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A SURGICAL METHOD FOR THE PREVENTION OF THE "TREPANNED SKULL" SYNDROME AND THE OPTIMAL TIMING OF ITS IMPLEMENTATION IN PATIENTS WITH POST-TRAUMATIC DEFECTS OF THE CRANIAL VAULT

Usmanov Rakhmatillo Fayzullayevich

Candidate of Medical Sciences, Assistant Departments of Maxillofacial Surgery Samarkand State Medical University

Article history:	Abstract:
Received: July 20 th 2024 Accepted: August 14 th 2024	It is known that patients with cranial arch defects, under the influence of atmospheric pressure and other provoking factors, gradually develop the "trepanned skull" syndrome, which includes several components in the form of meteopathy, asthenia, psychopathy, limb paresis, aphasia and epileptic seizures. Epileptic seizures are one of the frequent elements of the "trepanned skull" syndrome. The focal component of these seizures has a topographic localization corresponding to the localization of the bone defect. It has been established that the main cause of the development of epileptic seizures in patients with cranial arch defects is the development of an aponeurotic- meningocerebral scar in the area of the postoperative defect, which is confirmed by the development of a positive clinical effect after the operation "excision of meningocerebral scars followed by cranioplasty."

Keywords: Cranioplasty, trepanated skull syndrome

INTRODUCTION: Defects in the bones of the skull of different origins are not only a cosmetic problem, but also threaten the integrity of the brain, which becomes vulnerable at the site of deformation. As a rule, the clinical picture in such patients consists of a complex of symptoms caused, firstly, by a violation that caused damage to the bones of the skull (tumor, purulent process, trauma, hematoma, etc.), and secondly, by the so-called "trepanned skull syndrome". The second component inevitably arises under the influence of physical and psychological factors. An area devoid of a part of the skull is exposed to atmospheric pressure, the brain can be injured in the area of contact with the edges of bone tissue, cerebrospinal fluid circulation and hemodynamics cerebral are disrupted. The psychological factor is also quite significant. A person can feel the pulsation of the brain matter and acute insecurity from the influence of any factors. Often, a violation of the integrity of the skull is accompanied by paresis, epileptic seizures, psychopathies and asthenia. **RESEARCH MATERIALS AND METHODS: 117 (63** men, 54 women) patients with post-traumatic cranial arch defects were examined. In 67 (57%) patients, defects of the cranial vault remained after the operation of "decompressive trepanation of the skull" performed for a depressed fracture; in 34 (29%) patients, the defect of the cranial vault remained after the operation of cranial trefination with the removal of subdural hematoma and drainage of the hematoma cavity, and

in 16 (14%) patients, the defect of the cranial vault remained after the operation of decompressive trepanation of the skull with the removal of epidural hematoma.In 83 (71%) patients, the values of posttraumatic cranial arch defects were of average magnitude (diameter up to 6 cm), and in the remaining 34 (29%) patients, the values of post-traumatic defects of the cranial vault were large (diameter greater than 6 cm).For the timely detection of signs of the "trepanated skull" syndrome, clinical neurological, electroencephalographic (computer EEG), transcranial Dopplerography (TKDG) studies were performed dynamically (every 6 months for 3 years) in all patients and for the purpose of timely detection of aponeuroticmeningocerebral scar in all patients (every 6 months) dynamic magnetic resonance imaging (MRI) studies. TKDG was produced using the universal ultrasonic device "Aloca SSD-3500". It is known that according to the timing of cranioplasty surgery, there are primary (immediately after decompressive trepanation of the skull), primary delayed (up to 7 days after decompressive trepanation of the skull), early (up to 2 months after decompressive trepanation of the skull), late (more than two months after decompressive trepanation of the skull) [9]. Based on this, in order to determine the optimal period for cranioplasty, the clinical course of post-traumatic cranial arch defects was divided into 3 periods. 1) Acute period — the first 10 days after injury. During this period, primary and



primary delayed cranioplasty are performed; 2) Early recovery period - up to two months, after decompressive cranial trepanation - early cranioplasty; 3) Late recovery period — more than 2 months after decompressive cranial trepanation - late cranioplasty; It is known that for a long time in practical neurosurgery, an adequate way to close small and medium-sized defects of the cranial vault was considered the operation of "plasty of the defect of the cranial vault with an autosteal crumb" (3,6). However, the study of the long-term results of "plasty of the defect of the cranial vault with an autosteal crumb" showed that the bone crumb (taken from the edges of the bone defect by biting) covering the defect of the cranial vault gradually resolves, and the bone defect is filled with fibrous tissue. Resorption of bone chips leads to the development of the "trepanated skull" syndrome, which is why the method of "plastering a skull defect with an autosteal crumb" is rarely used in clinical practice.

