DECOMPRESSIVE CRANIECTOMY AND SYNDROME OF THE SINKING SKIN-FLAP

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Abstract

The syndrome of the sinking skin flap was introduced to explain the phenomenon of neurological deterioration after decompressive craniectomy. A 40 -year-old man was admitted with traumatic intracerebral hemorrhage. After failure of conservative treatment with intravenous mannitol infusion and hyperventilation to reduce intracranial pressure, decompressive craniectomy was done. Following this procedure patient gradually improved. Nine months later his neurological condition begun to deteriorate with depression of the skin flap. The patient recovered after cranioplasty. We present a patient who was successfully managed with cranioplasty for the syndrome of the sinking scalp flap with review of a pertinent literature.

Key Words: *Dcecompressive cranniectomy, Cranioplasty, Syndrome of sinking scalp flap, Traumatic Brain injury.*

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Introduction

Decompressive craniectomy procedures date back over 100 years. In 1905 Cushing [1] described this technique for treating intracranial pressure in a patient with a large intracranial tumor. This method was widely used after surgery of tumor, vascular lesion, trauma and cerebral abscess.[2] but in the current era decompressive craniectomy is widely used in neurosurgical practice for the treatment of intractable intracranial hypertension in patients with severe head injury, acute stroke, and severe brain edema after intracranial procedure[3]. However, this procedure paradoxically can cause neurological deterioration and marked depression of craniectomy site. The syndrome of the sinking skin flap was introduced to explain neurological deterioration after decompressive craniectomy.[2] This phenomenon may result from CSF hypovolemia, atmospheric pressure gradient that may be aggravated by CSF diversion and position change.[4] Therefore, it is important to understand pathophysiologic mechanism and treatment of this condition. We present a patient of traumatic brain injury who underwent bifrontal creniectomy for intractable intracranial pressure (ICP) and later neurological deteriorated despite sinking skin flap after initial improvement.

Case Report:

Rafiqul islam, 40 years of age, right handed nondiabetic, nonhypertensive worker had history of head injury due to fall from top of a running lorry on 5th August 2010 that occurred abroad. On arrival at the emergency department he was found to have Glasgow Coma Scale (GCS) -8 (E1M5V2) with bilaterally mildly dilated pupil without any focal neurological sign and stable cardiovascular status. He was taken to the ICU after CT scan of brain. On the CT scan there was bifrontal contusional hemorrhage with effaced lateral ventricle and enlarged bilateral temporal horns. As there was no improvement after conservative treatment, he underwent decompressive creniectomy (bifrontal) (Fig 2&3) on 9th August 2010. Postoperative neurological improvement was gradual and referred to district hospital on 20th March 2011 with Glasgow Coma Scale (GCS)- 12 (E4M5V3). After few weeks he begun to deteriorate and he was sent home as his company refused to bear his expenses.

On arrival at home his GCS was 8 (E2V2M4) on 16th April 2011 with sunken skin over frontal region (Fig.-4). On CT scan there was ventricular dilation with periventricular oedema without any corticle lesion (Fig.-1) .After improving his nutritional condition. We did a bifrontal cranioplasty on 26th April 2011(Fig.-5). His neurological condition improved postoperatively, started to communicate and walk with support and his catheter was removed. He was discharged on 12th postoperative day (Fig.-6).

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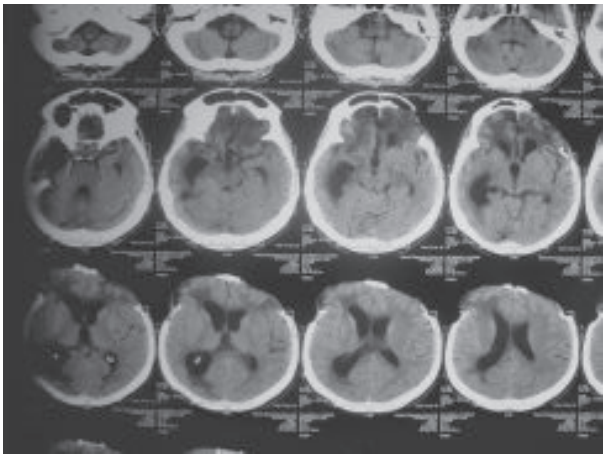

