Depressed Skull Fracture Over Superior Sagittal Sinus: Is the Elevtion Rightly Cotraindicated?

Amit Thapa, MS, MCh
National Institute of Neurological and Allied Sciences (NINAS)
Bansbari, Kathmandu, Nepal

Ajit Shreshta, MS
National Institute of Neurological and Allied Sciences (NINAS)
Bansbari, Kathmandu, Nepal

Ramesh Man Joshi, MS
National Institute of Neurological and Allied Sciences (NINAS)
Bansbari, Kathmandu, Nepal

Upendra P Devkota, MS, FRCS
National Institute of Neurological and Allied Sciences (NINAS)
Bansbari, Kathmandu, Nepal

Address for correspondence:
Amit Thapa, MS, MCH
Assistant Professor
National Institute of Neurological and Allied Sciences (NINAS)
Bansbari, Kathmandu, Nepal
Email: dramitthapa@yahoo.com

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The classical teaching in depressed skull fracture is to treat conservativelythose which lie over superior sagittal sinus. This may be true in view of expected blood loss and skills required to handle the sinus bleed. However conservative approach in such case exposes the patient to the risk of cerebral venous thrombosis and consequent venous infarct.

We report here a case of a 27 year male who met with an accident. He had depressed parietal bone fracture over superior sagittal sinus. Initially he was managed conservatively; however on 10th day of his injury, he started developing spastic paraparesis with gradual involvement of upper limb with deteriorating consciousness level.

Computerized tomography scan showed depressed parietal bone fracture with development of bilateral venous infarct over parietal lobe. He was immediately explored and fracture segment elevated. Post operatively, his weakness and consciousness level gradually improved.

The recent literature has started questioning this long held belief of conservative care. If expertise of tackling the sinus bleed is available, one must elevate the depressed skull fracture in previously considered no man's land.

Key words: Cortical venous thrombosis, Depressed skull fracture, Superior Sagittal Sinus, venousInfarction

Elevation of depressed fracture which overlie venous sinus in 11- 16% cases, is controversial.Risk of fatal venous bleed on attempt to elevate such fractures has led to practice of conservative care in such cases ([1, 2]). During the last decade, literature is full of complications of conservative care and reports of successful attempts of elevation of depressed fracture leading to dramatic recovery ([3-6]).

We present a case report and review of literature on this controversial issue.

Case Report

A 27 years gentleman presented to our casualty following a road traffic accident (RTA). He was initially managed in peripheral hospital. As his Glasgow coma score (GCS) was 3, he was intubated, resuscitated and then referred to our referral centre. However on arrival to our casualty he was flexing to pain but his verbal and eye opening did not improve. He was haemodynamically stable. On local

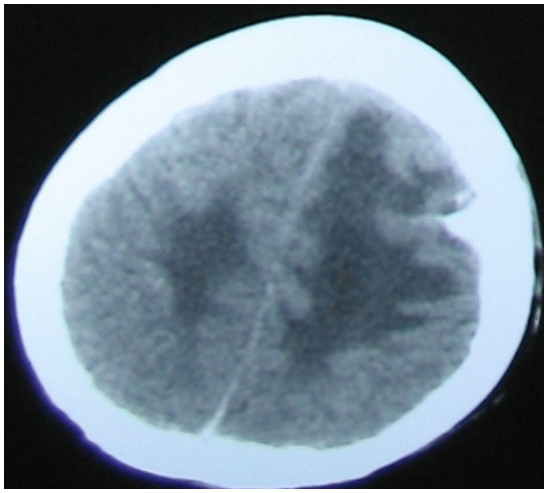

Figure 1. *Non contrast enhanced CT scan head showing venous infarct over bilateral fronto- parietal lobes with overlying depressed bone fracture*

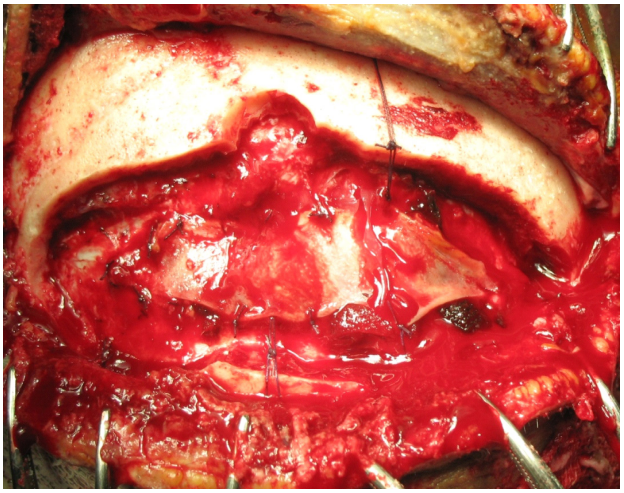

Figure 2. *Intra- operative photo showing technique of controlling sagittal sinus bleed using periosteal graft over gelfoam.*

examination, he was found to have 15 cm long lacerated wound over the parietal scalp with bruises over body. He was moving all limbs. Non contrast enhanced computed tomography scan of head showed depressed parietal bone fracture over midline without any underlying contusion or infarct. However there was brain edema.

