

Traumatic open depressed cranial fracture causing occlusion of posterior superior sagittal sinus

Case report

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Abstract

Rationale: The superior sagittal sinus (SSS) is the major dural sinuses that receive a considerable amount of venous drainage. Interruption of its posterior third has been suggested to cause intracranial hypertension and lead to potentially fatal consequences.

Patient concerns: We presented a 22-year-old man with a severe headache and scalp bleeding after a head chop wound. Physical examination identified a 20-cm straight laceration in his parietooccipital scalp. Computed tomography (CT) demonstrated a depressed cranial fracture (DCF) in the left parietooccipital bone, a fracture line across the midline to the right side, and penetrations of bone fragments into the brain parenchyma.

Diagnoses: Traumatic open DCF in left parietooccipital bone.

Interventions: An emergent left parietooccipital craniotomy, followed by cranioplasty to restore the depressed bone flap, was delivered to the patient. Postoperative CT confirmed successful elevation of the DCF and removal of intracerebral bone fragments. However, postoperative CT angiography (CTA) demonstrated an absence of venous flow distal to the fracture, suggesting occlusion of the posterior third of SSS. MRV revealed a persistent absence of venous flow in the posterior third of SSS with dilated cortical venous drainage. Anticoagulation treatment was initiated 3 days after surgery, and follow-up CTA and digital subtraction angiography showed gradually improved patency in the anterior and middle two-thirds of SSS.

Outcomes: Despite occlusion of the posterior third of SSS, patient's symptoms resolved after the operation and he was discharged without complications.

Lessons: The favorable clinical outcome after complete occlusion of the posterior third of the SSS has rarely been reported and it might be explained by our timely surgical intervention and development of compensatory cerebral collateral circulation.

Abbreviations: CT = computed tomography, CTA = computed tomography angiography, DCF = depressed cranial fracture, DSA = digital subtraction angiography, ICH = intracranial hypertension, ISS = inferior sagittal sinuses, MRV = magnetic resonance venography, SSS = superior sagittal sinus.

Keywords: CT angiography, digital subtraction angiography, magnetic resonance venography, open depressed skull fracture, superior sagittal sinus

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H-SS, FS, and JL conceived and wrote the manuscript. G-HB, F-CL, and D-DL did data collection and contributed to manuscript writing. NZ did the review and final approval of the manuscript.

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1. Introduction

Superior sagittal sinus (SSS) is the most commonly affected location (70–80%) among head trauma patients who are diagnosed with dural sinus injury.^[1] One common etiology for SSS injury is by depressed cranial fractures (DCFs) that overly the major venous sinus.^[2,3] For closed DCFs, nonsurgical management is recommended to avoid the associated risks of sinus bleeding or occlusion.^[4–6] Moreover, surgical intervention involving major sinus are suggested to be postponed until there is evidence of active bleeding or development of intracranial hypertension (ICH).^[7,8] Nevertheless, in cases with compound/open DCFs or when the posterior third of the SSS is affected, surgical intervention is warranted with the aims of the closure of the dura tear and prevention of the occurrence of fatal ICH.^[9] The authors present here a 22-year-old male with an open DCF in the left parietooccipital bone and the fracture extended across the posterior third of the SSS to the right parietal bone. Given the open nature of this injury, an emergent surgical intervention was delivered for wound debridement, removal of intracerebral bone fragments and restoration of the depressed skull. Although complete occlusion of the posterior third of the SSS was confirmed by postoperative angiography, the patient recovered well without delayed ICH or other complications.

2. Case presentation

A 22-year-old man presented with severe headaches and occipital scalp bleeding after a chop wound to his head by a sharp knife. He was first admitted to a local hospital where wound debridement and closure were performed before referral to our hospital 15 h after the initial injury. On admission to our hospital, he complained about a headache and physical examination showed he was conscious and alert (Glasgow coma scale = 15), with normal pupil size and light reaction in both eyes, and had a 20-cm straight wound at his parietooccipital scalp which had already been closed by sutures (Fig. 1A). The ophthalmological evaluation revealed normal vision and no visual field defects, abduction deficits, or optic disc edema in both eyes. There were no other noticeable pathological findings. Then the patient was referred to our neurosurgery department, and a diagnosis of open DCF with potential SSS injury was proposed.

