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"*Eppur si muove*" - Clinical case: evolutionary "saga" during the last 6 years: posttraumatic subdural hematoma, decompressive craniectomy, right hemiplegia and aphasia, cranioplasty, hydrocephalus and porencephaly, post-traumatic encephalopathy - in remission

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Abstract

The case reports a 59-year-old male patient who suffered a severe head injury (affirmative by accidental fall from 3 m) with multiple hemorrhagic lesions (bifrontal, bioccipital, biparieto-temporal) and left cerebral subdural hematoma, requiring a large fronto- temporo-parietal decompressive craniotomy for the mass lesion evacuation. Cranioplasty was performed after 6 months.
The paper synthesizes the evolution over six years of follow-up (12 in-patient admissions and 4 out-patient evaluations), like in a neurorehabilitation cinematographic "saga". The posttraumatic encephalopathy had a peculiar evolution, sugestively compared with the humps of a camel: the brain injury (determined coma, right hemiplegia and mixed aphasia, intense psycho-motor agitation, severe dysphagia for solids and liquids, neurogenic bladder, anemia), was followed by a slowly progressive favorable neuro-psychological evolution (after the decompressive craniectomy). A brutal neurological fall-down was noticed after the cranioplasty, and finally a gradually favorable ascending trend, towards a global neuro-psichological stabilization (with an almost imperceptible sequelary ataxic hemiparesis). The paper discusses the pathophisiological aspects focused on the decompressive craniectomy and cranioplasty, correlated to the patient's evolution. Complications of each neurosurgical procedures are succinctly depicted. The traumatic encephalopathy was complicated with post-traumatic seizures (therapeutically controlled) and active internal hydrocephalus with interstitial edema and an ischemic lesion. Finally it was a "happyend", with favorable clinical evolution, towards a stable and stationary normotensive asymmetric hydrocephalus, with a gigantic and deforming porencephaly. The paper advocates for a carefully follow-up and prompt intervention in order to prevent recurrences and/ or complications (secondary and tertiary prophylaxis).

Keywords: *traumatic brain injury, subdural hematoma, decompressive craniectomy; cranioplasty; internal hydrocephalus; post-traumatic encephalopathy; seizures; neurorehabilitation,*

Introduction

Decompressive craniectomy (DC) is an emergency neurosurgical procedure used in cases of severe intracranial hypertension, a condition commonly associated with severe traumatic brain injury (TBI). This procedure is often lifesaving, but it exposes the brain to atmospheric pressure during the subsequent rehabilitation period, which changes intracranial physiology and may lead to complications such as: hydrocephalus, hygromas, "syndrome of the trephined" (1)
DC for intractable intracranial hypertension due to stroke or TBI was proved for reducing mortality, but only at the expense of increasing the proportion of the severely disabled patients (2,3)
Cranioplasty (CP) is a neurosurgical intervention/ procedure aimed to repair or reshape irregularities, or imperfections in the skull. The earliest CP operation is dated to 3000 BC, in the Inca civilization. Precious metals were found in graveyards, next to trepanned skulls, suggesting that CP had been performed. In the Paracas region of Peru, was found a skull with a thin plate of gold covering a cranial defect, dating from 2000 BC.(4) The indications for cranioplasty after decompressive craniectomy are: cosmetic repair (aesthetic appearance) and mainly restoration of brain protection.
Different kinds of CPs to repair defects or gaps in the cranial bones and to restore the contour of the skull are performed with the original bone (cryo-stored of the autologous skull bone, after deep-frozen storage at -80 C) or with a contoured graft made from synthetic materials, such as: titanium (plate or mesh), synthetic substitute (acrylic, prefabricated or molded at the time of surgery).
Case report A 59-years-old male has suffered a severe head injury (on the 02 July 2014) by accidental fall from 3 m, with multiple hemorrhagic cerebral lesions (bifrontal, bioccipital, biparieto-temporal) and left subdural hematoma. A large fronto-temporo-parietal decompressive left cranio-tomy was performed, for the mass lesion evacuation (on the 04 July 2014). The patient`s acute evolution was exacerbated by a severe toxic hepatic cytolysis (probably induced by thienam).
Subsequently he was addmited to our neuro-rehabiliation Clinic (during 31 July 2014 – 24 October 2014.
At admission the biological status and neuro-psychological condition were severely degraded: he had right hemiplegia with global aphasia, intense psycho-motor agitation, severe dysphagia for solids and liquids, neurogenic bladder, anemia.

