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Literature Review

Neurological susceptibility to a skull defect

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Abstract

Background: There continues to be considerable interest in the use of decompressive craniectomy in the management of neurological emergencies. The procedure is technically straightforward; however, it is becoming increasingly apparent that it is associated with significant complications. One complication that has received relatively little attention is the neurological dysfunction that can occur due to the absence of the bone flap and the subsequent distortion of the brain under the scalp as cerebral swelling subsides. The aim of this narrative review was to examine the literature available regarding the clinical features described, outline the proposed pathophysiology for these clinical manifestations and highlight the implications that this may have for rehabilitation of patients with a large skull defect.

Methods: A literature search was performed in the MEDLINE database (1966 to June 2012). The following keywords were used: Hemicraniectomy, decompressive craniectomy, complications, syndrome of the trephined, syndrome of the sinking scalp flap, motor trephined syndrome. The bibliographies of retrieved reports were searched for additional references.

Results: Various terms have been used to describe the different neurological signs and symptoms with which patients with a skull defect can present. These include; syndrome of the trephined, posttraumatic syndrome, syndrome of the sinking scalp flap, and motor trephined syndrome. There is, however, considerable overlap between the conditions described and a patient's individual clinical presentation.

Conclusion: It is becoming increasingly apparent that certain patients are particularly susceptible to the presence of a large skull defect. The term "Neurological Susceptibility to a Skull Defect" (NSSD) is therefore suggested as a blanket term to describe any neurological change attributable to the absence of cranial coverage.

Key Words: Complications, decompressive craniectomy, syndrome of the trephined

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INTRODUCTION

Over the past two decades, there has been a resurgence of interest in the use of decompressive craniectomy in the management of neurological emergencies.[10,26] The surgery itself involves temporarily removing a large segment of the skull in order to provide extra space into which the injured or edematous brain can expand. The procedure can be

performed either unilaterally or bilaterally and its use been has been described most commonly in the context of traumatic brain injury[1,24,44] or ischemic stroke[12,50] and more recently following subarachnoid hemorrhage[25,52] severe intracranial infection[4,6] dural sinus thrombosis[16,39] and inflammatory conditions.[2,13]

THE CURRENT ROLE OF DECOMPRESSIVE CRANIECTOMY FOR NEUROLOGICAL EMERGENCIES

Numerous studies have demonstrated that the procedure can reduce mortality and this was most clearly demonstrated by the pooled analysis of the three European stroke trials that compared decompressive hemicraniectomy with standard medical management in patients who clinically deteriorated following cerebral swelling secondary to ischemic stroke. The results of this analysis confirmed a dramatic reduction in mortality in those patients treated surgically and this provides unequivocal support for the use of the procedure as a lifesaving intervention.[58] However, evidence that outcome is actually improved is less forthcoming. The DECRA (Decompressive Craniectomy in Patients with Severe Traumatic Brain Injury) compared early decompressive craniectomy for diffuse traumatic brain injury with standard medical therapy and found that patients in the surgical arm had worse outcomes than those treated medically.[11]

Notwithstanding a number of criticisms,[57] the trial has provided unequivocal evidence regarding two key points. First, lowering the intracranial pressure (ICP) by surgical intervention is not necessarily translated into an improvement in outcome. Second, at the particular ICP threshold at which these patients were enrolled, there was insufficient ongoing secondary brain injury and therefore any benefit obtained by lowering the ICP was offset by surgical morbidity.[27] Indeed it is becoming increasingly apparent that although the procedure is technically straightforward, it is associated with a number of complications that can have significant impact on long-term outcome.[28,62]

One complication that has received relatively little attention is the neurological dysfunction that can occur due to the absence of the bone flap and the subsequent distortion of the brain under the scalp as cerebral swelling subsides.

NEUROLOGICAL DYSFUNCTION DUE TO A SKULL DEFECT

In 1939, Grant and Norcross coined the term "syndrome of the trephine" to describe the symptoms of headache, vertigo, tinnitus, fatigue, insomnia, memory disturbance, seizures, mood swings, and behavioral disturbance that was observed in some individuals with a large skull defect.[22] Subsequently, a number of terms have been introduced that revolve around a common theme. Grantham coined the term "the post traumatic syndrome" to describe similar subjective symptoms to that of "syndrome of the trephined."[23] In the 1970s, Yamaura and Makino[61] used the term "syndrome of the sinking scalp flap" to describe the objective focal neurological deficits that can occur in patients with a hemicraniectomy defect and "Motor trephined syndrome" is an alternative term to describe objective motor deficits.[49,51,54] The classical descriptions of these phenomenon are of an initial period of improvement after the decompressive surgery followed by a period of neurological deterioration and the diagnosis is confirmed when the symptoms resolve or improve following replacement of the bone flap.[17]

Until recently, these conditions have been described as being either rare or uncommon; however, it is becoming evident that certain patients are particularly susceptible to neurological signs and symptoms relating to the presence of a large skull defect. Clinical presentation can range from the classical description of a reversal of neurological deficits,[9,14,19,21,36,43,56] to a more subtle but quantifiable improvement in neurocognitive function[3,63] or merely just a failure to clinically improve.[29] Indeed, given the variation in clinical signs exhibited, it is often difficult to provide a specific diagnosis. For example, a patient may develop postural headaches and increasing lethargy and therefore are deemed to have "Syndrome of the trephined," but they may also develop a focal deficits and are therefore given a diagnosis of "syndrome of the sinking scalp flap."