The choice of an adequate method of cranioplasty was carried out on the basis of literature data, according to which currently, to close small and medium defects of the cranial vault, the most effective and common method of cranioplasty is "explantation of a defect of the cranial vault with radiopaque bone cements with excision of aponeurotic-sheathed cerebral scars and plasty of a defect of the dura mater with silicone film" Based on this, we this method was used. The method includes: Exposure of a bone defect, excision of aponeurotic-meningocerebral scars, plasticization of a defect of the dura mater (if any) with silicone film, manual preparation and installation of an implant made of bone cement "PALACOS-R". Primary cranioplasty, that is, primary explantation of the cranial vault defect with bone cement "PALACOS-R" was performed in 27 (40%) patients with depressed fractures of the cranial vault, the remaining 40 (60%) patients failed to perform primary plastic surgery of the cranial vault defect due to the presence of cerebral edema. That is, these patients underwent decompressive trepanation of the skull without plasty of the ventral arch defect. After appropriate conservative treatment, these patients underwent "primary-delayed explantation of a cranial arch defect with PALACOS-R bone cement" (10 days after receiving TBI). 26 (39%) patients underwent early explantation of the cranial vault defect with PALACOS-R bone cement and plasty of the dura mater defect with silicone film, and the remaining 24 (20%) patients underwent explantation of the cranial vault defect in the late period. That is, these patients with post-traumatic defects of the cranial vault underwent late cranioplasty (they applied late). Statistical processing of the obtained data was performed using descriptive methods and the ANOVA model. A comparative assessment of the change in indicators compared to the baseline level was carried out using a t-test.

RESULTS AND DISCUSSIONS: 83 (70%) patients (67 with depressed fractures, 16 with epidural hematoma) underwent "decompressive craniotomy" operations, and 34 (30%) patients underwent "cranial trefination with emptying of subdural hematoma and drainage of its cavity". That is, in 83 (70%) patients, the defect of the cranial vault remained after the operation of "decompressive trepanation of the skull", and in 34 (30%) patients, the defect of the cranial vault remained after the operation after the operation of "trefination of the skull".

Primary explantation of cranial arch defects with PALACOS R bone cement was performed in 27 (23%) patients (20 with depressed fractures, 7 with epidural hematoma). And in other patients, due to the presence of contraindications (comatose states, severe general condition, the presence of severe somatic diseases, etc.), it was not possible to perform primary plastic surgery of the cranial arch defect. After appropriate treatment of traumatic brain injury, 18 (15%) patients underwent primary delayed explantation of a skull defect with PALACOS R bone cement. The remaining 72 (62%) patients did not agree to primary delayed cranial plastic surgery.

The clinical and neurological symptoms of the acute period regressed gradually. Long-term dynamic clinical neurological computer EEG, CTG and MRI observation showed that patients who underwent primary and primary delayed explantation of skull defects with PALACOS-R bone cement did not develop trepanated skull syndrome.

In the early recovery period (up to 2 months after the operation of decompressive trepanation of the skull), a dynamic clinical and neurological study showed that 39 (33%) patients developed the "trepanated skull" syndrome, which was expressed in the form of asthenization of the nervous system and increased sensitivity to meteorological changes. Clinically, no epilepsy attacks were noted, however, the results of additional research methods showed that in all patients, a diffuse increase in epileptic brain activity was noted on the computer EEG, and during mapping, a hypometabolic site was determined by the projection of a bone defect. TCDH showed a decrease in cerebral blood flow of the 2nd degree. The data presented show that this group of patients is at risk of developing epilepsy attacks. Based on this, in order to prevent the development of epilepsy, it was decided to perform operations, excision of aponeurotic-meningocerebral scars with plasty of the cranial arch defect with bone cement "PALACOS-R" - early cranioplasty.

