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SINKING SKIN FLAP SYNDROME: POST CRANIECTOMY COMPLICATION: A CASE REPORT

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ABSTRACT
Sinking skin flap syndrome (SSFS) is defined as serious disabling neurologic deficit and impairment of neurological status, with concave deformity and relaxation of the craniectomy skin flap which tends to develop several weeks to months after large craniectomy. The pathophysiology includes cerebrospinal fluid (CSF) hypovolemia and development of an atmospheric pressure gradient, that may be aggravated by CSF diversion, dehydration, and patients posture. The goal of treatment in patient with the syndrome of the sinking skin flap is restoration of the pressure exerted by depression of craniectomy site.

Abbreviations
SSFS – Sunken skin flap syndrome
NCCT – Non contrast computed tomography
GCS – Glasgow coma scale

INTRODUCTION

Sinking skin flap syndrome (SSFS) is defined as serious disabling neurologic deficit and impairment of neurological status, with concave deformity and relaxation of the craniectomy skin flap which tends to develop several weeks to months after large craniectomy [1].The goal of treatment in patient with the sinking skin flap syndrome is restoration of the pressure exerted by depression of craniectomy site [2]. Here we report a case of Sinking skin flap syndrome (SSFS) who presented with neurological deterioration five weeks after craniectomy procedure was done, following a road traffic accident.

Case Report

A 24 year old male, who suffered a road traffic accident in the year 2014, had closed head injury with loss of consciousness. Urgent NCCT head done at a peripheral hospital revealed large right frontotemporal subdural heamatoma & haemorrhagic contusions involving the right frontal, temporal & parietal lobes causing mass effect with a midline shift of 15mm towards Left side.Craniectomy was performed in view of the raised intracranial tension. The patient's neurological status improved in next 48 hrs post surgery to GCS - 12/15, with paraparesis left lower limb (power 3/5) & patient was shifted to the Command hospital Bangalore for further management. The NCCT scan done on arrival revealed right frontal & temporal post-craniectomy status with right frontal lobe herniating out of the craniectomy defect. There were hypodense areas seen within the brain parenchyma in right frontal, parietal & temporal regions [Fig.1]. The patient was managed with supportive care & remained clinically stable for next two weeks during his hospitalization, with improvement noted in his neurological status with GCS of 14/15 & Left lower limb power of Grade 4/5.

However, after five weeks patient suddenly started deteriorating neurologically, had two seizure episode and he complained of headache. On examination a crater like depression was noted at the site of craniectomy on Right side of the head. NCCT head was done, which revealed that the right frontal parietal & temporal lobes

have shrunk in size with underlying gliotic changes in the remaining brain parenchyma of the right cerebral hemisphere [Fig.2]. The lateral and third ventricles showed ex-vacuo dilatation, with no significant mass effect herniation or midline shift. No fresh intracranial haemorrhage was identified. On the basis of clinical background and imaging findings, the post-craniectomy complication of 'sunken skull syndrome / Trephine syndrome' was diagnosed.

Cranioplasty was done using craniectomy flap, which was replaced at the craniectomy defect, following which the patient gradually improved, with no fresh episode of convulsions or headache. The NCCT head revealed that the volume loss of Right fronto-temporo-parietal region noted in the pre-cranioplasty scan had reverted and the underlying brain parenchyma has expanded, with ex-vacuo dilatation of the ventricles [Fig.3.].

Figure 1. Non Contrast Computed Tomography showing Post right fronto-temporal craniectomy status with right frontal lobe herniating out of the craniectomy defect, hemorrhagic contusions right frontal , parietal & temporal regions.