Cranial computed tomography (CT) scans demonstrated a DCF in the left parietooccipital skull with the fracture line ran across the distal one-third of the SSS to the right parietooccipital bone (Fig. 2A and B). There were also multiple fractured bone fragments that penetrated the brain parenchyma and linear cerebral contusions in the parietal lobes which across both hemispheres. However, ICH signs like cerebral edema, cortical swelling, or hemorrhagic infarction that usually secondary to venous hypertension/congestions were not observed.

To remove the intracerebral bone fragments, restore the depressed bone and repair the dura tear, an emergent left parietooccipital craniotomy was performed 18 h after injury. The patient was placed in lateral position with all pressure points padded. Following removal of the wound sutures, some brain tissues were found to herniate through the bone fractures (Fig. 1B). After placement of a self-retaining retractor on the skin margins, we identified a linear bone fracture, about 1 cm in wideness, that ran across both left and right parietooccipital bones and an SSS injury was suspected (Fig. 1C). The craniectomy was completed by using the 2 burr holes on the left side of the SSS (Fig. 1D). Two additional burr holes at the right side of SSS were made for potential SSS reconstitution in the case of active sinus bleeding, which did not happen in our

patient. After removal of the fractured bone flaps, a 10-cm dural laceration and herniation of cerebral tissues through the dura opening were noticed. Three small pieces of bone fragments were then removed from the contused brain parenchyma. However, in consideration of the patient's mild symptoms and lack of radiological evidence suggesting ICH, SSS repair was not attempted as it also carries additional risks such as venous bleeding. After complete debridement and removal of devitalized brain tissues, routine dual tack-up sutures were placed at the craniotomy margin, and duraplasty was performed with periosteum (Fig. 1E). Cranioplasty was achieved with mini titanium plates, and the incision was closed in layers (Fig. 1F).

The patient woke up with relief of symptoms and was discharged 7 days after surgery. No complication was observed during the 9-month clinical follow-up. CT performed 1 day postoperatively showed complete removal of intracerebral bone fragments (Fig. 2C). CT angiography (CTA) performed 3 days postoperatively revealed distorted and compromised venous flow through the sinus lumen distal to the wound (Fig. 2D and E). Full anticoagulation treatment was initiated with the injection of low molecular weight heparin (CLEXANE) 40 mg by subcutaneous route every day for 12 days, which was then replaced with 100 mg oral aspirin intake for 2 months. MR performed 10 days postoperatively showed a subacute-phase thrombus (high signal on T1-weighted images and moderate signal on T2-weighted images, similar to the signals of adjacent brain contusion) in SSS and MR venography (MRV) confirmed the loss of patency in the posterior one-third of the SSS and part of the straight sinus, and formation of thrombosis (Fig. 2F–J). Follow-up CTA performed 45 days (Fig. 2K and L) and 7 months (Fig. 2M and N) postoperatively showed progressive improvements in the venous flow in the anterior and middle two-thirds of SSS and reduction in the volume of the thrombosis. Digital subtraction angiography (DSA) performed 8 months postoperatively demonstrated loss of patency in the posterior one-third of the SSS and drainage of venous blood to transverse sinus and sigmoid sinus via superior and inferior anastomotic veins (Fig. 2O).

Consents to conduct and report of this study were obtained from the Ethics Committee of Second Affiliated Hospital of

