Case Report / *Olgu Sunumu*

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Traumatic Middle Cerebral Artery Occlusion: A Case Report

Travmatik Orta Serebral Arterin Tıkanması: Olgu Sunumu

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Abstract

Traumatic occlusion of the middle cerebral artery (MCA) is a rare cause of cerebral infarction. Malignant infarction of the MCA is associated with a mortality rate of 80%. Arterial dissection, cerebral vasospasm, and thrombosis may be the pathogenesis of this condition. Poor admission Glasgow coma scale score, low systolic blood pressure, and brain herniation are significantly associated with development of posttraumatic cerebral infarction. There is no effective medical treatment for malignant MCA infarction. Decompressive craniotomy is the last solution to prevent severe damage or death. We report a 51-year-old female having craniofacial injury, multiple chest trauma, hemothorax, pneumothorax, and cerebral infarction after a traffic accident. Despite decompression surgery, she died two days after the injury.

Keywords: Middle cerebral artery, trauma, decompressive craniectomy, posttraumatic cerebral infarction

Öz

Orta serebral arterin (OSA) travmatik kapanması serebral damar tıkanıklığının nadir sebebidir. Malign OSA damar tıkanıklıkları %80 mortalite oranı ile ilişkilidir. Arter duvarının yırtılması, serebral damar spazmı ve trombozis bu durumun patogenezinde rol oynuyor olablir. Glasgow koma skorunun düşük olması, düşük sistolik kan basıncı ve beyin herniasyonunun travma sonrası serebral damar tıkanıklığı gelişmesi ile manidar ilişkisi vardır. Malign OSA damar tıkanıklığının etkili bir medikal tedavisi yoktur. Dekompresif kraniyotomi devam eden hasar veya ölümden korunmada son çözüm yoludur. Biz, trafik kazası sonrası yüz ve kafa travması, çoklu gögüs travması, hemotoraks, pnömotoraks ve serebral damar tıkanıklığı olan 51 yaşında bayan hastayı sunduk. Dekompresif cerrahiye rağmen hastamız travma sonrası iki gün sonra kaybedilmiştir.

Anahtar Sözcükler: Orta serebral arter, travma, dekompresif kraniektomi, travma sonrası serebral damar tıkanıklığı

Introduction

Posttraumatic cerebral infarction (PTCI) is a well-known complication of traumatic brain injury, with a frequency ranging from 1.9% to 10.4 %. After head trauma, PTCI may be seen as a complication (range: 1.9%- 10.4%) (1-4). Despite appropriate medical and surgical interventions, PTCI has poor clinical outcome with a high mortality rate. The prognosis of malignant middle cerebral artery (MCA) infarctions is poor, with case fatality rates in intensive-care-based series of nearly 80% (5).

PTCI risk factors are lower Glasgow coma scale (GCS) score, hypotension and higher intracranial pressure (ICP). Early identification of patients who are at a particular risk for PTCI would be extremely helpful in surgery. Early decompressive surgery (DC) confers a survival benefit (2,4,6).

Cerebral vasopasm, vascular injury, and hypoperfusion may cause PTCI. (1,3,4). Traumatic brain injury pathophysiology includes brain swelling, increased ICP, reduction in blood and oxygen supply, energy failure, and cell death (4). Thrombus formation is unclear. Acceleration,

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deceleration, or rotational motions between the brain, skull, and neck may cause vascular injury (7).

First of all, the patients should be checked out about cardiac, vascular and blood diseases (8).

After severe traumatic brain injury, in order to control brain swelling, either medical or surgical therapies should be used. ICP should be lower than 20-25 mmHg. Simple therapeutic maneuvers, including sedation, ventilation, and head-up position, should be done as the first step. After these, more advanced medical treatment, including application of inotropes, hypertonic saline, mannitol, and hypothermia can be performed. External ventricular drainage of the cerebrospinal fluid can be useful. If these modalities fail to control ICP, we need more advanced therapy, especially barbiturate coma therapy or DC (5,9-11).

