Keywords: Anesthesia; Spinal; Cerebrospinal fluid; Spinal puncture

INTRODUCTION

Spinal anesthesia is a common form of anesthesia used in surgeries of lower part of the body including the lower extremities. When a spinal needle enters the subarachnoid space after dural puncture, a clear cerebrospinal fluid (CSF) emerges. If the color of the CSF is not clear, it is necessary to rule out the cause of such a change including subarachnoid hemorrhage (SAH). However, it might be difficult to identify the cause at the time of anesthesia and the anesthesiologist may be hesitant whether spinal anesthesia can be performed or not. In this case report, we discuss a case of spinal anesthesia which was converted to general anesthesia due to yellow CSF, with literature review on several possible etiologies.

CASE REPORT

A seventy-nine-year-old female with no specific past medical history other than a previous vertebroplasty on T10, T12 and L4 due to compression fracture visited our institution with left femur fracture that occurred while walking at home. The patient was admitted to the orthopedics department and was scheduled for a left hemiarthroplasty. At the time of trauma, there was no accompanying injury other than the left femur. During the preoperative evaluation process, acute pyelonephritis in both kidneys was found and treated with antibiotics. Preoperative echocardiogram

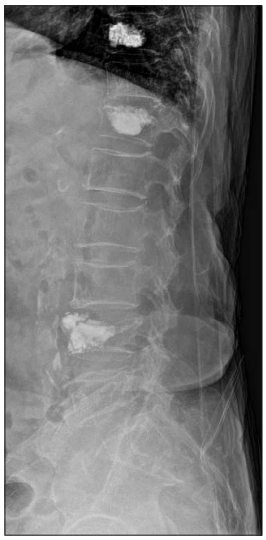

Fig. 1. Pre-operative L-spine X-ray. It shows status post vertebroplasty in T10, T12, and L4, and compression fracture at L5.

showed an ejection fraction of 65%, moderate tricuspid regurgitation, mild pulmonary hypertension with pulmonary artery systolic pressure of 57 mmHg. Pulmonary function test showed a moderate obstructive, mild restrictive pattern. On arrival, the patient's initial hemoglobin was 8.9 g/dL, but it decreased to 7.3 g/dL the day after the injury and 1 pint of packed RBC was transfused. Even after the transfusion, hemoglobin level was consistently low, so an additional 2 pints of packed RBC were transfused, resulting in a hemoglobin level of 11.1 g/dL on the day of surgery, six days after the trauma.

Lumbar spine X-ray image taken on hospital arrival was significant for status post vertebroplasty in T10, T12, and L4, as well as compression fracture at L5 (Fig. 1). The patient was not taking any anticoagulant drug. Laboratory findings including coagulation and platelet count were within normal. There was no absolute or relative contraindication for spinal anesthesia. Also, the patient preferred spinal anesthesia over general anesthesia. As a result, we decided to proceed with spinal anesthesia.

In the operating room, the patient's vital signs were stable with blood pressure of 155/56 mmHg, heart rate of 87 beats/min, respiratory rate 22 breaths/min, oxygen saturation 99% on room air and temperature was 37.1 degree of Celsius with no fever. Since the injured site was the left femur, the patient took a right lateral down position. Skin preparation was done with chlorhexidine-ethanol solution and a 25 gauge Quincke spinal needle was inserted at the L4-5 level by paramedian approach. Free flowing CSF with a viscosity similar to that of the normal CSF appeared, but the color was pale yellow. Although there was no history nor symptoms of SAH, other factors such as infection could not be ruled out. Consequently, the method of anesthesia was converted to general anesthesia. Yellow CSF was collected for additional tests.

General anesthesia was done with 40 mg propofol, 30 mg rocuronium for induction, and continuous infusion of remifentanil and desflurane for maintenance. The surgical procedure was uneventful; it took an hour and 40 minutes with an estimated blood loss of 200 mL. The patient recovered successfully in the Post-anesthesia care unit (PACU) without any complication. Her vitals were stable, and was sent to the ward fully awake.

CSF analysis showed specific gravity of 1.009, 260/μL RBC count, 0/μL WBC count. Gram stain and acid-fast bacilli stain were negative. CSF culture was also negative for any bacteria or fungus. Other CSF studies including protein count were unavailable due to lack of CSF sample volume.

To report this case, a written informed consent form was obtained from the patient.

DISCUSSION

Unlike normal colorless CSF, yellowish CSF is called xanthochromia, which derived from the Greek "xanthos" meaning yellow [1]. Xanthochromia can be caused by primarily RBCs in CSF including SAH. Blood in subarachnoid space is degraded after 6 to 12 hours, resulting in byproducts such as oxyhemoglobin, bilirubin, and methemoglobin in the CSF, which cause color change of the CSF.

As the morbidity and mortality of SAH are high, differentiating it from traumatic lumbar puncture, or "traumatic tap" is important. Traumatic tap occurs when the venous plexus located around the spinal sac is punctured. Although the incidence is not precisely known, some authors say it is 20% and Eskey et al. state that incidence of traumatic tap was 10.1% during 1489 procedures. One way to differentiate traumatic tap from SAH is a 3-tube test. In a traumatic tap, CSF is bloody in the first tube, but becomes more transparent in the second tube, with the third tube being the most transparent of all three tubes. On the other hand, the color

change of the CSF is consistent in all three tubes in the case of SAH [2]. However, visual inspection may not be accurate when the amount of pigment is small. In this case, spectrophotometry can be helpful, and the absence of bilirubin in the CSF can be seen in traumatic tap [3].