In view of this variation it may be simpler to use a blanket term that applies to all clinical manifestations attributable to the absence of a bone flap such as "Neurological Susceptibility to a Skull Defect" (NSSD)

NEUROLOGICAL SUSCEPTIBILITY TO A SKULL DEFECT-PATHOPHYSIOLOGY

The underlying pathophysiology responsible for the various neurological manifestations has yet to be established, however, a number of theories have been proposed including direct effects of atmospheric air on the brain,[23,54,59] alterations in cerebrospinal fluid (CSF) hydrodynamics[17] and changes in cerebral blood flow and metabolism.[15,55,59,60,63]

Direct effects of atmospheric air on the brain

One of the most easily observed consequences of a decompressive craniectomy is the development of a sunken concave deformity that occurs once the original brain swelling subsides. Because the "closed box" or skull has become open the principles of the Monroe–Kellie doctrine no longer apply and the brain cannot "float" in supportive CSF.[17] Exposure of the brain to atmospheric pressure will cause distortion not only of the cerebral cortex but also other intracranial structures leading to posture-related signs and symptoms such as headache,

altered sensorium, and cranial nerve palsies.[8,36,42,48] This distortion may also contribute to some of the CSF hydrodynamic and cerebral blood flow disturbances.

Disturbance of CSF hydrodynamics following decompressive craniectomy

In normal circumstance when there is complete cranial coverage, the ICP will often be negative in the upright position. However, in the presence of a large skull defect, the ICP will tend to equalize with that of the atmospheric pressure. In these circumstances, the ICP may actually be higher than normal when patients are in the upright position. These changes were demonstrated in studies that used CSF infusion tests before and after cranioplasty and demonstrated that hydrodynamic abnormalities present before cranioplasty were reversed after the bone flap was replaced.[17] In certain instances, this was accompanied by a clinical improvement, however, this was not always the case.

Another manifestation of the hydrodynamic disturbances that can occur is the development of subdural effusions and hydrocephalus. This is most commonly seen in the context of trauma, however, it has been reported in other pathological conditions.[37,38] The term "Subdural hygroma" was coined by Dandy in 1945 to describe this phenomenon and the pathophysiology is thought to be related to traumatic shearing at the amorphous interface between the dura and subarachnoid space.[11] This may be compounded by a mechanical or inflammatory blockage of the subarachnoid space such that the resistance to CSF outflow is increased.[30] In certain cases, resolution of the subdural collections is followed by the development of hydrocephalus and it has been suggested that the two clinical entities may be related.[30]

Disturbance in cerebral blood flow and metabolism

A number of studies have used either dynamic computed tomography (CT) scanning,[47,54,55] Xenon CT,[3,34,46] or transcranial Doppler ultrasonography[60] to demonstrate the alterations in blood flow that can occur following a decompressive craniectomy and the subsequent improvement in blood flow that can occur following cranioplasty.[59,60]

The pathophysiology underlying this vascular response is unknown. It may merely be a reflection of the transmission of atmospheric pressure on to the cerebral vasculature or impairment of venous return as a result of direct cerebral compression by the inwardly distorted scalp.[17,46,51,61] However, while this may explain the local effects on blood flow, it does not in itself account for an improvement in cerebral blood flow remote from the site of surgery such as in the thalamus.[34,63] Other more subtle factors that may contribute include normalization of CSF compliance, cerebrovascular resistance, and autoregulatory function.[55]

These changes in cerebral blood flow may also relate to changes observed in cerebral metabolism following cranioplasty. Fludeoxyglucose (18F) (^{18}FDG) Positron emission tomography (PET) scanning has demonstrated depressed cerebral metabolism in the underlying hemisphere in patients who have had a unilateral hemicraniectomy.[60] Following cranioplasty, significant increase in uptake of ^{18}FDG in the underlying hemispheres indicated that cerebral metabolism was improved following restoration of normal cranial anatomy. In addition, there was a smaller but still significant increase in global uptake indicating that overall metabolism in the brain is improved and in some patients, this correlated with an observed neurological recovery.[59,60] It has been suggested that some of these observations may relate to recovery of cerebral autoregulation and although the correlation with clinical recovery would support this hypothesis, it remains to be clearly established.

Overall the effect that the skull defect has on neurological function may not be due to a single pathophysiological mechanism rather it may in fact be multifactorial. Indeed support for this hypothesis would come from the wide variety of clinical manifestations reported.

