

Re-occlusion of the superior sagittal sinus after surgical recanalisation

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Summary A 24-year-old woman was struck on the head by a hammer. Because of early signs and symptoms of intracranial hypertension, she underwent surgery for elevation of the depressed fragments which was compressing the superior sagittal sinus (SSS). After operation, the intracranial pressure (ICP) once decreased, but it gradually increased again. After hypothermia and barbiturate therapy, she recovered fully except for partial visual field defect due to brain contusion. A carotid angiogram 28 days after injury revealed complete occlusion of the whole SSS with good collateral circulation. After brain edema had subsided, a follow-up angiogram revealed normal blood flow through the SSS. Elevation of depressed bony fragments is required for a case presenting with early signs and symptoms of intracranial hypertension due to sinus compression. In a case with severe destruction of the SSS, one needs to know that re-occlusion of the dural sinus may occur after surgical recanalisation.

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INTRODUCTION

There are several reports on cases with intracranial hypertension due to superior sagittal sinus (SSS) compression secondary to depressed skull fractures presenting with the early signs and symptoms. In those cases, symptoms improved after elevation of the bony fragments.^{1–4} Here we present a rare case of woman who underwent surgery (elevation of the bony fragments); the intracranial pressure (ICP) once decreased, gradually increased and the SSS occluded again.

CASE STUDY

A 24-year-old woman was struck on the head by a hammer in a criminal act. The Glasgow coma score (GCS) on admission was 14. She showed bilateral diffuse and dappled visual field defect. A computed tomography (CT) scan demonstrated depressed fracture in the occipital area with bony fragments overlying the

posterior third of the SSS (Fig. 1). Three hours after the injury, her GCS decreased down to 6; the right pupil was dilated with left hemiplegia. A CT scan demonstrated diffuse brain swelling without significant intraparenchymal hematoma. She underwent surgery to elevate the depressed fragments which was compressing the SSS. Before the elevation of the bony fragments, the posterior part of the SSS was confirmed to be occluded with an ultrasonic doppler and direct vision. Several cortical veins were also found to be injured and occluded. When the depressed fragments were removed, profuse bleeding occurred from the largely lacerated sinus and several cortical veins. Hemostasis was accomplished by placing Oxycel cotton and fibrin glue; several cortical veins were sacrificed. After the operation the ICP dropped below 10 mmHg, and remained below 20 mmHg for the following 2 days with hypothermia and barbiturates therapy. However, it increased and became uncontrollable after the third day (Fig. 2). On the fifth day after operation, the ICP exceeded 60 mmHg for 30 min. Surgery was not carried out because the patient's general condition was poor with disseminated intravascular coagulation syndrome. On the 13th day, she recovered neurologically intact



Fig. 1 A CT scan on admission showing a depressed fracture overlying the superior sagittal sinus.

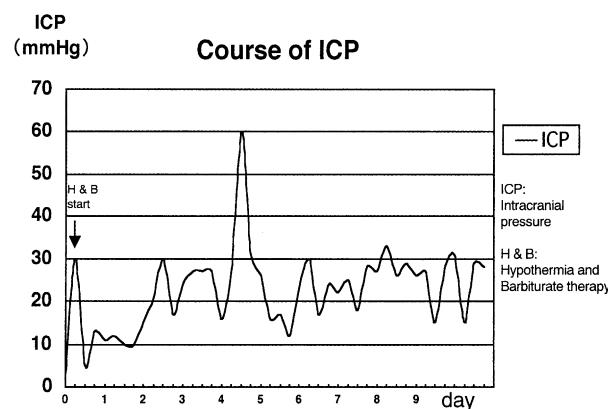


Fig. 2 The course of the intracranial pressure (ICP) after operation.

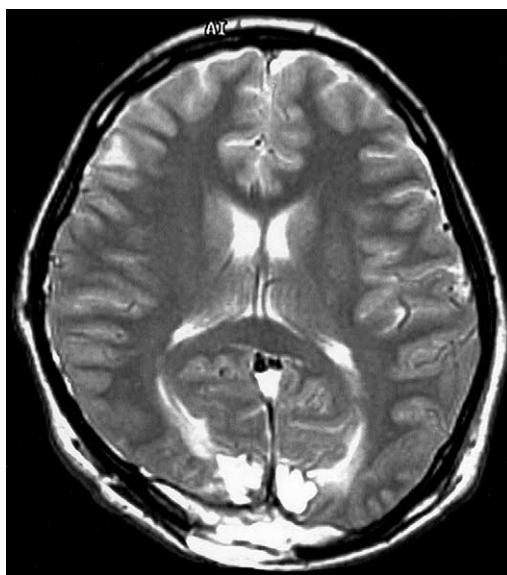


Fig. 3 An MRI (18 days later) showing contusion and edema in bilateral occipital lobes.



Fig. 4 The internal carotid angiogram (18 days later) showing complete occlusion of the superior sagittal sinus with congested superior cerebral veins.

except for partial visual field defect. An MRI 18 days after injury revealed contusion and edema in bilateral occipital lobes (Fig. 3). An angiogram 28 days after injury revealed no blood flow in the SSS. Collateral circulation from the parietal part of the cortical vein developed into the Sylvian vein (Fig. 4). An angiogram 6 months after injury revealed normal blood flow through the SSS with partial stenosis, and collateral circulation disappeared completely (Fig. 5). She returned to her previous job.