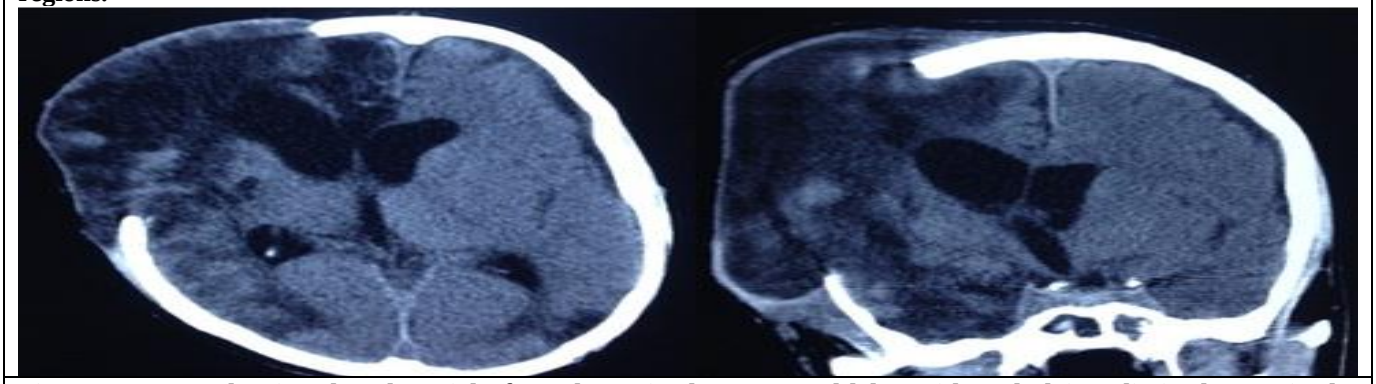


Figure 2. NCCT showing shrunken right frontal, parietal & temporal lobes with underlying gliotic changes in the remaining brain parenchyma of the right cerebral hemisphere. The lateral and third ventricles showed ex-vacuo dilatation with no significant mass effect herniation or midline shift.

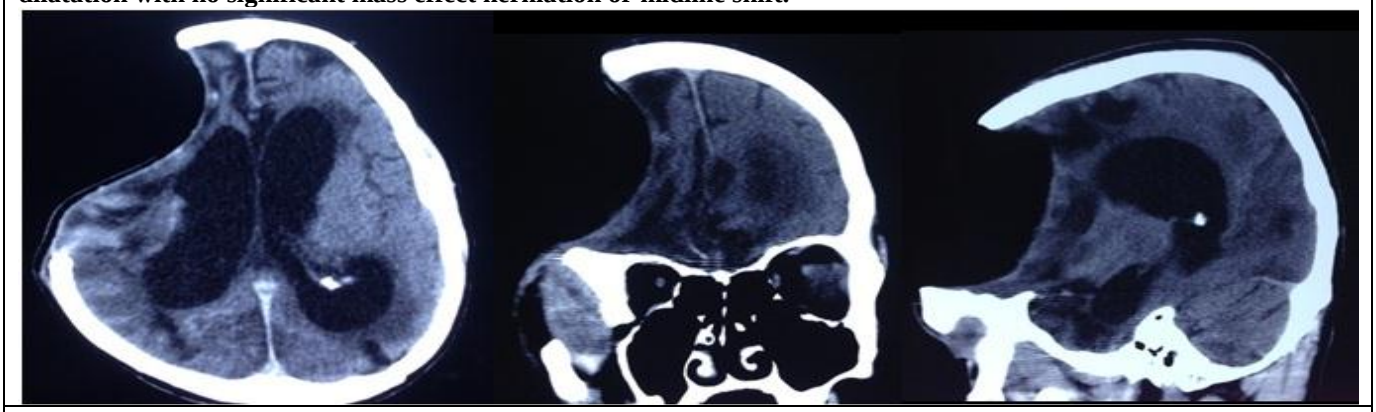
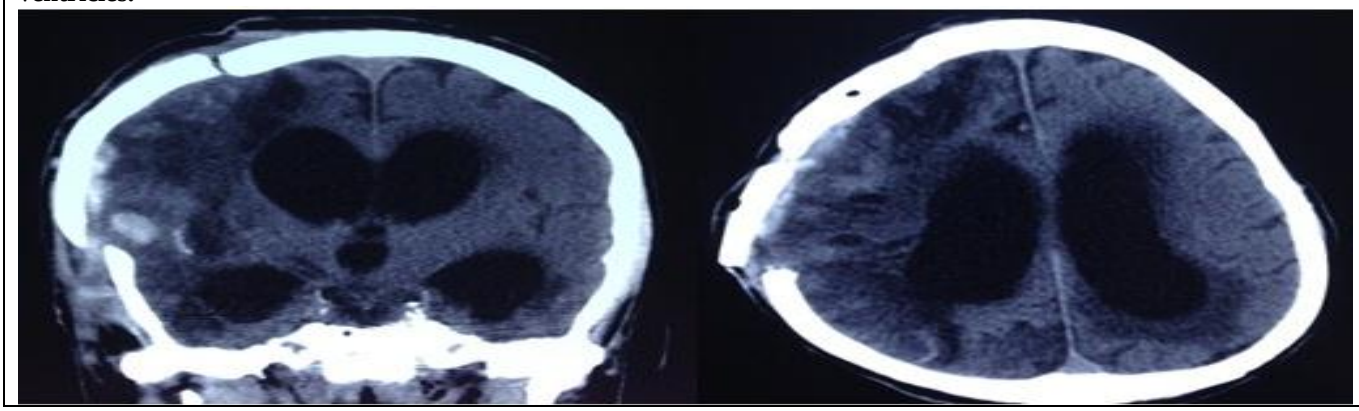