Our patient's CSF contained 260/μL RBC count, which could have attributed to the patient's xanthochromia. Thus, further investigation on its origin is necessary, especially to rule out SAH. The patient had no previous history of brain hemorrhage such as SAH or intracranial hemorrhage. The patient claimed no injury other than the left femur fracture at the time of the trauma, and there were no accompanying neurologic symptom. However, silent SAH could not be completely ruled out because no brain imaging has been performed. The possibility of traumatic tap is low, as CSF came out at once without blood on visual inspection. Moreover, the color of the CSF did not fade during the observation. Nevertheless, there still remains the possibility of continuous microbleed with small amount of RBC just enough to be clear yellowish, but not bloody in the naked eye due to puncture of microvasculature.

Causes of xanthochromia other than blood in CSF include jaundice due to hemolytic disease or liver disease, increased CSF protein level, and drugs. In case of jaundice, the serum-to-CSF bilirubin ratio in adult ranges from 1:10 to 1:100 [4], but the CSF discoloration is visible only when total serum bilirubin is 10–15 mg/dL or more. The color change is more apparent when the protein concentration in CSF exceeds 150 mg/dL, which is due to an increase in albumin-bound bilirubin.

High protein levels in CSF appear in Froin's syndrome, Pseudo-Froin's syndrome, radicular demyelination, carcinomatous meningitis, intracranial neoplasm, and cryptococcal or tuberculous meningitis. CSF pigmentation has also been reported in patients taking rifampin, a tuberculosis drug.

Froin's syndrome was first described by a French physician named Georges Froin, and is characterized by xanthochromia with high protein levels and hypercoagulated CSF [5]. This phenomenon occurs when CSF flow is blocked by tumor mass or abscess, and the CSF protein level rises upto at least 5 gm/L, which is much higher than the normal value of 0.15 to 0.45 gm/L. In pseudo-Froin's syndrome, patients complain of back pain and sciatica due to disc bulging. CSF stagnation also occurs and high protein levels are observed [6].

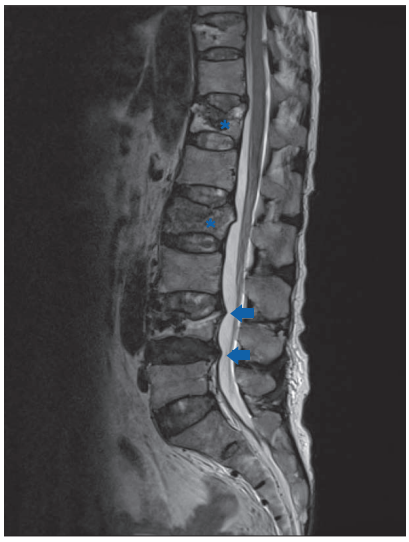

Fig. 2. Post-operative L-spine MRI. Asterixs indicate T12 and L2 compression fracture. Arrow indicates L3–4, 4–5 mild disc space bulging.

Our patient's total serum bilirubin level was 0.7 mg/dL, which is normal, thus primarily ruling out discoloration of the CSF due to hyperbilirubinemia. The patient was not taking any tuberculosis medication, and also had no history of meningitis nor neurologic symptoms. However, the possibility of Pseudo-Froin's syndrome could be considered, as the patient continued to complain of back pain even after her acute pyelonephritis proved.

In our case, according to the patient's L-spine MRI taken after the surgery, L2 vertebral body compression fracture was the cause of the patient's back pain (Fig. 2). There was also disc bulging in L4-5 level, which was mild, but still could have been the cause of some degree of CSF stagnation, leading to pseudo-Froin's syndrome. Also, a small increase of protein in CSF with concurrent microbleed, if this patient had, could have resulted in exaggerated color change of CSF due to albumin-bound-bilirubin. Because there may be ambiguous cases such as this one, CSF analysis can be an essential clue in determining the cause of CSF discoloration. Although we conducted CSF study, only the cell count could be confirmed due to lack of sample volume. Failure to confirm protein and coagulation level remains as a limitation.

In addition to patient-related factors, we also speculated color change owing to foreign substance in the spinal needle. We considered the possibility of malproduction or con-

tamination of spinal needle during the manufacturing process, but in that scenario, yellow CSF should have appeared in other cases that used spinal needles with the nearest serial number from the same manufacturer. Our case, however, was the only case where yellow CSF occurred for several months. Another possibility of foreign substance was antiseptic solution used for skin disinfection which could have entered the spinal needle during skin puncture; this could be excluded in our case because colorless, clear chlorhexidine-ethanol solution was used instead of betadine.

In summary, the etiologies of xanthochromia can vary widely; blood in CSF due to SAH, traumatic tap, patient factors such as hyperbilirubinemia or high CSF protein are the most common etiologies. Although it was reported that spinal anesthesia was performed successfully without any complication in patients with pseudo-Froin's syndrome [7], it is difficult to diagnose it at the very moment without appropriate CSF analysis. With still the possibility of undiagnosed SAH, one should not proceed with spinal anesthesia. Thus, in conclusion, we suggest against proceeding with spinal anesthesia in patients with xanthochromia unless general anesthesia is absolutely contraindicated. To rule out several causes mentioned above, CSF analysis including total protein, albumin, immunoglobulin, glucose, lactate, cell count and cytology is recommended. Also serum bilirubin, neurologic symptoms and history, history of tuberculosis drug administration must also be investigated [8].

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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