Initially, thorough lavage and debridement of parietal scalp wound was performed. He was electively ventilated overnight. He improved gradually and had spontaneous eye opening with spontaneous limb movements. However on 10th day of his injury, he developed spastic paraparesis with gradual involvement of upper limb. His Glasgow coma score (GCS) also deteriorated and started flexing to pain. Fundoscopy revealed bilateral papilloedema. CT scan showed depressed parietal bone fracture with development of venous infarct over bilateral parietal lobe **(Figure 1)**. The condition of the patient did not allow us to undertake angiography with retrograde venous catherization. He was immediately explored. He was laid supine with neck flexed and head end elevated. Linear incision through the previous lacerated wound was made. Subperiosteal dissection was done to expose the normal bone around the depressed fracture. Burr holes were drilled over the normal bone across the sagittal sinus. Using powered drill, craniotomy was performed around the depressed segment. Using the free bone flap, depressed segment was raised in toto. Sinus bleed was controlled using the periosteal graft over gelfoam **(Figure 2)**. Bone flap was restored and fixed. Post operatively, his weakness improved and consciousness level also improved. He was discharged with Glasgow outcome score of 3.

Discussion

Classical teaching in depressed skull fracture over superior sagittal sinus (SSS) is to avoid elevation. The only reason given is for fear of fatal venous hemorrhage ([1, 2]). This widely held belief seems to be targeted for the residents to call their consultants for help.

Depressed skull fracture over the superior sagittal sinus causes venous sinus occlusion leading to elevated intracranial pressure (ICP) and cortical venous thrombosis ([3-7]) with evident encephalopathy. First reported by Caudill et al ([8]), total of 12 cases of elevated ICP due to depressed skull fracture over superior sagittal sinus had been reported till 2005 (5).

Intracranial hypertension develops within 48 hours to 1 month (5). For diagnosis, Magnetic Resonance Imaging (MRI) with venography and angiography are two useful studies. MR venography shows venous obstruction. Angiography with retrograde venous catherization shows high pressure gradient between venous flow upstream and downstream from the compressed zone. Successful conservative management with repeated lumbar punctures, oral acetazolamide and anticoagulation has been described for superior sagittal sinus thrombosis leading to raised ICP, particularly if pressure gradient is not too high. Spontaneous recanalisation of the SSS has been described ([9]). However conservative management in the face of sinus thrombosis is risky when there is surgically remediable cause. Studies have shown immediate recovery of intracranial hypertension ([10, 11]) as well as total restoration of superior sagittal sinus patency on elevation of depressed

fracture ([5]). The clinical improvement has been rapid and dramatic.

Three different scenarios can arise. If patient has compound depressed fracture over venous sinus, in presence of available surgical expertise, debridement and elevation of fracture can be attempted. It has been seen that hemorrhagic complication are more common if procedure is attempted in absence of raised intracranial hypertension ([1, 2]). Hence if not confident in tackling sinus bleed, one can safely debride and later attempt decompression, if intracranial hypertension develops. In closed fractures, decompression should be attempted only if features of raised ICP develop.Technique of elevating a depressed bone fracture has been standardized, as elucidated earlier.

However various methods of tackling sinus bleed have been described ([6]). Notable amongst them are digital pressure with gelfoam or free muscle flap or direct repair of defects on the sinus wall using simple suturing, suturing with periosteal patch, hitching of the dura to the bone adjacent to the sinus, ligature of the rostral part, clip-occlusion of the anterior segment, reconstruction with an autologuos vein, using artificial sinus prosthesis, balloon catheter and T-drainage. We have found head end elevation and digital pressure with gelfoam followed by periosteal grafting over the sinus a useful technique **(Figure 2)**. Bony spicules from fractured segment may cause serious sinus bleed. They should be waxed or gently nibbled piecemeal over the sinus.

Following decompression of venous sinus after elevation of fracture segment, it is essentially to follow patients for recovery of intracranial hypertension by looking for reversal of papilloedema and radiological evidence of patency of venous sinus on MR venography. In presence of partial stenosis, one should perform retrograde venous catheterization with pressure measurement. If pressure gradient is found to be high, stent placement on the sinus can be attempted ([12]).

Hence we recommend that if expertise to control sinus bleed is available, all patients with significant depressed fracture over superior sagittal sinus particularly over middle or posterior 1/3, should be elevated as conservative management would entail risk of sinus thrombosis and consequent fatal intracranial hypertension, venous infarct or encephalopathy. Early surgical decompression is all the more important if patient is symptomatic for sinus thrombosis or raised intracranial pressure with high pressure gradient on angiography with retrograde venous catherisation. However patients who are asymptomatic or with depressed fracture over anterior 1/3 superior sagittal sinus, can be conservatively managed.

Conclusions

The recent literature has started questioning this long held belief of conservative care in depressed skull fracture over sagittal sinus. If expertise of tackling the sinus bleed is available, one must elevate the depressed skull fracture in previously considered no man's land.

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