Fig.-1: *Bifrontal craniectomy with periventricular oedema in CT brain*

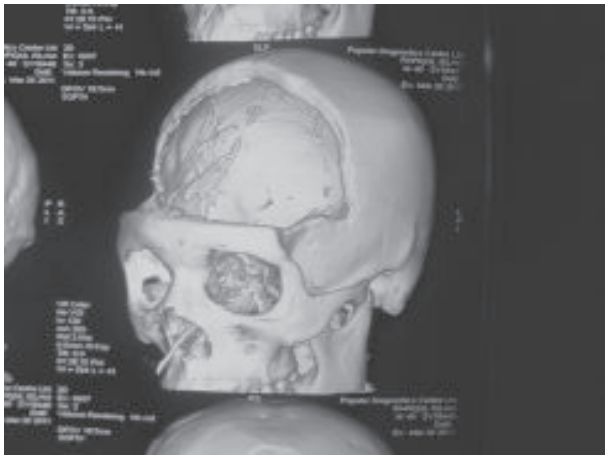

Fig.-2: *Bifrontal craniectomy with 3D reconstruction*

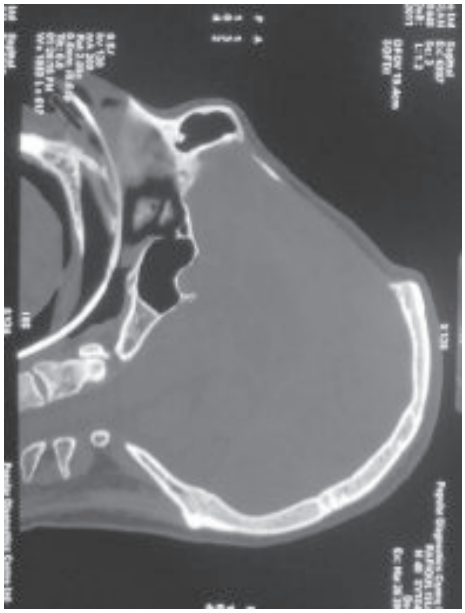

Fig.-3: *Frontal craniectomy in sagital Section of CT brain*

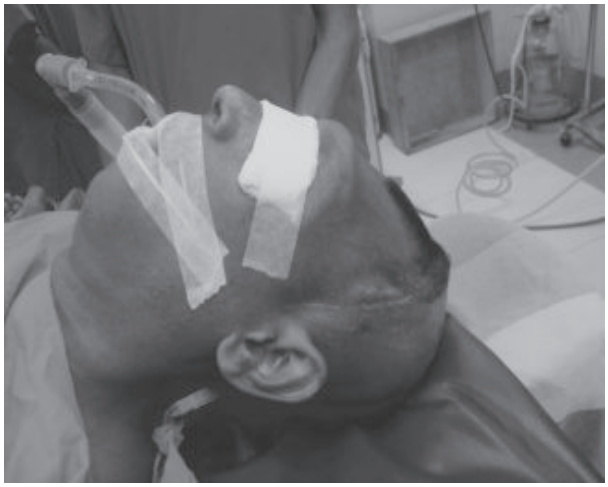

Fig.-4: *Patients peroprative view with sinking skin*

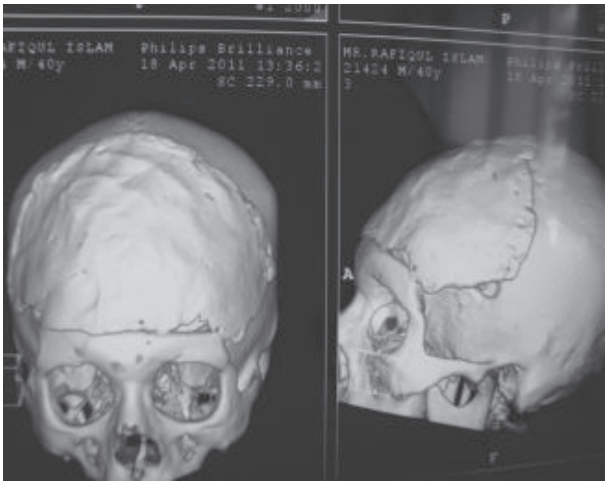

Fig.-5: *Bifrontal cranioplasty in CT brain with 3D reconstruction*

Fig.-6: *Patient on 8th Postoprative day*

Discussion:

The phenomenon of the sinking scalp flap syndrome with progressive neurologic deterioration has seldom been reported in the literature especially with regard to neurologic improvement following cranioplasty. The general symptoms sometimes seen after craniectomy were first defined by Grant and Norcross [5] in 1939, as the syndrome of the trephine. This syndrome includes dizziness, undue fatigability, vague discomfort at the site of the defect, a feeling of apprehension and insecurity, mental depression, and intolerance of vibration. Yamaura and Makino[2] first defined the "syndrome of the sinking skin flap" as objective neurological abnormalities that could be explained solely due to the concavity of the skin flap and the pressure of the atmosphere on the underlying brain tissue, which improved after cranioplasty. They found neurological improvement following cranioplasty in their three consecutive presentations.[2,6,7] Neurological improvement occurred in those with moderate neurological deficits and with skin flap of the sinking or flat type among their 4 types of skin flap (sinking, flat,full, bulging), and less possibility in those with minimal or severe neurological deficits including persistent vegetative state. Mean time elapse was 9.8 months in patients who responded with neurological improvement to the cranial repair like our presenting case. Fodstad et al.[8] believed that only symptoms reduced or relieved by cranioplasty should be included in the definition of "syndrome of the trephined." Stiver et al [9]found that decompressive hemicraniectomy was at significant risk for developing a delayed, reversible motor deficit starting at distal contralateral upper-extremity weakness. The mean time of onset was 4.9 months and the deficit progressed slowly in severity. They called it motor trephine syndrome, which showed as a reversible and treatable problem. First case described this phenomenon in relation to deformity of the skin flap was published by Uemura et al [10] in 1975 when a 48-year-old male, a college faculty member, underwent right craniotomy for massive subdural hematoma on Jan. 20, 1969. The bone flap had to be removed subsequently because of postoperative complications. Three months after surgery, he noticed weakness of the left upper extremity starting from fingers with concavity of the skin flap. On May 20, 1969, cranioplasty was performed using the preserved autogenous bone flap, followed by gradual improvement of the neurological deficits. He eventually returned to his previous occupation. In July, 1970, the skin flap began to sink again, accompanied by the same neurological deterioration. On Sept. 2, 1970 the atrophied and sunken bone flap was removed and a methyl methacrylate plate was molded. This procedure was followed by prompt neurological improvement on the 6th postoperative day.1 year later Tabaddor and LaMorgese [11] in 1976 presented another case of 41 years of age of acute subdural hematoma following closed head injury who underwent creniectomy and removal of clot. Later he was discharged and joined his work but deteriorated again and improved after cranioplasty. The first pathophysiologic explanation for this syndrome was suggested by Gardner et al. [12] in 1945, who claimed that, unlike the brain in the closed calvaria, in a trephined skull the brain pulsates with every alteration of arterial or venous pressure. According to this hypothesis, the in-and out pathologic movement of the brain is responsible for the symptoms, and therefore an immobilizing cranioplasty should relieve these symptoms. Gardner again quotes Falconer and Russell [13] who who performed a decompressive craniectomy in 270 animals, suggested that the migration of the ventricle towards the side of a traumatic skull defect is not entirely due to loss of brain tissue and scar contraction from the trauma, but is partly due to progressive gliosis and atrophy of the pulsating portion of the brain beneath the skull defect. Gardner, therefore, claimed that if the cranioplasty is performed immediately after the craniectomy, the ST will not occur. In 1968, Langfitt et al. [14] proved that in a patient with a large cranial defect the lumbar cerebrospinal fluid pressure in the upright position is greater than in a patient with closed calvaria. He also demonstrated that after cranioplasty, the lumbar cerebrospinal fluid pressure was normalized. The suggested theory to explain these findings was that the atmospheric pressure is transmitted to the intracranial cavity, causing inward rotation of the scalp over the cranial defect. This pressure acting over the cerebral cortex may cause neurologic deficit. Guido et al. [15] described in 1976 two patients with a large cranial defect and a lumbar cerebrospinal fluid leak after a spinal drainage. These patients deteriorated while in the upright position. Both patients after having their leak sealed by an epidural blood patch, improved neurologically, while the concavity of the skin flap filled up. In 1985 Stula et al. [16] studied patients with neurologic deficits after craniectomy. All these patients showed

