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Successful recovery of a young man with traumatic brainstem ischemia in a motor vehicle accident. A case report

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Abstract

Introduction: The most common etiological factors of brainstem stroke and ischemia are atherosclerosis, thromboembolism, tumours, arterial dissection, and trauma. Infarction of the medulla oblongata in 73% of cases occurs as a result of stenosis of the vertebral arteries, 26% due to arterial dissection, while the topic of traumatic brainstem ischemia is still under-researched and under-represented.

Case report: We present a case of a 26-year-old male patient who was admitted to the Emergency Department due to multiple injuries of the head, chest, and upper, and lower extremities sustained as a motorcyclist in a traffic accident. A cranial CT scan revealed the presence of a minor subdural hematoma, traumatic brainstem ischemia, and a ruptured peduncular artery. From a neurosurgical perspective, surgical treatment was not indicated, thus a conservative therapeutic approach was adopted. After five days after admission, initial radiographic signs of ischemia regression were recorded, followed by complete absence.

Conclusion: Timely patient transportation from the accident site to the Emergency Center, followed by an appropriate multidisciplinary treatment approach and intensive neuroprotective and neuroregenerative therapy, along with organism reperfusion, played a pivotal role in the neurological, and subsequently, overall patient recovery in this case presentation.

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Introduction

The brainstem is located in the posterior part of the brain and consists of the medulla oblongata, pons, and midbrain. It represents the connection between the cerebrum, cerebellum, and spinal cord. The brainstem is vascularized by the vertebrobasilar system (8).

A lack of oxygen to any part of the brainstem causes a brainstem stroke. Timely detection and intervention reduces the rate of morbidity and mortality (7).

Stenosis and occlusion of the posterior circulation cause significant hypoperfusion of the brainstem. The most common etiological factors of brainstem stroke are atherosclerosis, thromboembolism, tumors, arterial dissection, and trauma. Infarction of the medulla oblongata in 73% of cases occurs as a result of stenosis of the vertebral arteries, 26% due to arterial dissection (2).

Case report

A 26-year-old male patient was admitted to the Emergency Department due to multiple head, neck, chest, upper and lower extremity injuries sustained in a motor vehicle accident. The transportation from the accident site to the Emergency Department lasted 45 min. A CT scan of the head was performed on the day of admission, 21.06.2023., which showed multiple punctate hemorrhages, a left-sided laminar subdural hematoma, a tentorial subdural hematoma, and brainstem ischemia (Figures 1A, B, C).

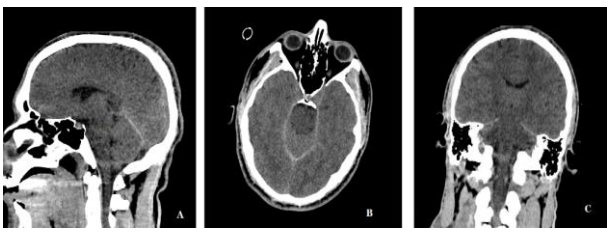
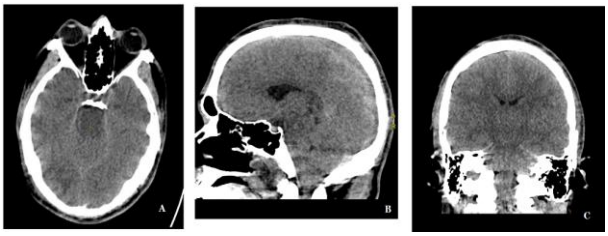


Figure 1.

Figure 2.



A follow-up CT scan of the head and CT angiography were performed on day 26.06.2023., showed a trace of subarachnoid hemorrhage in the interpeduncular cistern, and early signs of regression of brainstem ischemia. The right vertebral artery was not opacified in the V1 and proximal V2 segments, and was heterogeneously opacified distally. A hematoma was seen retroclavicularly in the prescapular segment of the right subclavian artery (Figures 2 A, B, C, and 3 A, B).

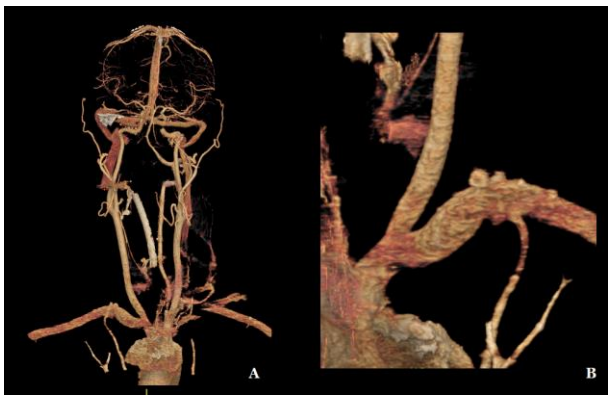


Figure 3.

