Cranioplasty for the Relief of Chronic Pain: 70 Years after the Debridement of the War-Penetrating Craniocerebral Injury

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Abstract

We report the case of a 75-year-old female patient who underwent primary debridement of the penetrating craniocerebral injury at the age of 5 years. The injury was caused by the explosive shrapnel wound in the parietal right-side region because of an air raid in Yugoslavia in 1944 during World War II (WWII) combat. The scull defect that remained was not repaired. The patient developed the severe allodinic pain syndrome in the skin over the cranial defect, 70 years after the surgery. The skin over the cranial defect was infolded inside the skull and stretched over the bony ridge. The pain was relieved by cranioplasty that restored the cranial vault and reversed the infolding skin over the craniectomy defect. The mechanism of the pain and its relation with the morphologic changes of the primary craniectomy and brain debridement over time are discussed.

Keywords
- chronic pain
- penetrating brain injury
- explosive wound
- debridement of the gunshot brain injury
- cranioplasty

Introduction

It has been shown that 35 to 40% of all combat deaths occur due to brain wounds. The craniectomies around the entrance site of a projectile and aggressive debridement and an attempt to extract all retained bone and/or metallic fragments have been the favored technique in previous military conflicts.[1] We report the case of the 75-year-old female patient who suffered penetrating craniocerebral injury at the age of 5 years. The injury was caused by the explosive shrapnel wound in the parietal right-side region because of air raid in Yugoslavia in 1944 during World War II (WWII) combat. She was comatose after the injury and was treated by using primary surgical debridement in local hospital. After the hospital staff had been taken as war prisoners, she was observed, attended, and helped by her mother only. There were no additional details regarding the clinical course of this patient. She eventually recovered and lived normal life for 70 years until development of severe pain syndrome in the skin over the craniectomy skull defect, as the late and rare complication of previous surgery (►**Fig. 1**). The computed tomographic (CT) scan showed the cranial defect over the right parietal region, oval in shape with diameter of 8 cm. The postdebridement cavity walls appeared clear, with no retained bone or metallic fragments, suggesting successful initial debridement that was radical as it was a rule of WWII surgical strategy[1] (►**Fig. 2**).

However, given the size of the skull at the age of 5 years, we can speculate that the broad craniectomy that secured thorough decompression was what might allow for her survival since she lost the proper medical treatment shortly after the surgery, due to war situation. The onset of severe pain syndrome in the skin over the cranial defect, 70 years after the surgery, was treated by the neurologist, with analgesics, gabapentin, and pregabalin (Lyrica), and eventually Durogesic flaster was applied. However, all these medications failed to relieve pain sufficiently for normal daily activities and sleeping pattern. Thereafter, the patient was referred to neurosurgical assessment for possible pain treatment.

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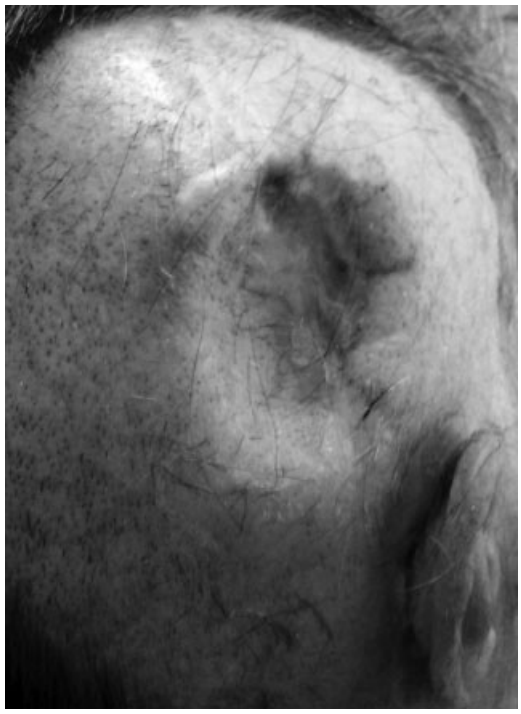

Fig. 1 The craniectomy skull defect: infolding and stretching of the skin over the bone edge.