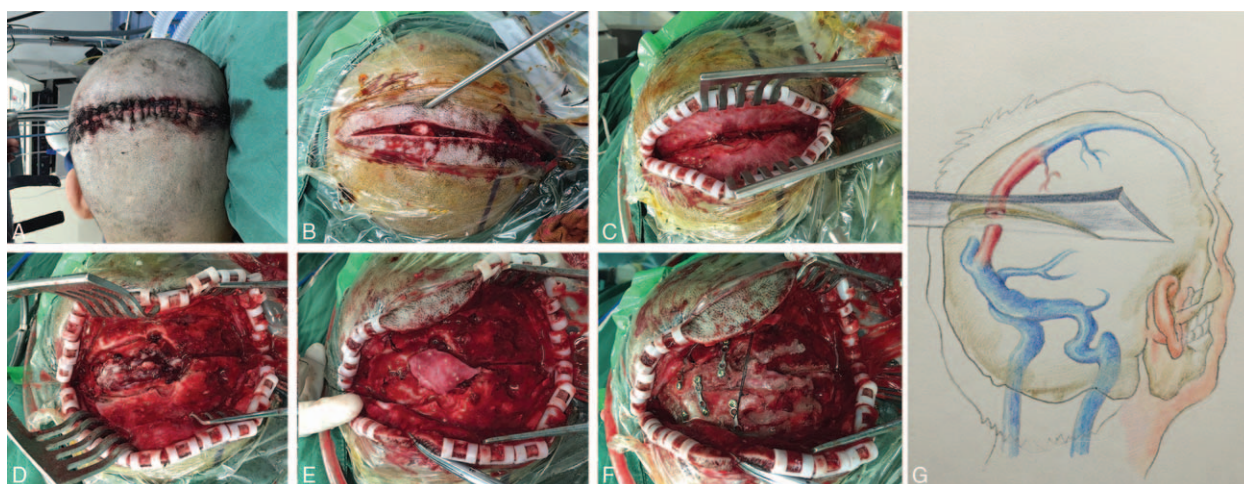


Figure 1. Intraoperative images of the patient with an open depressed skull fracture and superior sagittal sinus injury. (A) A 20-cm chop wound was identified at the patient's parietooccipital scalp, which had already been closed by sutures at a local hospital. (B) Following removal of the wound sutures, brain tissues were found to herniate through the left parietal bone fractures. (C, D) A self-retaining retractor was used to retract the skin margins, and 2 burr holes were made for the left parietooccipital craniotomy. (E, F) After complete debridement and removal of devitalized brain tissues, periosteum was used for duraplasty, and cranioplasty was achieved by using mini titanium plates. (G) A schematic illustration of the mechanism of superior sagittal sinus injury.