All patients had interoperatively detected aponeuroticsheathed or aponeurotic-sheathed cerebral scars, which are easily corroded. All patients after early cranioplasty received conservative pathogenetic treatment with



antihypoxants and anticonvulsants every 6 months. After a course of conservative pathogenetic treatment, the "trepanated skull" syndrome regressed in all patients, increased epileptic activity on a computer EEG hypometabolic exercises and passed, changes pathomorphological characteristic of apovneurotic-sheathed aponeurotic-sheathed or cerebral scars disappeared on MRI, cerebral blood flow normalized on TCD, no epilepsy attacks were noted. Thus, as a result of the pathogenetic conservative treatment, the "trepanned skull" syndrome regressed, that is, these patients recovered.

In the late recovery period, the "trepanned skull" syndrome developed in 24 (20%) patients. The syndrome of the "trepanned skull" in these patients was expressed in the form of asthenization of the nervous system, increased sensitivity to meteorological changes, the development of a mild hemisyndrome (in 8 (7%)) patients), the appearance or increase in epileptic seizures. Epilepsy attacks in these patients developed six months later (on average after the operation of "decompressive treponation of the skull"), 9 (8%) patients had attacks of focal seizures without generalization, and in the remaining 11 (9%) patients, epilepsy attacks began with a focal component followed by generalization. The focal component of epileptic seizures in all cases topically corresponded to the site of the skull defect, and 5 (4%) patients had a primary generalized form of epilepsy. In all patients, computer EEG showed a diffuse increase in epileptic activity with the development of hypometabolism of brain tissue under the defect of the cranial vault, dynamic MRI showed an increase in aponeurotic-sheathed or aponeurotic-sheathed cerebral scars with tightening of the anterior horn or the body of the lateral ventricle (on the side of the defect). A decrease in cerebral blood flow of the 3rd degree was noted on TCDH. And in 4 (3%)% of patients with post-traumatic cranial arch defects, despite the absence of epilepsy attacks, an increase in epileptic brain activity was noted on computer EEG.

Considering that 20 (17%) patients had frequent epilepsy attacks (2-3 per week) with a diffuse increase in epileptic brain activity on EEG and dynamic MRI studies showed signs of an increase in aponeuroticmeningocerebral scars and in 4 (3%) patients, despite the absence of epilepsy attacks, diffuse EEG was noted on computer EEG increased epileptic brain activity, that is, these patients were at risk of developing epilepsy attacks. It was decided to perform the operation "explantation of the cranial arch defect with PALACOS-R bone cement" Intraoperatively in 11 (9%) patients, strengthened aponeurotic-sheathed scars were found, and in the remaining 13 (11%) patients, aponeuroticsheathed brain scars were found. In all patients, the postoperative period proceeded smoothly, but the frequency of epileptic seizures did not decrease in any patient. In this regard, long-term conservative treatment with the use of energy-protective and anticonvulsive therapy was carried out, on average for 3 years. After the start of conservative therapy, the frequency of epilepsy attacks began to decrease after the third course of pathogenetic treatment, and after the 6th course, epilepsy attacks stopped.

CONCLUSIONS: Thus, 9 (8%) patients, despite the presence of the "trepanated skull" syndrome, did not agree to the late "explantation of the cranial vault defect". An adequate way to prevent the "trepanned skull" syndrome in patients with post-traumatic cranial arch defects is timely plastic surgery of the cranial arch defect (cranioplasty) with bone cement "PALACOS-R". The optimal period of cranioplasty surgery preventing the development of trepanated skull syndrome is acute (the first 10 days after injury) and early recovery (up to 2 months after cranial trepanation) periods during posttraumatic cranial arch defect. This means that the most effective way to prevent trepanated skull syndrome is primary and primary delayed cranioplasty. Early cranioplasty with pathogenetic conservative treatment increases the effectiveness of treatment of the already developed trepanated skull syndrome and prevents the development of epilepsy.

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