improvement of their neurologic deficit following cranioplasty. To explain this, Stula suggested that the atmospheric pressure acting on the unprotected brain, produces brain compression and cranioplasty normalized this situation. In 1984 Fodstad [8] performed extensive cerebrospinal fluid hydrodynamic studies on 40 patients, 22 cerebrospinal fluid infusion test was performed before and after cranioplasty. He claimed that the skull defect creates a siphon effect on the cerebrospinal fluid dynamics that creates a midline shift and affects the local cerebral blood flow. The mechanism of this effect is not yet clear but appears to be connected to distortion of the dura and underlying cortex, venous sinuses, and subarachnoid space by scar tissue and atmospheric pressure. Richaud et al. [17] explained in 1985 the neurologic improvement after cranioplasty in the trephined patients by demonstrating with xenon inhalation studies that after operation there was a 15%-30% improvement in cerebral blood flow to the cortex under the cranioplasty site. They claimed that the subarachnoid space became obliterated by the pressure of the atmosphere and the scar. This causes the cerebral venous return to become susceptible to local pressure changes, and, in addition, there was a direct compression effect on the cortex by the inward displacement of the skin flap. Segal et al. [18] supported this theory in 1993 and summarized the mechanisms to include: 1) impeding venous return and thereby altering local cerebral hemodynamics; 2) direct pressure effect from the scars, dura, skin, and atmosphere on the cortex; and 3) local changes in cerebrospinal fluid dynamics. Cerebral hemodynamics is compromised in the presence of a cranial defect by the following mechanisms. The subarachanoid space is obliterated over the cortex in that area by scarring of the pseudodura or reapproximated dura, thus compromising any vessel on the cortex that extends into the subarachanoid space. The venous return is especially succeptible to this damage. In addition, a direct compressive effect on the cortex evident by the inward displacement of the pseudodura and skin flap also impedes venous return. There appears to be an increase in the local intracranial pressure (ICP) in the area adjacent to the cranial defect. Stula et al. [16] measured the ICP for 4 days before and after cranioplasty with epidural monitors. Increased ICP was found in 6 of 12 patients monitored preoperatively. These six patients also all had neurological deficits. After cranioplasty, all six patients had a return of their ICPs to normal. This was accompanied by a progressive improvement of clinical neurological deficits. These findings suggest that the scarplate formed between the cortex, dura, and skin exerts direct pressure on the intracranial contents, causing an increase in local ICP and contributing to the neurological deficits. Cranial defects that compromise the sagittal sinus caused by scarring and pressure not only compromise venous return but also the absorption of cerebrospinal fluid contributing to increase ICP. Our patient deteriorated approximately nine month after the incident and he had moderate neurological deficiency with sinking type skin flap that correspond with Yamaura and Makino.[2] The preoperative findings correlate with Segal et al. [18] findings which is responsible for neurological deterioration . Although the mechanism remains controversial, it is clear that cranioplasty may improve neurological function in certain patients. Large concave skull defects may offer the best potential for improvement. Although currently accepted indications for cranioplasty are for cosmesis and for protection of intracranial structures, the potential improvement of neurological function should be added as an acceptable indication for cranioplasty.

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