Remote Cerebellar Hemorrhage Presenting with Cerebellar Mutism after Spinal Surgery: An Unusual Case Report

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Dural injury during spinal surgery can subsequently give rise to a remote cerebellar hemorrhage (RCH). Although the incidence of such injury is low, the resulting hemorrhage can be life threatening. The mechanism underlying the formation of the hemorrhage is not known, but it is mostly thought to develop after venous infarction. Cerebellar mutism (CM) is a frequent complication of posterior fossa operations in children, but it is rarely seen in adults. The development of CM after an RCH has not been described. We describe the case of a 65-year-old female who lost cerebrospinal fluid after inadvertent opening of the dura during surgery. Computerized tomography performed when the patient became unable to speak revealed a bilateral cerebellar hemorrhage.

Key Words: Cerebellum · Hemorrhage · Mutism · Spine.

INTRODUCTION

The development of a remote cerebellar hemorrhage (RCH) is a rarely seen but important life-threatening complication. Chadduck first described an RCH in 1981.1 All previous RCHs were due to inadvertent opening of the dura during surgery.7-9,11 The incidence of inadvertent dural opening is reported as 3.5–17.4%.13 The incidence of RCHs after lumbar spinal operations is 0.08%.3 Cerebellar mutism (CM) refers to a reduced or complete loss of the ability to speak. In addition to mutism, ataxia, hypotonia, and emotional lability have been observed in patients with CM. The mutism may be temporary, or it can be permanent, with long-term effects.10 CM has mostly been reported after posterior fossa tumor operations. There are no previous reports of mutism presenting with RCH. We describe a rare presentation of CM following an RCH in a patient following surgery.

CASE REPORT

A 65-year-old female patient presented to our clinic with lower back and left leg pain. L4–S1 spinal stenosis and segmental instability were identified on lumbar magnetic resonance imaging (MRI) and dynamic graphic scans of the patient. As a result, L4–S1 posterior transpedicular fixation and L4–5 total laminectomy were completed. During the operation, the dura was accidentally cut, and primary dural repair was performed. The patient woke from the anesthesia and was taken to the ward. A neurological examination was nor-
normal. Four hours later, the patient was unable to speak and appeared confused. She was also unable to walk and began vomiting. Although unable to speak, the patient was able to respond to verbal commands. Computerized tomography (CT) of the brain revealed a bilateral cerebellar hemorrhage. The hemorrhagic area showed a typical zebra sign appearance. Air and lateral ventricle dilatation were observed in the frontal parasagittal area at the bilateral interhemispheric and suprasellar levels (Fig. 1). Four days later, the patient regained the ability to speak, although she showed dysarthria. Ten days later, her speech was completely normal. One month later, the patient’s neurological examination and walking were completely normal. CT performed in the second month of follow-up revealed no hemorrhage in the affected area (Fig. 2).

DISCUSSION

After cranial and spinal operations, cranial hemorrhages remote from the area of surgery may develop. The hemorrhages

Fig. 1. A bilateral cerebellar hemorrhage (zebra sign), frontal and suprasellar pneumocephalus, and slight ventricular dilatation observed on postoperative CT: computerized tomography.

Fig. 2. Follow-up CT performed 2 months after the surgery showing no hemorrhage and a reduction in the ventricular size to a normal level. CT: computerized tomography.
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In 1981, Chadduck first described the development of an RCH after a spinal operation. After that, there are only a few cases of remote cranial hemorrhage that have been reported due to the rarity, although it might result in fatal condition.

The reported incidence of remote hemorrhages after spinal surgery (0.08%) may be higher than thought, as some cases may remain asymptomatic. According to one series, the prevalence of asymptomatic RCH was 0.8%. The rate of asymptomatic cases is higher because imaging is carried out only in patients with symptoms.

Various factors, such as age, gender, pathology, and the type of surgery, have been put forward in the literature to explain the development of RCHs. However, there is no consensus on the exact mechanism. The common characteristic among nearly all RCH cases is inadvertent dural opening and subsequent cerebrospinal fluid (CSF) leakage. The incidence of this well-known complication is between 3.5 and 17.4%. RCHs are thought to have a venous origin. The latter is supported by data showing that the arterial bleeding is unilateral and that the hemorrhage is in the upper vermis and cerebellar sulcus, the drainage area of the cerebellar veins. Another hypothesis is that CSF loss causes caudal prolapse in the cerebellum, resulting in the development of cerebellar venous occlusion. This situation may cause secondary venous rupture and give rise to a hemorrhage in cases where reperfusion reoccurs or the venous pressure increases. In general, CT of the brain in cases of RCHs reveal typical zebra sign images, with hypodense and hyperdense irregular wavy lines resembling zebra skin.

CM is rare and is generally observed only in children after operations for posterior fossa tumors. The rate of CM in 500 adults following posterior fossa operations was 1.2%. In children after medulloblastoma operations, the incidence of CM was 24%. There are no reports in the literature of the development of RCHs presenting with CM after spinal operations.

CM refers to a reduced or complete loss of speaking ability. In addition to mutism, ataxia, hypotonia, and emotional lability, there have been observed in CM. The mutism may be temporary or it may be permanent, with long-term effects. Our case had temporary CM. The pathophysiological mechanism and anatomic correlation of CM are not well understood. CM is thought to develop after lesions in bilateral paravermian structures involving the dentate nucleus, vermis, lateral cerebellar hemisphere, and cerebrocortical pathways. In our case, we think that the CM depends on affecting the bilateral paravermian structures.

Generally, conservative medical treatment is sufficient for RCHs, and the long-term prognosis is good. However, some cases may require craniotomy of the posterior fossa and ventricular shunt surgery.

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Generally, conservative medical treatment is sufficient for RCHs, and the long-term prognosis is good. However, some cases may require craniotomy of the posterior fossa and ventricular shunt surgery.

Usually, a headache is the most common symptom of CSF leakage. The patient should be monitored closely if a headache is accompanied by neurological complaints, and a neurological examination should be performed. As an intracranial hemorrhage is a life-threatening complication, RCH is to be suspected for patients having inadvertent dural injury. If those patients show any neurological changes, immediate imaging is recommended, in order to reduce mortality via the early diagnosis and treatment of the RCH.
CONCLUSION

RCH is to be considered in case of neurologic changes after spinal operations having dural injury. As RCHs can occur some time after the surgery, the patient’s neurological status should be carefully monitored. As an RCH is a potentially fatal complication, it is important that the treatment begin as soon as possible. In addition, RCH might be revealed as cerebellar mutism, probably by affecting paravermian structures, as described in our case.

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