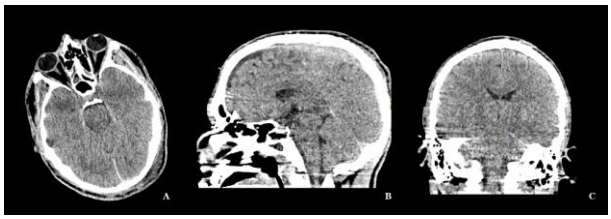


Figure 4.

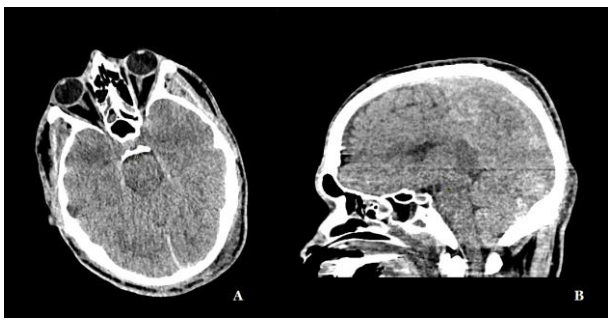


Figure 5.

A follow-up CT scan of the head was performed on day 7, which showed further resolution of the hemorrhagic collection (Figure 4 A, B).

A follow-up CT scan of the head was performed on day 10, which showed no evidence of brainstem ischemia (Figures 5A, B, C).

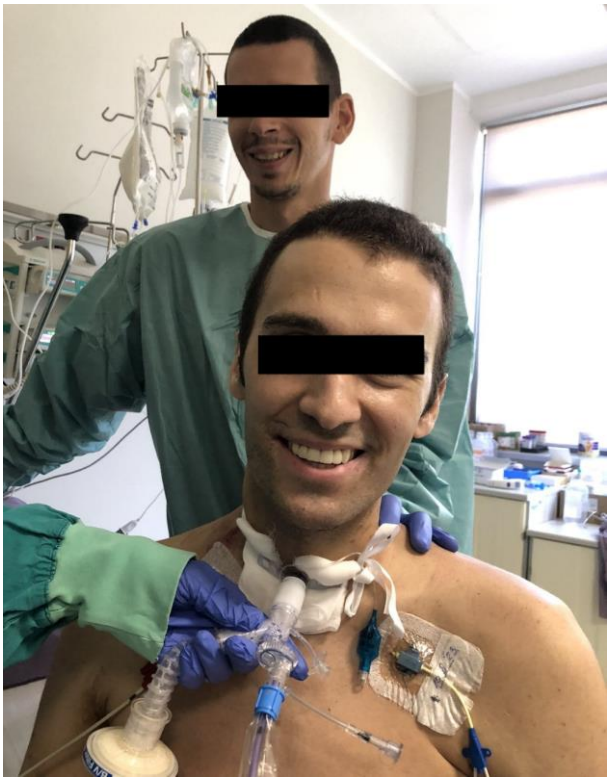


Figure 6.

The patient was treated conservatively with medication and adapted treatment modality due to other injuries. Antibiotics where administered the whole time during the hospotalisation, while intensive polyvitamin therapy, receiving vitamin B1 and B6 for the first three days, along with vitamin C in high doses and MgSO4 were administered. The patient was continuously sedated with midazolam, while propofol was used three times per day, also noradrenaline, and anti-edema therapy with mannitol for the first five days of hospitalization were administered alongside with phenobarbital. In addition to this therapy, the patient was administered infusion solutions to replenish fluids and stimulate the body's perfusion, as well as the necessary doses of blood transfusions and blood products.

On the fourth day of hospitalization, the patient was given dexamethasone, for the next seven days. On the fifth day of hospitalization, folic acid was added to the therapy, and mannitol was excluded from the therapeutic module with the last dose given. The homocysteine level of this patient was normal the whole time.

During the first three days of hospitalization, no anticoagulant therapy was administered, as it was contraindicated. The patient showed neurological progress with a visible regression of the radiographic findings of brainstem ischemia.