Cerebral CT examination revealed the persistence of a small residual bifronto-parieto-occipital hematoma (resolved gradually during hospitalization). (Fig.1)

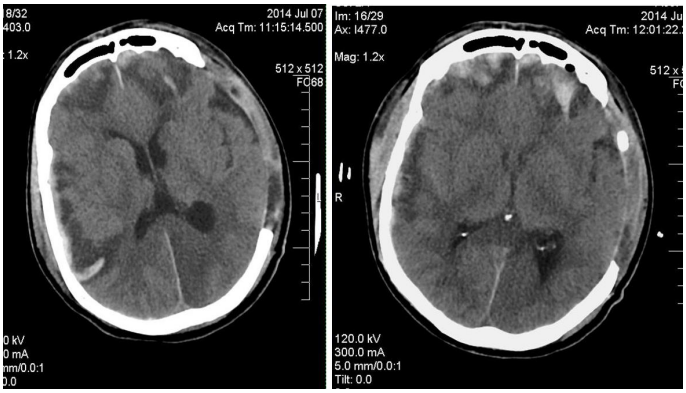

Fig.1 Cerebral CT (July 2014) status post large DC. Small residual bifronto-parieto-occipital hematoma

The patient had a favorable neurological evolution; he gradually recovered the receptive, then (partially) the expressive component of the language. Despite the precarious biological condition and severe brain injuries (post-traumatic and post-surgical ones) the neurological evolution was favorable. At discharge the swallowing and day-time bladder continence were significantly improved, hand functional gripping ability was mostly recovered, and the subject could walk on small distances (suported and supervised by the kinetotherapist). He requiried permanent support and supervision for the daily activities related to nutrition, self-care, problem solving and interrelation (Barthel disability score was 35%).

Some issues persisted after the first neuro-rehabilitative cure: the psycho-organic syndrome with psychomotor agitation and episodes of hetero-aggressiveness, the disturbance of the sleep-wake rhythm and negativism were only partially solved.

In January 2015 (after 6 months from the TBI) cranioplasty was neurosurgicaly performed, using a composite material (methacrylate, fig.2)

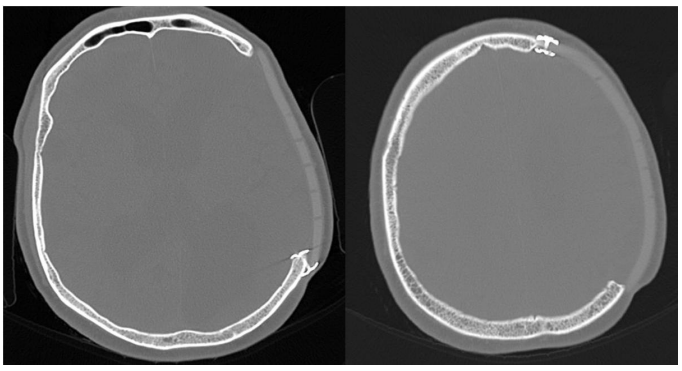

Fig.2 Cerebral CT bone incidence (Jan 2014): status post cranioplasty with methacrylate

A rapid decline of the neurological status was noticed after CP: the locomotor capacity was worse, with severe ataxia (constant tendency to right latero-deviation, requiring bilateral support), speech and swallowing (especially for fluids) were deteriorated, neuropsychiatric agitation and negativism increased, with disruption of the cognitive and executive superior cerebral functions, and incontinence re-emerged. MMSE assesment was 6/30, and the Barthel score collapsed at 5%. Complications were due to the active internal hydrocephalus (fig.3)