Pain Syndrome Assessment

The pain was determined using McGill Melzack Pain Questionnaire and visual analog scale (VAS) scale.[2] The patient suffered from severe pain, VAS /10. The nature of the pain was incisive, throbbing, and sharp without thermal pain equivalents. The skin over the cranial defect was atrophic, with irregular, stellate-like scar, infolded inside the skull, and stretched over the bony ridge, tense on palpation with no fluctuation (►**Fig. 1**). The territory of pain was in the skin in the injured area. There was severe allodinic pain at that same territory that disturbed her sleeping pattern completely. It was our impression that possible mechanism of the pain might have been the stretching of the cutaneous nerves over the bone edge, with possible neuroma formation caused by the progressive infolding of the skin over time, due to the atrophic changes of the brain and forced by the external atmospheric pressure.[3] The most intensive allodinic pain was elicited on the posterior edge of the craniectomy where the stretching of the skin over the bone edge was most intense. For the assessment of the underlying pain mechanism, the repeated diagnostic local infiltration of the skin over the posterior ridge of the craniectomy defect was done with lidocaine. This infiltration relieved pain completely. Such result indicated the stretching of the nerves over the bony edge as underlying mechanism of the pain. Thereafter, we believed that the cranioplasty that was aimed to restore cranial vault convexity, thus reversing and relaxing the infolded skin over the craniectomy area, could relieve cutaneous nerves stretching as supposed mechanism of this severe pain syndrome. The skin that was atrophic and the irregular scar, located in the central area of the infolding skin flap raised an important concern—the necrosis of the skin flap after its elevation for cranioplasty. Scalp management is challenging in all types of cranioplasties. The quality of the soft tissue envelope surrounding a cranioplasty implant is critical to long-term success. Poor-quality soft tissues are associated with cranioplasty failure.[4,5] It is advised that the incision for cranioplasty should follow the prior incision, with care taken to stay directly on the scar to avoid necrosis of the scalp.[6] Reflection of the scalp flap is often difficult because the normal tissue planes are usually distorted. However, in this case the skin incision was done in the horseshoe fashion distant from the existing scar area, providing broad basis and securing irrigation of the cutaneous flap. The skin layer was carefully and successfully dissected in the sufficient thickness using sharp preparation. The cranioplasty was done with methylacrilat.

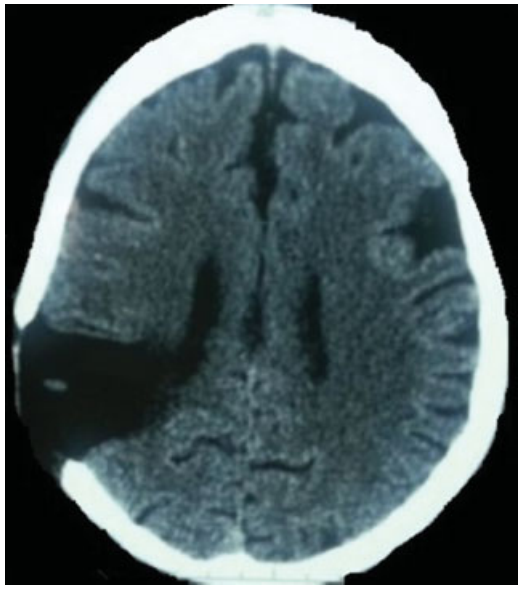

Fig. 2 CT scan: brain debridement cavity 70 years after the surgery.

Result

In the postoperative course, the patient reported the complete pain relief. The wound healing was successful. There was no necrosis of the skin flap. In the follow-up of 34 months, the pain relief is complete, and the local wound status is normal. The CT examination, done after 6 months, showed the brain tissue expanded and filled former postdebridement cystic space (►**Fig. 3**).

