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Cranioplasty Flap Lifting Caused by Intracranial Hypertension Literature Review

Yakhya CISSE^{1*}, Jean Michel NZISABIRA¹, Abdoulaye DIOP², Ansoumane DONZO¹, Louncény Fatoumata BARRY¹, Rokhaya DIAJHETE³, Nantenin DOUMBIA¹, Papa Ndiouga LO¹, Aissatou KEBE¹, Fatou SENE¹, Alioune Badara THIAM¹, Momar Codé BA¹ and Seydou Boubakar BADIANE¹

¹Neurosurgery Department - Fann University Hospital Center - Dakar, Senegal

²Neurosurgery Unit, Ziguinchor Regional Hospital - Ziguinchor, Sénégal

³Geriatric Department - Fann University Hospital Center - Dakar, Senegal

ABSTRACT

Cranioplasty is a neurosurgical technique that replaces a bone defect in the skull with hard replacement tissue. It is indicated in particular after a decompressive craniectomy performed in severe head trauma in order to control intracranial hypertension refractory to medical treatment. Cranioplasty is sometimes associated with a significant number of complications, including hydrocephalus. In this article, we report the case of a cranioplasty flap lifting on intracranial hypertension following postoperative hydrocephalus and discuss the clinical relevance with a review of the literature.

INTRODUCTION

Cranioplasty is a neurosurgical procedure that restores the shape and function of the skull with replacement hard tissue. This technique, performed after decompressive craniectomy in severe head trauma, often improves the clinical condition of patients [1]. However, it is sometimes associated with a large number of complications including hydrocephalus [2-4]. In this article, we report the case of cranioplasty flap lifting caused by intracranial hypertension in an adult having undergone a decompressive craniectomy for an acute post-traumatic subdural hematoma.

CASE REPORT

The patient is 57 years old, male, with no specific history admitted to the emergency room for severe head trauma by road accident. At the clinical examination he presented altered consciousness, Glasgow Coma Scale 7 (E1 V1 M5) with right hemibody deficit. Brain CT revealed an acute left hemispherical subdural hematoma with a significant shift. A left hemispherical decompressive craniectomy was performed urgently to control intracranial hypertension. There was significant clinical improvement and the patient discharged from hospitalization after a few days. A left hemispherical cranioplasty was performed after 3 months using surgical cement. Two months after cranioplasty, the patient is readmitted to hospital for convulsive seizures of the right half of the body. Glasgow at 8 (E4V1M3), anisocoria with photo reactive left mydriasis and cranioplasty uplift (Figure 1). Brain CT showed

*Corresponding author

Yakhya CISSE, CHU de Fann -Avenue Cheikh Anta DIOP- BP: 5035 - Dakar/Senegal

Tel: +221-773-776-698

E-mail: yakhycisse@hotmail.com

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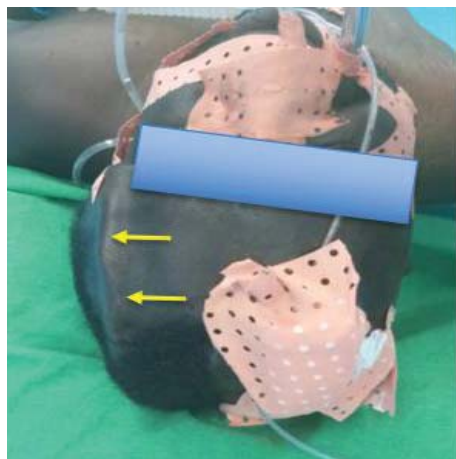


Figure 1 Macroscopic image of Cranioplasty lift (yellow arrow).

hydrocephalus, loss of fixation of the cranioplasty flap with expansion of the brain parenchyma out of the cranial box (Figure 2). An External Ventricular Drainage (EVD) was placed urgently with clinical improvement with regaining of consciousness and arrest of seizures. A Ventriculoperitoneal Shunt (VPS) was placed afterwards (Figure 3) and the patient discharged from hospitalization after a few days (Figure 4). We didn't fix the cranioplasty after the ventriculoperitoneal shunt. Nevertheless we plan to replace the cranioplasty flap by another and fix it.

DISCUSSION

Hydrocephalus is defined as an active dilatation of the ventricles due to a disorder of the hemodynamics of CSF [5]. Overproduction or defective absorption may be the cause. In our case the patient developed a postoperative hydrocephalus (Following a cranioplasty after decompressive craniectomy). In fact, the resistance to the flow of the CSF is reduced and the cerebral compliance increased after a decompressive craniectomy. When in addition there is hydrocephalus



Figure 2 Cerebral TDM without contrast in coronal reconstruction showing uplift with Hydrocephalus.



Figure 3 Post VPS image showing a lowering of the shutter.

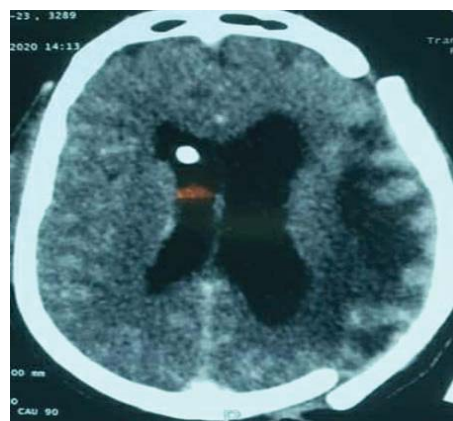


Figure 4 CT scan without contrast showing lowering of the Cranioplasty flap after VPS.

preoperatively and the craniectomy is important, it can facilitate the irreversibility of the ventricular dilation over weeks or months [6]. The decompressive craniectomy, in our patient, was performed urgently. It made it possible to control intracranial hypertension with clinical improvement. Indeed, decompressive craniectomy reduces cerebral blood flow and improves oxygenation at the level of the decompressed hemisphere and at the contralateral level, while the metabolism is little modified. This throughput reduction may participate in the reduction of the ICP [7].

Cranioplasty may be a factor promoting hydrocephalus by restoring resistance to CSF resorption [7,8]. Several authors support this unconfirmed hypothesis. In a retrospective and non-randomized study by Bonis, et al. [9] 9 patients developed hydrocephalus after decompressive craniectomy crossing the midline out of a total of 26 patients. This study does not confirm with certainty that the hydrocephalus was related to decompressive craniectomy. Waziri, et al. [10] found 15 postoperative communicating hydrocephalus in a total of 17 patients and five of these patients received ventriculoperitoneal CSF diversion. He concluded that the diagnosis of hydrocephalus in the hemicraniectomy population is necessarily subjective. Nasi, et al. [11] found 37

postoperative hydrocephalus in 130 patients and 34 of these patients received ventriculoperitoneal CSF diversion.

In fact, after cranioplasty, the dilated ventricles can, via the cortical mantle, obstruct the subarachnoid space and thus constitute a resistance to the resorption of the CSF. However, injuries caused by head trauma can also lead to hydrocephalus.

The cranioplasty component was raised by intracranial hypertension caused by the increase in the intracranial fluid compartment [6,12]. Waziri and colleagues have recently suggested that decompressive craniectomy may play a role in the “flattening” of the normally dicrotic CSF pulse waveform seen in patients who undergo DC, due to the transmission of the pressure pulse out through the open cranium [10].

CONCLUSION

Cranioplasty may be a contributing factor to hydrocephalus in that it increases resistance to CSF resorption. It is important to take this into account when performing any cranioplasty.

AUTHORS' CONTRIBUTIONS

All the authors contributed to this work.

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