Figure 3. Post cranioplasty NCCT head showing volume loss of right fronto-temporo-parietal region noted in the pre-cranioplasty scan had reverted and the underlying brain parenchyma has expanded with ex-vacuo dilatation of the ventricles.



DISCUSSION AND CONCLUSION

The terms syndrome of the sunken skin flap [1] / sinking skin flap syndrome SSFS [1] /trephine syndrome / syndrome of the trephined [4] / motor trephine syndrome [3], have been used interchangeably in past to explain a complication of craniectomy,which tends to develop several weeks to months after large craniectomy. The features consist of serious disabling neurologic deficit and impairment of neurological status, with concave deformity and relaxation of the craniectomy skin flap.

The term syndrome of the sunken skin flap was coined by Yamura and Makino in 1977 to describe neurological complications due to craniectomy defect [1] characterized by neurological symptoms spectrum including headache, epileptic seizures, tinnitus, vertigo, dysesthesias, dysphagia, loss of concentration, loss of memory, apraxia and paresis, which improve after cranioplasty [3]. There are few reports of SSFS associated with delayed motor deficits, designated as "motor trephine syndrome"[3].

The incidence of syndrome of the trephined was reported to be 10 of 38 (26%) patients by Stiver while following up those who underwent cranioplasty in a series of 170 patients who had undergone decompressive craniotomy for traumatic brain injury [4]. Huang et al reported that 54 of 108 patients developed complications related to surgical decompression [5].

Various factors implicated in the pathophysiology of sunken skin flap include stretching of the dura and underlying cortex due to the atmospheric pressure, cicatrical changes occurring between the cortex, dura and the skin exerting pressure on the skull contents, and impairment of the venous return due to the atmospheric pressure acting on the region of skull defect [6,7]. Several authors proposed that a negative gradient between atmospheric and intracranial pressure, which is aggravated by changes in the CSF compartment following CSF hypovolemia, to be the mechanism of neurological deterioration after craniectomy [2]. The SSF may progress to "paradoxical herniation" as a consequence of the atmospheric pressure exceeding intracranial pressure and may eventually lead to coma and death[8]. Our patient also presented both the phases of herniation of the brain parenchyma underlying the craniectomy defect. Prolonged dehydration and up-right position may also precipitate this phenomenon [9,10].