Discussion

Skull defects that require reconstruction are common in primary debridement of penetrating brain injuries. The cranioplasty restores normal intracranial pressure relationships within the skull and improves cerebrospinal fluid (CSF) flow dynamics.[4] The goal for the repair of these defects may be cosmetic, but, however, cranial bone provides protection not only from the possible risk of accidental impact but also from the constant atmospheric pressure through the skull defect. Before the cranioplasty is planned, the overlying scalp must be well healed and vascularized, the neurologic

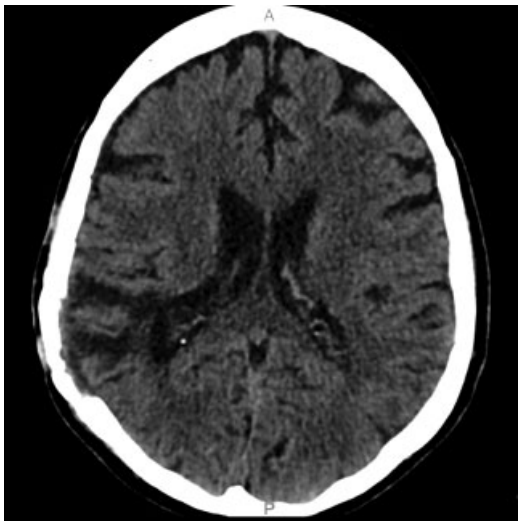

Fig. 3 CT scan 6 months after cranioplasty: brain tissue expanded and obliterated debridement cavity.

status should be completely stabilized, and infections must be fully treated. Although cranioplasty is typically performed nearly 3 months after traumatic brain injury, recent reports indicate that in select, otherwise healthy, patients, early cranioplasty after 5 to 8 weeks may aid recovery.[4] In our patient, the cranioplasty was done 70 years after the injury that she sustained at the age of 5 years. The patient showed normal mental development and functional life for this long time. Eventually, the reason that urged medical attention was a pain syndrome located in the skin over the craniotomy defect. Based on the diagnostic local skin infiltration and the result of cranioplasty that achieved complete pain relief, we concluded that the pain was caused by the stretching of the nerves over the bone edges due to the infolding of the skin over the bony skull defect. To minimize the risk of the necrosis of the skin flap, of crucial importance was to identify the plane between the galea and the dura mater, avoiding thinning and perforation of the skin flap in particular. This case confirms the fact that cranial defect that was not covered by cranioplasty in the long term caused epicranial skin infolding due to the atmospheric external pressure through cranial defect on the one hand side and progressive atrophy of the brain tissue itself on the other hand side. The progressive skin stretching caused intensive, unbearable pain syndrome developed most probably due to the stretching of the cutaneous sensory nerves. However, we failed to identify any formation in the skin flap that might have been consistent with neuroma formation. After cranioplasty was done, the brain tissue expanded and filled former cystic formation. This confirms that external atmospheric pressure exerted pressure on the brain tissue through the cranial defect. However, the mental performance of the patient did not improve.

Conclusion

The case reported proved that the reconstruction of the cranial vault after decompressive craniectomy for craniocerebral penetrating wound debridement was not necessary for the recovery and normal mental performance of the patient in the long period of 70 years after the injury. It revealed that the cranial defect is a dynamic condition that undergoes changes driven by the aging process and brain atrophy on the one hand side and constant atmospheric pressure across the skin over the cranial defect on the other hand side. The resultant infolding and stretching of the skin over the bony edges caused severe, unbearable pain syndrome that eventually required cranioplasty. Regardless of this case, we recommend routine cranioplasty after the primary debridement of penetrating gunshot or explosive craniocerebral wound.

Note
This article has never been submitted, published, or reported elsewhere. All authors approve the article for publication.

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The authors have no financial disclosures to report.

Conflict of Interest
The authors have no personal or institutional conflict of interest to report.

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