Subdural hematoma occurred after spinal anesthesia in a human immunodeficiency virus-infected patient

Introduction

Human immunodeficiency virus (HIV) infection is a generalized disorder that brings about immune system dysfunction, ultimately resulting in dysfunctional respiratory, gastrointestinal, cardiovascular, and hepatobiliary systems as well as inducing hemodynamic instability. HIV damages vascular walls through the release of inflammatory mediators that cause smooth muscle proliferation and endothelial dysfunction, resulting in vasculopathy, which in turn may induce hemorrhagic complications.[1] Moreover, due to these properties of HIV infection, HIV-infected patients are at higher risk for spontaneous intracranial hemorrhage.[2]

During a preoperative evaluation, special attention must be given to the HIV patients' current immune status, treatment agents with their side effects, and any accompanying diseases. Furthermore, the anesthetic method should be determined with regards to the effects of the disease on anesthesia and of surgery. In particular, a headache after spinal anesthesia can be caused by several factors; thus, the accurate cause must be pinpointed with certainty.

The authors report a case of an SDH occurred after spinal anesthesia in an HIV-infected patient.

Case Report

A 25-year-old male patient (63 kg, 180 cm) visited our hospital for an anal condyloma. Excisional biopsy under spinal anesthesia was scheduled. On preoperative examination, the blood test showed positive for HIV antibodies. Thus, a lymphocyte subset test was performed, and the patient was diagnosed with HIV based on the following

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results: CD4(+) T-cells 324/µl, CD4 14.2% (28.4–56.4), and CD4/CD8 0.2 (0.9 – 3.6). There was no record of particular significance in the patient’s past medical history. His blood test showed following: Hemoglobin 15.8 g/dl, hematocrit 45.7%, and platelet 217,000 cell/mm³. The patient’s blood coagulation was also in the normal range with a prothrombin time (PT) of 13.5 s (INR 1.3) and an activated partial thromboplastin time of 35.7 s. His chest X-ray and electrocardiograph findings were also normal.

Without premedication, on arriving at the operating room, the patient’s vital signs read as follows: Blood pressure (BP) 147/66 mmHg, pulse 70 beats/min, and O₂ saturation 100%.

Spinal anesthesia was performed in sitting position with midline approach at L3–4 space, using a 25-gauge, Quincke (Hakko Co. Ltd, Chikuma, Japan) needle. The drug used 8 mg of 0.5% bupivacaine (Marcaine®, AstraZeneca) was administered successfully in the first attempt. His vital signs were stable. The operation was concluded in 35 min without any abnormal findings. After 4 h, the patient started to experience a headache without nausea and vomiting in the general ward.

The symptom was not related to the position. A headache improved partially following bed rest and taking 650 mg of acetaminophen (Tylenol ER®). The patient was discharged 1 day after the surgery with no abnormal symptoms.

One day after the discharge, the patient experienced a headache with accompanying nausea and vomiting. The patient visited the emergency room after a headache had aggravated further. BP was 150/81 mmHg, and pulse was 95 beats/min at the time. There were no neurologic anomalies found during the physical exam. He was hospitalized at the neurology department for further evaluation and conservative management.

On the 1st day, the patient’s headache did not improve despite fluid management, nonsteroidal anti-inflammatory drugs (NSAIDs), and bed rest. We performed an enhanced brain magnetic resonance imaging to identify the cause of a headache and found a small amount of SDH in the left occipital region and interhemispheric fissure [Figure 1].

On the 2nd day, an epidural blood patch (EBP) was performed because the patient’s symptoms still did not improve. A 20-gauge Tuohy needle (Portex®, Smiths Medical) was used to administer 10 ml of autologous blood through an L3–4 epidural space. After EBP, a headache (numeric rating scale, level 6) regardless of the patient’s posture was continued. After that, a headache improved gradually following bed rest and hydration.

On the 7th day, a computed tomography scan was performed; the amount of SDH decreased, and there were no other abnormal organic findings that may have induced headache [Figure 2]. The patient was discharged because his headache was mitigated with no other neurologic symptoms.