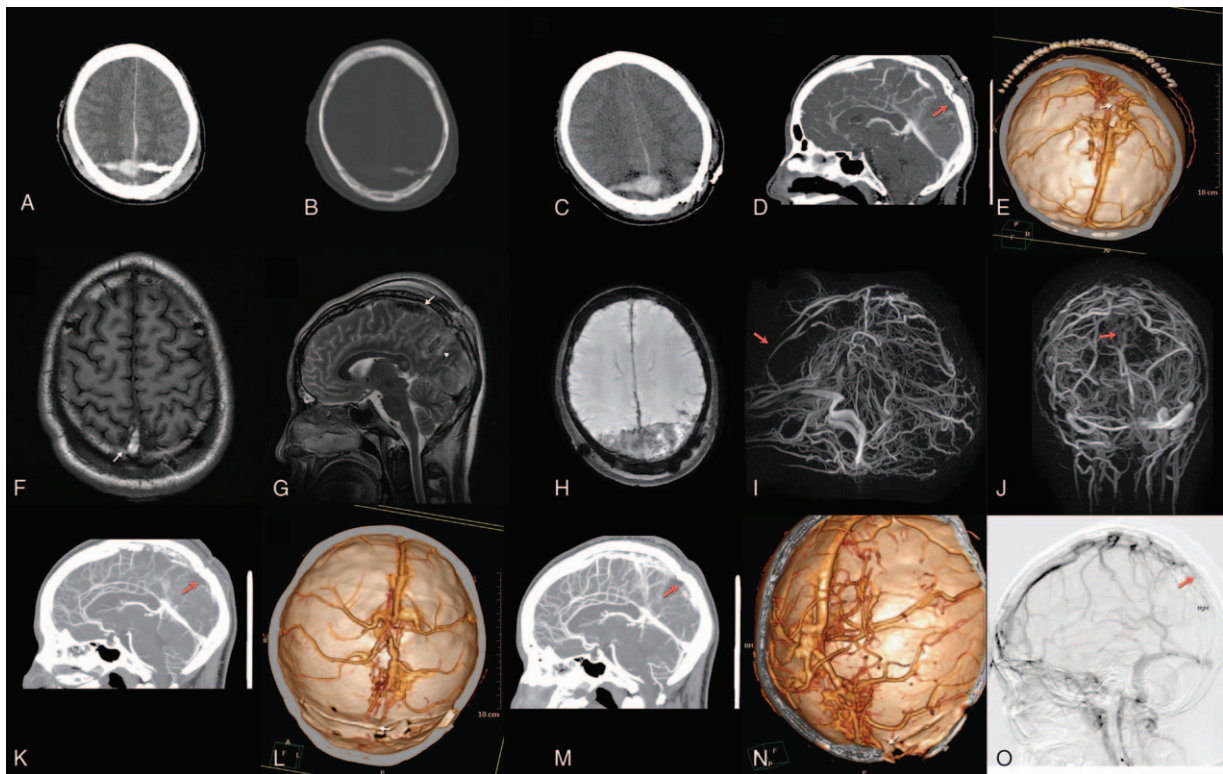


Figure 2. Neuroimaging of the patient with open depressed skull fracture (DCF) and superior sagittal sinus (SSS) injury. (A, B) Preoperative cranial CT scans demonstrated a DCF in the left parietooccipital skull with multiple fractured bone fragments that had penetrated the brain parenchyma and linear cerebral contusions in the parietal lobes of both hemispheres. (C) Postoperative CT performed (1 day) showed complete removal of intracerebral bone fragments. (D and E) CT angiography (CTA) performed 3 days postoperatively showed compromised venous flow through the posterior 3rd of SSS (arrows) and formation of thrombosis. (F and J) MR performed 10 days postoperatively suggested contusions in both parietal lobes and the corpus callosum on T1 FLAIR (F), T2WI (G), and SWI (H) sequences. The thrombosis within SSS had high signal on T1-weighted images (F: arrow) and a moderate signal on T2-weighted images (G: arrow for thrombosis, arrow head for contusion), suggestive of subacute phase hematoma. MR venography (MRV) confirmed block of venous flow in the posterior one-third of the SSS (arrows) and part of the straight sinus and formation of thrombosis (I and J). Follow-up CTA performed 45 days (K and L) and 7 months (M and N) postoperatively showed progressive improvements in the venous flow of the anterior and middle two-thirds of SSS and reduction in the volume of the thrombosis. Arrows indicate compromised venous flow through the posterior 3rd of SSS. (O) Digital subtraction angiography performed 8 months postoperatively demonstrated loss of patency in the posterior one-third of the SSS (arrow) and drainage of venous blood to transverse sinus and sigmoid sinus via superior and inferior anastomotic veins. CT = computed tomography, MR = magnetic resonance.

Wenzhou Medical University. Written informed consent regarding the publication of this case report and its accompanying images was obtained from the patient. Copies of the written consent are available for review upon request.

3. Discussion

In contrast with closed DCFs, where nonsurgical management is recommended to avoid the risk of intraoperative sinus bleeding,^[10] surgical interventions are usually needed in open DCFs to remove the potentially infectious foreign materials, closure of the dura tear, restore the depressed bone flaps, and reconstruction of the dural sinus when necessary.^[11] Another reason for surgical treatment of DCFs is to prevent the occlusion of major intracranial venous structures like SSS and subsequent development of ICH, which can potentially lead to fatal brain swelling and venous infarcts.^[12,13] Several surgical techniques have been proposed to decompress the DCFs and improve the venous flow in SSS.^[1,14,15] For example, the bone fragment could be left in place and drilled to an eggshell thickness when there was a high risk of venous sinus bleeding.^[7] Angiographic imaging like CTA and MRV are highly recommend in all patients presenting with

symptoms of increased intracranial pressure after DCFs for early diagnosis of SSS occlusion and appropriate surgical planning.^[2,15]

