A Case of Unknown Origin Subarachnoid Hemorrhage Immediately Following Drainage for Chronic Subdural Hematoma

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Summary: A 56-year-old man treated with anticoagulants complained of a gradually worsening headache. A left chronic subdural hematoma (CSH) was shown by head computed tomographic (CT) scans and the operation, one burr hole surgery under local anesthesia, itself was performed uneventfully. However, immediately after we began draining the hematoma at the patient’s bedside, the patient complained of a sudden headache. CT scans showed a subarachnoid hemorrhage (SAH). Cerebral angiography was immediately performed, but the source of hemorrhage could not be found. The next day, a CT scan showed that most of the SAH had disappeared. To our knowledge, there are no previous reports of SAH of unknown origin following surgery for CSH. The likely mechanism for the occurrence of the SAH, in addition to a coagulopathy due to anticoagulant therapy, could include the possibility that the drainage of the hematoma produced a movement of the hemisphere along with hyperperfusion that resulted in the rupture of a weak subarachnoid vessel, such as a perforating artery.

Key words chronic subdural hematoma, burr hole surgery, drainage, complication, subarachnoid hemorrhage

INTRODUCTION

Chronic subdural hematoma (CSH) can be cured by comparatively easy burr hole surgery. However, some severe complications, such as intracerebral hematoma and acute subdural hematoma, have been reported. We report a case of a subarachnoid hemorrhage (SAH) of unknown origin occurring immediately after we began draining the CSH. On computed tomographic (CT) scans this SAH was washed out at the early stage. To our knowledge, there are no previous reports of SAH of unknown origin after the surgical treatment of a CSH. The possible etiology of the SAH is discussed.

CASE REPORT

A 56-year-old male was admitted to our hospital with a history of cerebral infarctions being treated with anticoagulants (warfarin: 2 mg/day, ticlopidine: 200 mg/day). He complained of a headache that had developed over the previous few days. Neurological examination revealed that the patient had a Glasgow Coma Scale score of 15, a slight right hemiparesis and dysarthria. A CT scan revealed a left CSH with a shift of the midline structures (Fig. 1A). On laboratory examination, his International Normalized Ratio (INR) was 2.80. A closed-system drainage of the hematoma was performed via one burr hole under local anesthesia after the INR had fallen to 1.97 with...
Fig. 1. CT scans on admission (A) shows a left CSH. After the operation, the left CSH is being removed, however an SAH is imaged focusing on the right perimecencephalic and Sylvian cistern (B). The day after operation, most of the SAH has disappeared (C).

vitamin K. The operation itself was performed uneventfully, and the patient’s blood pressure was well controlled. However, immediately after we began draining the hematoma at the patient’s bedside, the patient complained of a sudden headache. A CT scan showed an SAH focusing on the right perimegencephalic and Sylvian cistern (Fig. 1B).

Cerebral angiography was immediately performed, but the source of the hemorrhage could not be found (Fig. 2). However, a left middle cerebral artery (MCA) occlusion, a transdural anastomosis, and a leptomeningeal anastomosis from the left anterior cerebral artery (ACA) and the posterior cerebral artery were shown (Fig. 3). The next day, the CT scan showed that most of the SAH had disappeared (Fig. 1C). After that, there were no further symptoms of rebleeding or vasospasm. The clinical course was good and the patient was discharged.

DISCUSSION

CSH can be cured by comparatively easy burr hole surgery. However, some severe complications have been reported. Mori et al. [1] examined 500 consecutive surgically-treated CSH cases and reported that 13 cases developed an acute subdural hematoma and 4 cases developed tension pneumocephalus. Moreover, there have been reports of other complications, including intracerebral hematomas [2,3], epidural hematomas [4], and the hyperperfusion syndrome [5]. However, to our knowledge, there are no previous reports of SAH of unknown origin occurring immediately after the surgical treatment of CSH.

Pituitary apoplexy can cause a SAH of unknown origin. On CT scan, however, there was no evidence to support this diagnosis. Cocaine abuse and sickle
cell disease were also negative in the patient’s past history. The fact that the hematoma was almost washed out on the next day suggested that the course was compatible with a traumatic SAH or a non-aneurysmal SAH.

Brodersen et al. [6] have reported that cerebral blood flow (CBF) falls before surgery, and then increases after surgery, as shown by intraarterial 133 Xenon CBF studies of CSH patients. Koike [7] has shown experimentally that leaky bleeding can arise in intraparenchymal brain tissue due to rapid hemodynamic changes. Thus, if CBF or cerebral autoregulation is impaired due to long-term compression by a CSH, and a decrease of intracranial pressure occurs due to drainage of a hematoma, hyperperfusion can arise. In this case, the blood flow of both ACAs was dominantly supplied from the right side. Furthermore, a leptomeningeal anastomosis from the left ACA to areas of the occluded left MCA existed. Therefore, one could postulate that hyperperfusion occurred in the A1 segment of the right ACA. Tatter et al. [8] reported two cases of SAH that might have been caused by rupture of a perforating artery. In our case, it is reasonable to assume that the hyperperfusion

*Fig. 2. Right common carotid angiography. Anteroposterior (A) and lateral (B) view do not reveal the source of hemorrhage, such as aneurysm, arteriovenous malformation, nor do right internal carotid angiography of right (C) and left (D) anterior oblique 30°.*
Fig. 3. Left common carotid angiography. Anteroposterior (A) and lateral (B) view show a MCA occlusion, a transdural anastomosis and a leptomeningeal anastomosis, but do not reveal the source of hemorrhage, nor do anteroposterior (C) and lateral (D) view of left vertebral angiography.

occurred immediately after the drainage procedure, and a weaker perforating artery from the right A1 segment ruptured.

In this case there was a history of a coagulation disorder secondary to the anticoagulant therapy. Mattle et al. [9] reported three cases that had no possible cause for an SAH other than anticoagulant therapy. In our case, the coagulopathy was controllable. Therefore, it is not possible that the coagulopathy was the sole cause of bleeding. However, since the INR was still elevated after administration of vitamin K, the coagulopathy might have promoted the bleeding.

On the other hand, Kotwica et al. [10] reported six cases of CSH that presented with the clinical findings of an acute SAH. The cause of the SAH was considered that a sudden increase in the intracapsular pressure could lead to rupture of the arachnoidea with bleeding from the hematoma into the subarachnoid space. Our case presented with the CSH in the left, but the SAH was in the right. It is unlikely that the CSH could extend to the subarachnoid space of the contralateral side.

The etiology of the SAH in our case could be, in
addition to the coagulopathy due to anticoagulant therapy, the drainage of the hematoma that caused movement of the hemisphere and resulted in hyperperfusion with the rupture of a weak subarachnoid vessel. Since the SAH was located in the right perimesencephalic and Sylvian cistern, it is possible that the rupture of a perforating artery of the right ACA (A1 segment) was the cause of the SAH. If an occlusion or stenosis of a major cerebral artery is present, then even greater hyperperfusion caused by the drainage of the CSH may occur in the artery which is the main blood flow supply. In a patient with a history of cerebral infarction and transient ischemic attack it could be seen to be prudent to do magnetic resonance angiography and other investigations before the operation. On the other hand, comparatively quick and easy burr hole surgery was also required on a timely basis. The CSH maintained a sort of balance intracranially by compressing the hematoma over a long period of time. One should avoid sudden decompression in the treatment of CSH and carefully tailor the treatment to the patient’s individual condition.

REFERENCES