Discussion

HIV infection is consistently on the rise in the world. According to the UNAIDS, the number of people infected HIV in the world reported annually burgeoned from 9,000,000 cases in 1990–34,900,000 cases in 2011. HIV infection is accompanied by an array of symptoms depending on the stage of the disease, including opportunistic infections in the respiratory, cardiovascular, and hepatobiliary systems due to reduced immune function induced by cell-mediated immunity as well as malignant tumors and hemodynamic instabilities.

During a preoperative evaluation, immune status (CD4+ cell count) of the patients and the presence of an opportunistic infection and malignant disease should be evaluated. The method of anesthesia and operation for an HIV-infected patient must be carefully determined only after a comprehensive review of the data. In this case, spinal anesthesia was performed only after the confirmation of normal platelet count and coagulation.

According to Chow et al., studied spontaneous intracranial hemorrhages in HIV-infected patients, the frequency of intracranial hemorrhage among patients not infected with...
HIV was 1.23 persons per 1000 persons, while that among HIV-infected patients was 2.29 persons per 1000 persons. The higher frequency among HIV-infected patients is thought to result from a pathologic change in the intracerebral vessels due to HIV infection. HIV-related vasculopathy is induced by vascular wall damage as a result of viral-induced inflammatory responses. The glycoprotein gp120, a viral envelope glycoprotein component on the surface of the HIV, promotes the production of a pro-inflammatory mediator that damages endothelial cells. The release of interleukin-1 (IL-6), and IL-8, tumor necrosis factor-α facilitated by HIV infection induces inflammation and escalates HIV replication. Moreover, Tat protein, an HIV transactivator of transcription protein, promotes inflammatory responses in vessels, multiplying the production of cytokines and adhesion molecules. Pulsatile blood flow and growth factors also contribute to the damage of vessels. Such a wide array of immune mechanisms beget vasculopathy in small vessels, and perivascular dilatation, pigment deposition, vessel wall mineralization, and perivascular cell infiltration can occur with no signs of vasculitis. Progressive vessel dilatation occurs in large vessels, and aneurysms may develop. These changes in vessels interfere with the autoregulation of the brain, significantly increasing risk of bleeding. As noted, HIV-infected patients are more prone to spontaneous intracranial hemorrhage. Furthermore, there are some abnormalities in the coagulation cascade. The production of autoantibodies directed against platelets and circulating immune complex deposited on the platelet membrane. It is possible to produce a bleeding and thrombosis in HIV patients. A prolonged partially activated thromboplastin time test, the production of a lupus anticoagulant and anticardiolipin antibodies, and several abnormalities in the natural-occurring anticoagulants are most common.

Therefore, it is essential that anesthesiologist promptly recognizes and treat symptoms of a headache, nausea, and vomiting in HIV patients.

Among the complications of spinal anesthesia, intracranial hemorrhage was mostly reported in association with myelography. Few studies have reported SDH-associated with simple spinal anesthesia, and its accurate frequency with regards to spinal anesthesia has not been reported. SDH occurred after spinal anesthesia is known to develop as a result of damage in the veins due to traction between the brain and the subdural veins. This is a result of separation of the arachnoid membrane and dura membrane due to reduced CSF pressure, which is in turn caused by excessive CSF leakage through the dura puncture. However, the factors associated with this process are not clear.

In this case, because the patient was infected with HIV, did not respond to NSAIDs, and did not improve with bed rest, we suspected that the patient’s headache was not a PDPH. Therefore, we performed more tests and treatments. The results of imaging tests showed a small amount of SDH. We believe sufficient to conservative treatment such as bed rest, fluid management, and NSAIDs. However, we performed an EBP because HIV infection can alter the natural course of an SDH and the size of the hemorrhage can be enlarged if there is CSF leakage.

A headache that arises after spinal anesthesia in HIV-infected patients can be a PDPH or can even be caused by other factors, such as central nervous system infection or an intracranial hemorrhage. Therefore, anesthesiologists should closely observe the changes and developments of a headache before diagnosing PDPH. If appropriate management is not provided cause neurological damage and may also result in life-threatening situations; hence, it is imperative that anesthesiologists perform exhaustive neurologic examinations and imaging for an accurate diagnosis due to HIV infection can alter the course of natural clinical development. Although spinal anesthesia is not contraindicated in patients with HIV patients, before underwent spinal anesthesia, considering the complications that may arise in the future and I hope that other anesthetic methods implemented by contemplating once more.

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Conflicts of interest
There are no conflicts of interest.
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