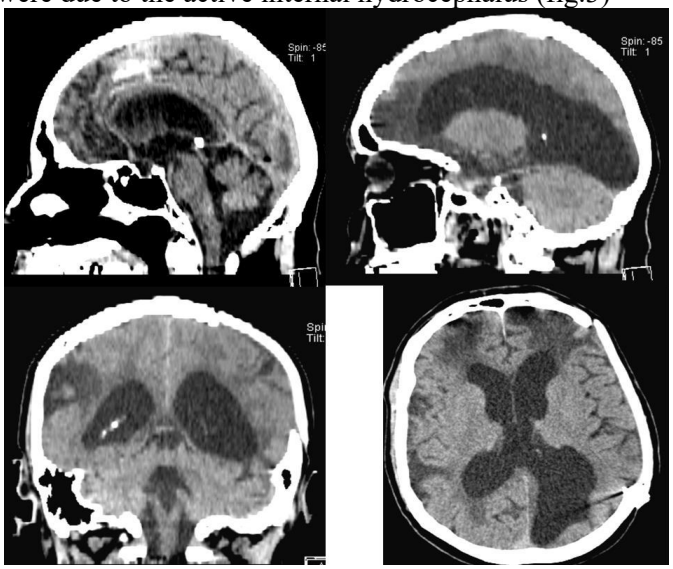

Fig. 3 Cerebral CT scan (2016): active hydrocephalus, the 3rd ventriculus diameter increased from 10-11 mm (in 2014) to 18 mm (2016), cerebral interstitial edema, and an associated vascular injury.

The active hydrocephalus was associated with atrophic changes of the nerve optic papilla, and an increased CSF pressure (manometry detected a CSF pressures over 200 mm H2O). A decompressive shunt was proposed, but the family refused this alternative.

During the next 7 months a gradual neurological improvement was achieved. This time interval was required for re-balancing the intracranial pressure. In November 2015 he still had important dysmnesia (MMSE was 18/30), but no longer urinary incontinence. The vertical balance was improved, and the Barthel score improved to 45%.

Medical treatment consisted in a synergistic combination aimed at diminushing the CSF pressure: inhibitors of CSF secretion (acetazolamide 750 mg/day), intermitent dexamethasone administration, and neurotrophic factors, sedatives and neuroleptics.

Around the winter Holidays in 2015 evolution was complicated with post TBI epilepsy (focal seizures with secondary generalization). Anticonvulsant treatment consisted of daily combination of 1000 mg levetiracetam and 300 mg oxcarbazepine (trileptal), and convulsions with motor manifestations ceased.

In 2016 the family reported two short episodes of temporo-spatial disorientation and dizziness, interpreted as complex psycho-cognitive focal symptomatic epilepsy (in the context of the bioelectrical EEG modifications).
Repeated EEGs have revealed persistence of the focal pathological bioelectrical pattern, but without clinical manifestations, during the entire time interval 2016-2019.
The aforementioned anticonvulsant schedule was maintained.
The good clinical evolution went in parallel with the favorable aspects of the brain imaging: the active internal hydrocephalus with signs of evolution and interstitial edema (noticed in 2016), "calmed down" to a stable and stationary normotensive asymmetric hydrocephalus and porencephaly (although gigantic and deforming; fig.4).

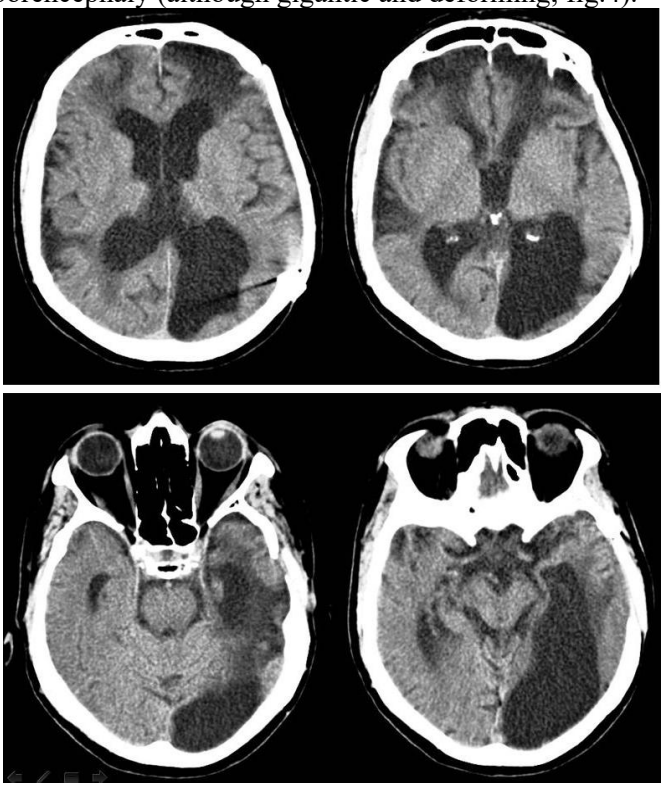

Fig.4 Cerebral CT scan (May 2019): stable and stationary normotensive asymmetric hydrocephalus with gigantic and deforming porencephaly