As intracranial volume is fixed, any increase in cranial contents(swelling, edema, haemorrhage, mass lesion) in patients with head injury, acute stroke and severe brain edema after intracranial procedures will produce an increase in intracranial pressure [11], warranting decompressive craniectomy as an effective therapeutic neurosurgical option for relief of intractable intracranial hypertension [12,13]. Decompressive craniectomy allows for intracranial volume expansion, avoiding intracranial hypertension [11]. Winkler et al. demonstrated improved blood flow and cerebrovascular reserve capacity after cranioplasty in 12 cases using transcranial Doppler sonography and 18-FDG positron emission tomography [14]. George et al showed in a series of angiographies that there was a correlation between the restoration of the midline shift and clinical improvement following cranioplasty [15].

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CONFLICT OF INTEREST: NIL

REFERENCES

1. Yamaura A, Makino H. (1977). Neurological deficits in the presence of the sinking skin flap following decompressive craniectomy. (Tokyo). *Neurol Med Chir*, 17, 43-53.
2. Abdul Rashid Bhat, Altaf R Kirmani, FurqanNizami, Ashish Kumar, Mohammed AfzalWani. (2011). Sunken brain and scalp flap syndrome following decompressive extra-craniectomy. *Indian Journal of Neurotrauma (IJNT)*, 8(2), 105-8.
3. Kemmling et al. (2010). Case report of MR perfusion imaging in Sinking Skin Flap Syndrome: growing evidence for hemodynamic impairment. *Neurology*, 10, 80. (http//:www.biomedcentral.com/1471-2377/10/80).
4. Stiver SI. (2009). Complications of decompressive craniectomy for traumatic brain injury. *Neurosurg Focus*, 26, E7
5. Huang AP, Tu YK, Tsai YH, Chen YS, Hong WC, Yang CC, Kuo LT, Su IC, Huang SH, Huang SJ. (2008). Decompressive craniectomy as the primary surgical intervention for haemorrhagic contusion. *J Neurotrauma*, 25(11), 1347-1354
6. Segal DH, Oppenheim JS, Murovic JA. (1994). Neurological recovery after cranioplasty. *Neurosurgery*, 34, 729-731
7. Fostad H, Love JA, Friden H, Lilliequist B. (1984). Effect of cranioplasty on cerebrospinal fluid hydrodynamics in patients with the syndrome of the trephined. *Acta Neurochir*, 70, 21-30
8. Sarov et al. (2009). Sinking skin flap syndrome and paradoxical herniation after hemicraniectomy for Malignant Hemispheric Infarction. *Stroke*, 41, 560-562 (DOI: 10.1161/STROKEAHA.109.568543)
9. Oyelese AA, Steinberg GK, Huhn SL, Wijman CA. (2005). Paradoxical herniation secondary to lumbar puncture after decompressive craniectomy for a large space occupying hemisphere stroke: case report. *Neurosurgery*, 57, 594
10. Schwab S, Erbuguth F, Aschoff A, Orberk E, Spranger M, Hacke W. (1998). Paradoxical herniation after decompressive trephining, 69, 896-900.
11. Margules, Andrew and Jallo, Jack. (2010). Complications of Decompressive Craniectomy, *JHN Journal*, 5(1), Article 4. Available at: http://jdc.jefferson.edu/jhnj/vol5/iss1/4

12. Liao CC, Kao MC. (2002). Cranioplasty for patients with depressed skull bone defect after cerebrospinal fluid shunting. *J Clin Neurosci*, 9, 553-555
13. Schiffer J, Gur R, Pollak L. (1997). Symptomatic patients after craniectomy. *Surg Neurol*, 47, 231-237
14. Winkler PA, Stummer W, Linke R, Krishnan KG, Tatsch K. (2000). Influence of cranioplasty on postural blood flow regulation, cerebrovascular reserve capacity, and cerebral glucose metabolism. *J Neurosurg*, 93, 53-61
15. George AE, Morantz RA, Abad RM, Rovit RL, Chase N. (1974). Neuroradiology of the post hemicraniectomy patient with special emphasis on the radiology of unilateral atrophy. *Radiology*, 111, 627-631.