The most distinctive feature of the current case is the complete occlusion of the posterior third of the SSS without the development of ICH or associated morbidity and mortality. Given the nature of the chop wound and intraoperative finding (e.g., the wideness of the bone fracture), it was highly likely that the patient's SSS had been injured by the mechanic force of the knife (Fig. 1G). However, the thrombus might develop gradually as our patient did not show typical signs of ICH in a relatively long period (the interval between injury and surgery was 18 h) and postoperative magnetic resonance imaging confirmed the thrombus was in subacute phase. It is well recognized that scarification or disturbance of the anterior third of the SSS is generally tolerable, while interruption of the posterior third would inevitably provoke potentially fatal ICH, which cannot always be controlled efficiently with antiedema therapy.^[16] According to literature, about 20% of cases would develop severe ICH with progressive loss of vision and encephalopathy.^[17] However, given his mild symptoms and no intraoperative active sinus bleeding, we cautiously decided not to attempt intraoper-

ative SSS repair or thrombus removal. Instead, complete debridement with bone fragments removal and antibiotics treatments was delivered to reduce the risk of infection.

One possible explanation for his favorable clinical outcome is that effective compensatory cerebral collateral circulation might have developed in the subacute phase. For example, cortical venous system, such as the vein of Labbe and the transcerebral venous system,^[18] might partially explain the excellent result in our patient. Moreover, DSA suggested the blood from SSS was drained to transverse sinus and sigmoid sinus via superior and inferior anastomotic veins and those collateralized venous outflows were located anterior to the wound site. Besides, other venous sinuses in the falx cerebri have been identified in addition to the superior and inferior sagittal sinuses (ISS).^[19] These falcine venous sinus/plexus could communicate the blood between the SSS and ISS and thus help to drain the blocked venous flow.^[20] Second, as indicated by the herniation of the cerebral tissues through the DCF, the development of ICH in the acute phase might be prevented by the release of the intracranial pressure through the fractured bone and lacerated dura. This also makes time for the development of collateral circulation in the subacute phase. Finally, the anticoagulant therapy which was initiated 3 days after surgery might be helpful to resolve the thrombosis. However, anticoagulant therapy will also increase the risk of hemorrhagic complications, especially in trauma patients, and therefore its timing would be the determinant factor for whether benefit could outweigh the associated risks of bleeding.^[21–23] Similar to our case, another study commenced a therapeutic dose of unfractionated heparin 4 days after decompressive craniectomy and reconstitution of the SSS in a patient with parietal DCF, SSS rupture, and secondary thrombosis.^[24] Although the sinus thrombosis in the posterior third of SSS had not been resolved at the last radiological examination (8 months after surgery), there was a clear improvement in the venous flow in the anterior and middle third of the SSS.

One potential argument might be the SSS injury in our case was of iatrogenic origin. However, given the clinical, radiological, and intraoperative findings, it is highly likely patient's SSS was lacerated directly by the mechanical force of the initial chop wound. There is still controversy over the surgical indication and timing of intraoperative SSS reconstruction and thrombus removal in patients where major sinus injury has occurred. We propose it should be dependent on factors like location of the DCF, severity of SSS injury, the speed of thrombus development as well as patient's symptoms. In our case, there was no direct compression of the SSS by the DCF and there was no evidence of SSS rupture or active bleeding. Given the clinical course of this patient, that is, mild symptoms and no sign of ICH, the secondary thrombosis probably developed relatively slowly and we expected him to be at low risk of morbidity associated with ICH. Therefore, more aggressive management of the lacerated sinus like SSS reconstitution was not performed as these procedures would carry additional risks of iatrogenic injuries.^[1,7] Instead, meticulous intracerebral bone fragments removal and duraplasty with periosteum were performed to reduce the risk of complications like infection and CSF leak.

4. Conclusion

The development of ICH would depend on the severity as well as the location of SSS injury. Aggressive SSS reconstitution and thrombus removal might not be necessary in patient with no sign and symptoms of ICH. Angiography, such as CTA, MRV, and

DSA, demonstrated an improved hemodynamic feature of the SSS by our decompressive surgery and postoperative anticoagulant therapy. The occlusion of the posterior third of the SSS without the development of ICH might be explained by the slowly formation of thrombosis and a timely establishment of compensatory collateral circulation.

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