NEUROLOGICAL SUSCEPTIBILITY TO A SKULL DEFECT-CLINICAL FINDINGS

The number of patients who exhibit some degree of neurological dysfunction due to the absence of cranial coverage remains unknown. Most publications have been a combination of case reports and retrospective cohort studies and although they detail some impressive neurological recoveries, there is rarely a baseline denominator that records the number of patient in which the cranioplasty has no clinical impact. Most reports have described the various neurological manifestations as rare or uncommon, however, it is becoming increasingly apparent that some form of neurological improvement is more common than previously appreciated.

Fodstad[17] assessed 40 patients, of whom 14 (35%) were adjudged to be "true Syndrome of the Trephined." The case mix was, however, very heterogeneous and included a number of patients with focal deficits such as hemiparesis, which resolved following cranioplasty. The diagnosis in these patients would fit more with syndrome of the sinking skull flap. Yamaura[61] assessed 33 patients who were undergoing a cranioplasty and found that among the 29 patients whose neurological status was "abnormal", 9 (30%) patients exhibited a neurological improvement and this most commonly occurred in those patients in whom the scalp defect was more sunken prior to the cranioplasty being performed. The authors noted that although the 'nature of the neurological deficits,' which showed improvement, varied considerably, "syndrome of the sinking scalp flap" was distinct from "syndrome of the

trephined" or "the post traumatic syndrome" because the improvement in neurological function could be confirmed by objective examination rather than an improvement in subjective complaints. Stiver[54] retrospectively assessed 55 patients had had a decompressive hemicraniectomy and found that 10 patients (26%) developed a delayed monoparesis (motor trephine syndrome), which was reversed following cranioplasty.

More recently, a prospective cohort study found a slightly lower incidence of clinical recovery, however, there was a measureable improvement in some aspect of neurological function in 4 (16%) out of 25 patients who were assessed a few days before and after cranioplasty.[31] (This included one patient who exhibited a classical presentation of "syndrome of the trephined.").

Overall it would appear unequivocal that some patients are particularly susceptible to having a large skull defect and further studies will be required in order to determine not only the true incidence but also what factors predispose patients to this susceptibility. These issues may be important when considering the impact that this condition can have on rehabilitation and also on the timing of the cranioplasty procedure.

NEUROLOGICAL SUSCEPTIBILITY TO A SKULL DEFECT-IMPLICATIONS FOR REHABILITATION

Rehabilitation facilities are a valuable resource that have been shown to improve neurological outcome by improving motor and cognitive function and by re-engaging individuals into meaningful activities of daily living and enhancing social integration.[20,41,53] These resources are, however, relatively scarce and all efforts must be made to ensure that they are deployed appropriately. Given the impact that a large skull defect can have on neurological recovery, it has been suggested that intensive neurocognitive rehabilitation should not be undertaken until a cranioplasty has been performed.[35] However, adopting this position would mean that those patients not affected by the skull defect would miss out on the potential benefit of early rehabilitation. A more realistic approach would be to highlight the need to recognize the condition in susceptible individuals so that rehabilitation may perhaps be delayed until an appropriate neurosurgical referral has been made and the cranioplasty performed. This leads on to the issue of cranioplasty timing.

NEUROLOGICAL SUSCEPTIBILITY TO A SKULL DEFECT-IMPLICATIONS FOR CRANIOPLASTY

The conventional role of cranioplasty has been to reconstruct the cranial vault in order to provide protection[32] and to restore cosmesis, however, as previously stated it is becoming clear that in some patients, it may have a therapeutic role.[5]

The optimal timing of cranioplasty has not been clearly established, however, for many years it was suggested that the procedure should be delayed in order to reduce the risk of infection.[18,45] More recently, this practice has been called into question with a number of studies suggesting that early cranioplasty can be safely performed.[7,33,40]

In view of these findings it would seem logical to replace the bone flap as soon as clinically possible not only to provide protection and restore cosmesis but also to provide therapeutic benefit to some patients. This would also avoid the need for prolonged use of protective head-gear, which is often uncomfortable and not particularly dignified.

CONCLUSIONS

Patients who have had a decompressive craniectomy face a particularly challenging recovery and all efforts should be made to maximize the potential for neurological recovery. It would appear that certain individuals are particularly susceptible to having a skull defect and although the precise pathophysiology remains to be established it would appear to be multifactorial. The term NSSD is a reflection of the wide variety of clinical manifestations with which this susceptibility can present.

Recognition of this susceptibility is important in order to expedite earlier treatment in some cases and prevent scarce rehabilitation resources being unnecessarily apportioned to patients who cannot derive maximal benefit.

A final consideration is perhaps not why patients improve following restoration of cranial coverage, but why most patients seem to tolerate considerable sinking of the scalp with obvious distortion of the cortical surface. This would be an interesting focus of future research.

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