Anticoagulant therapy was started on the fourth day of hospitalization. The patient did not show any radiographic or neurological deterioration until the end of hospitalization. The patient remained on a multivitamin therapy, and adequate infusion solutions according to the patient's metabolic needs until discharge from the hospital with full neurological recovery (Figure 6.).

Discussion

Pathophysiology

The pathophysiology of all infarctions is the lack of oxygen in the tissues, which consequently leads to tissue death. Although the human brain accounts for only 2% of the total body mass, it requires 20% of oxygen (4).

The blood flow through the brain is regulated by autonomic mechanisms, thus maintaining constant perfusion and adequate venous drainage. The brain is also unique in that it has no or very little energy stores and uses glucose as its primary source of energy, with ketone bodies as metabolic products that are only used during starvation (5).

The cellular cascade processes that occur are: depletion of ATP due to lack of aerobic respiration of mitochondria; loss of membrane ion pump function and disturbance of the voltage gradient along the membranes with subsequent cellular edema; excitotoxicity of neurons due to the release of glutamate and synaptosomal-associated protein 25, causing further deterioration of energy levels and membrane potentials; production of free radicals; subsequent cell death (1).

The previously mentioned apoptotic and necrotic pathophysiological pathways activate protective pathways such as: expression of heat shock protein 70, B-cell lymphoma 2 gene family, and prion protein to prevent activation of the apoptotic cascade; release of Neurotrophin-3, Interleukin-10, and Granulocyte-colony stimulating factor; aimed at helping to activate survival pathways and reduce pro-inflammatory cytokine activities (1).

The cellular cascade is potentially reversible, which can lead to subsequent vasogenic edema within the next few hours. Vasogenic edema causes an increase in pressure in the surrounding tissue,

resulting in a mass effect and worsening of the clinical state of the patient (3).

Traumatic brainstem ischemia, if diagnosed, is predominantly fatal. The case report by Yoshikuni Mimata et al. showed us that there are 38 reported cases of ischemia in the vertebrobasilar region as a result of cervical spine trauma, with men being predominantly represented. The age range was from 20 to 83 years (average 40.5 years) at the time of the trauma. Of these, 18 cases were of traffic accident origin, falls in 10 cases, sports injuries in 5 cases, while the remaining cases were of origin using brute force. In the aforementioned study, a 69-year-old male patient was presented who was injured in a fall down the stairs and sustained head injuries. A C5-C6 level injury and ventral epidural hematoma were diagnosed on MRI. CT angiography of the neck showed occlusion of the right vertebral artery at the C4 level of the spine. Anticoagulant therapy was applied after the diagnosis of arterial occlusion. The patient died 52 hours after the injury (6).

The second case report by Pengyu Huang et al. showed us a 26-year-old patient who was diagnosed with brainstem infarction. The patient fully recovered after 5 months (9).

Our patient, in comparison to the previous ones, can be distinguished by the therapeutic approach and the origin of brainstem ischemia. We used a wide pharmacological spectrum, considering the origin of polytrauma.

Midazolam and propofolol in combination with intensive multivitamin and antioxidant therapy, and anticoagulant therapy, proved to be beneficial.

Midazolam exhibits its neuroprotective effect in certain neuropathological conditions. An experimental study conducted by Zhiyin Tang et al. showed the neuroprotective effect of midazolam. They showed that dysregulation of EAAT2 expression may be associated with neural injury induced by H/R in rat pups (10).

Propofol also exhibits a neuroprotective effect. In an experimental study conducted by Felix Ulbrich et al. the mechanism of propofol was investigated by modulating the TLR-4-NF-kB pathway. Propofol preserved the integrity of the mitochondrial membrane in oxygen-glucose deprivation injured cells. Propofol diminished TLR-4 surface expression and preserved the DNA-binding activity of the protective hypoxia inducible factor 1 (HIF-1) transcription factor (11).

Our case report of a 26-year-old patient with traumatic brainstem ischemia leaves room for thought about how much the multidisciplinary approach had an impact, together with the timely transport of the patient from the accident site to the Emergency Department, on the withdrawal of ischemia. Numerous studies are needed on both spontaneous and traumatic brainstem ischemia in order to gain knowledge from different experiences and achieve improvement for our patients.

Conclusion

Timely patient transportation from the accident site to the Emergency Center, followed by an appropriate multidisciplinary treatment approach and intensive neuroprotective and neuroregenerative therapy, along with organism reperfusion, played a pivotal role in the neurological, and subsequently, overall patient recovery in this case presentation.

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