Kinetotherapy programs were gradually adapted, from rehabilitative nursing with correct posturing in bed, and ROM limb mobilization (from passive, to semi-active, active, active with resistance movements), to transfer maneuvers in the wheelchair and the gym apparatus (standing, trellis, pulley, electric and mechanical ergonomic bicycle, suspended frame, trampoline, stepper, roller device).
Assisted walking exercises with unilateral support on a fixed bar, then on a tetrapod cane, progressed to an independent walking (in 2019).

Discussion

The clinical case reports a male patient, monitored for 6 years (there were 12 in-patient short admissions and 4 out-patient evaluations).
Craniectomy has many known complications, with an the overall rate up to 53.9% (5). Literature emphasized that an extended craniectomy, a Barthel score ≤ 70 and age over 45 years are independent predictors for neurologic complications occured after DC; our presented subject cumulated all the negative predictive factors, mentioned above.
CP optimized the neurological recovery, both physiologically and/or clinically in our patient. It restored normal intracranial physiology with respect to changes in intracranial and postural pressures.
Neurological function improvement was noticed in an overwhelming majority of patients after CP (6-11). The metabolic deficits observed in the injured as compared with the noninjured hemisphere, were found to improve after reimplantation of the skull bone flap.
Although neurological status improvement after CP was repeatedly emphasized in the literature, the reasons for this still remains unclear. A systematic review suggests that CP improved cerebral blood flow and the hydrodynamic balance of the CSF, with a concomitant improvement of the neurological function (previously disturbed after DC). (12)
A famous paradigm says: "Time is Brain". The optimal timing for CP after DC for trauma remains unknown. (13)
It is believed that CP performed 3 to 6 months after decompression may significantly improve both motor and cognitive recovery. (14,15)
Early CP (preferably below 12 weeks, as soon as the brain is lax), is advisable to prevent long-term complications of the DC, and is warranted to facilitate rehabilitation in patients after DC. (16,17)
A better functional outcome was noticed after an early CP, comparing to the patients with a late intervention ($p<0.05$). (18)
In our subject CP was performed six month later, and that explains the major differences between pre-/ and post-cranioplasty neurological status, respectively the neuropsychic "collapse" and rapid CT image degradation.
One must emphasise that CP after DC is still associated with a relatively high complication rate, ranging between series from 12% to 50%, but good neurological outcomes after surgery always outweighs the complications.
Surgery-associated complications after bone flap reimplantation are: subgaleal collection (5.6%), hydrocephalus (4.2%), seizures (4.2%), bone flap infection (2.8%), intracerebral hematoma (2.8%), empyema (1.4%), subdural hematoma (1.4%). (17,18).

Another redutable post TBI and CP complication is represented by epilepsy. Post-traumatic seizures (generalized or focal / partial, simple or complex type) may happen early (in the first week after TBI), or late post-traumatic epilepsy. (17-19)

Late seizures usually occur after 3 months, peak at 6 months, and have a double risk of appearance if cranial fractures were associated (an open, penetrating trauma). Approximately 5% of all TBI patients will experience at least one chronic seizure.

Case particularity

The reported case depicts a six-year follow-up interval (like a neurorehabilitation "saga"). The global neurological evolution was gradually favorable (towards minimal sequels, with an almost imperceptible ataxic hemiparesis, and no aphasia), reaching a Barthel score of 75% and good psychological recovery.

One must notice the spectacular contrast between the impressive residual cerebral imagery and the incredibly good neuropsychological status, reached in November 2019, and maintained until August 2020.

Conclusions

"Nothing is lasting out of time," and neurorehabilitation is a long-life process. The paper advocates for a carefully follow-up and prompt intervention, in order to prevent recurrences and / or complications (secondary or tertiary prophylaxis).

Disclosure Statement

The authors have no conflicts of interest to disclose.

Statement of Ethics

The manuscript was prepared in compliance with all ethical and confidentiality guidelines and principles. Written informed consent was obtained from the patient's next of kin for publication of this case report and accompanying images. The Ethic Committee of Teaching Emergency Hospital Bagdasar-Arseni approved publishing

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