DISCUSSION

Generally, thrombosis arises as a result of (1) structural changes in the vessel wall, (2) stasis of blood flow, (3) hypercoagulability of blood. Cerebral venous thrombosis, however, rarely arises only as a result of blood stasis in the vessel, because fibrinolysis is activated in normal venous wall, and even once thrombosis occurs it easily disappears.⁵⁻⁷ In the SSS, which is firmly supported by surrounding dura mater and fixed to the cranium tightly, structural change of the wall is unlikely to occur. Even if the SSS is compressed secondary to depressed skull fracture, intracranial hypertension may easily be relieved with elevation of the bony fragments. There have been 4 reports in which the SSS was occluded by bony depression and symptoms were relieved after removal of the bony fragments compressing the SSS. In all cases, symptoms due to intracranial hypertension improved only with elevation of bony fragments and re-occlusion did not occur.¹⁻⁴ In the present case, however, the SSS re-occluded after surgical recanalization. Re-occlusion may have occurred due to several reasons; (1) direct compression of the SSS by the surrounding edematous brain since the surrounding supporting structures were destroyed by bony fractures, (2) structural change of the wall of the SSS due to bony fractures induced obliteration of the SSS itself, (3) hypercoagulability due to disseminated intravascular coagulation syndrome.

It remains unknown when the SSS re-canalised. However, several mechanisms are considered: (1) collateral circulation might gradually develop during extensive treatment with hypothermia and barbiturate administration and (2) sinus thrombosis might spontaneously dissolve with improvement of the patient's general condition.

CONCLUSION

Emergent elevation of depressed bony fragments is required for a case presenting with early signs and symptoms of increased intracranial pressure secondary to depressed skull fracture.

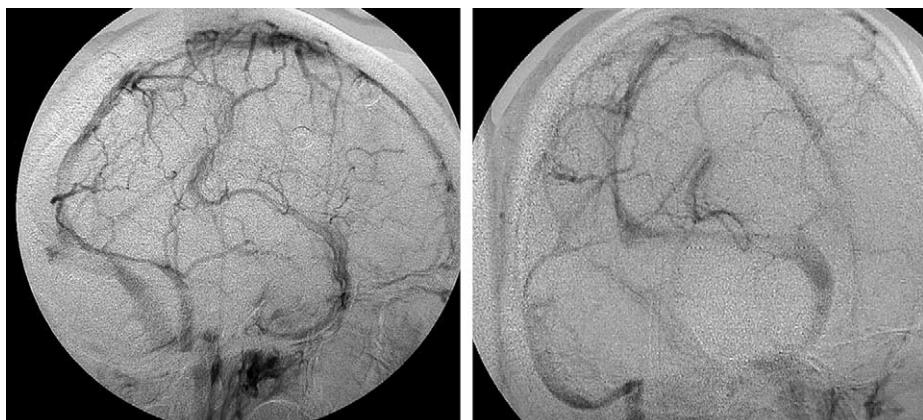


Fig. 5 The internal carotid angiograms (6 months later) showing normal blood flow through the SSS with partial stenosis. Left: lateral view; right: oblique view.

After recanalization of the SSS, however, it may re-occlude as in the present case. This is a rare case in which surgically recanalized SSS re-occluded during the postoperative course. One needs to know that re-occlusion of the SSS may occur possibly due to a change in the surrounding structure and hypercoagulopathy.

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A giant fusiform basilar aneurysm treated by bilateral vertebral artery occlusion

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Summary *Objective and importance.* Fusiform aneurysms of the vertebrobasilar arteries that progressively enlarge causing symptomatic brainstem compression are dangerous and their treatment is difficult. A patient with such an aneurysm treated successfully with staged, microsurgical occlusions of the proximal vertebral arteries is described, and the literature pertaining to this rare condition is briefly reviewed. *Clinical presentation.* A 48-year-old man with a fusiform basilar trunk aneurysm of uncertain etiology presented initially with transient ischemic attacks (TIAs) of the posterior circulation that ceased with anticoagulation. Four years later he presented again with progressive ataxia, dysphagia and dysphonia due to considerable enlargement of the aneurysm causing brainstem compression. *Intervention.* Staged microsurgical vertebral artery occlusions proximal to the aneurysm were performed. The second (left) vertebral artery was clipped only after the patient passed its temporary occlusion with an endovascular test balloon. The aneurysm subsequently thrombosed, the distal basilar artery kept patent by a single (left) posterior communicating artery. The patient's clinical condition improved markedly over a number of months as the aneurysm mass atrophied. *Conclusion.* Giant vertebrobasilar aneurysms are rare but treacherous lesions, sometimes justifying aggressive management. Carefully selected patients with progressive and severe symptoms due to brainstem compression may tolerate proximal vertebral artery occlusions, provided there is adequate collateral flow to the basilar termination and all of its perforating branches.

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INTRODUCTION

It has been estimated that as many as 80% of patients with symptomatic and untreated giant aneurysms become severely disabled or die within 5 years of presentation.¹ Presenting most commonly with either brainstem compression (including blockage of cerebrospinal fluid [CSF] flow and hydrocephalus) or ischemia,² these aneurysms can also rupture.³

Elimination of these aneurysms is difficult because their fusiform morphology usually prevents maintenance or reconstruction of the parent vessel, leaving as the only option proximal parent arterial occlusion (sometimes referred to as "Hunterian ligation" in older literature).³ We present the case of a man who had become unable to walk, maintain a safe airway, or swallow due to severe brainstem compression by a large and growing vertebrobasilar fusiform aneurysm, who was treated successfully with bilateral vertebral artery occlusion.

CASE PRESENTATION

A 48-year-old Caucasian male presented in 1998 after four discrete episodes of ataxia combined with left-sided weakness, each attack lasting minutes and resolving completely. The patient was a



Fig. 1 Axial CT scan of the posterior fossa and base of the brain showing a fusiform and partly calcified aneurysmal dilatation of the basilar artery.