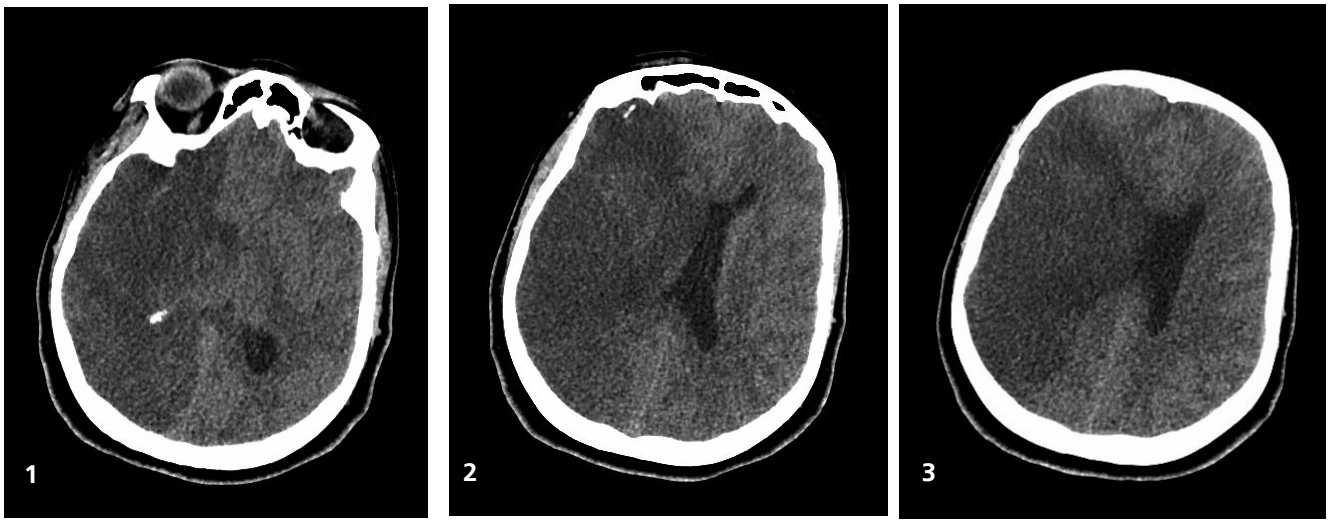
DC (removal of a part of the skull and duraplasty) is a good way to accommodate shifts of brain tissue and normalize ICP. DC is a radical and fast way of solution for the management of brain tissue shift and ICP. Surgical decompression reduces death rate and improves the functional outcome of patients. DC is mostly used as the last solution to prevent severe damage or death. In MCA infarction, DC reduces mortality by nearly 52.8%, but there is no consensus on if and when to proceed to surgery. DC may save one's life at the expense of creating a vegetative state and severe disability (10,12).

Computed tomography (CT) is useful in the detection of various cerebral pathologies, including infarction. Magnetic resonance imaging (MRI), MR angiography, CT angiography, and CT perfusion can be used for the diagnosis of cerebral infarction (2,13).

Case

A 51-year-old female was admitted to our emergency department with multiple trauma after a traffic accident. On presentation to the emergency department, she became confused, disoriented, and combative. She was noted to have left-sided hemiparesis and facial paralysis and was not obeying commands. Her systolic blood pressure was low (80 mmHg). After admitting emergency department, maintain the hemodynamic balance, preventing hypotension, hypoxia, elevation of the head by 20° to 30°, hyperosmolar therapy with mannitol or hypertonic saline treatments were started. On CT images, maxillofacial fractures, multiple rib fractures, hemothorax, and pneumothorax were seen. A chest tube was inserted urgently. After admission, the patient was taken to the intensive care unit. After three hours, the patient's GCS score was 6, and she had anisocoria but did not lose pupillary reaction to light. She had irregular breathing and pulse and underwent mechanical ventilation with controlled hyperventilation and sedation (using propofol and fentanyl). CT scans revealed right-sided hypodensity, MCA infarction and midline shift (Figures 1-3). Despite the conservative medical therapy, the ICP was over 25 mmHg. We could not take cerebral angiography or MRI, be cause of her poor general condition.

Except for the international normalized ratio (INR), other blood tests were normal, and she did not require an anticoagulant therapy, but her INR was 6. Fresh frozen plasma was given to the patient in order to reduce the INR. High-dose steroid treatment was given on the day of admission, and after 12 hours, the INR was normalized. After obtaining written informed consent from her family, operation was performed.



Figures 1-3. Preoperative computed tomography scans show the hypodense infarction area and midline shift effect

Surgery

DC consisted of a duraplasty and the creation of a large bone flap. Removal of head bone and enlargement of dura with duraplasty is DC's mainstays. In summary, a large skin incision in the shape of a question mark based at the ear is made. Frontotemporal question mark skin incision was made. A frontotemporoparietal bone flap was removed. The dura was opened, and a fascia lata graft was used for duraplasty. Dural tenting sutures were used in order to stop epidural hemorrhage. The temporal muscle and skin flap were then reapproximated and sutured.

After surgery, the patient was transferred to the intensive care unit. We did not use sedation or muscle relaxants. Her GCS score was 3 after the day of injury. A control CT scan revealed right-sided malignant MCA infarction and decreased midline shift (Figures 4-6). She died on the third day of her admission to the hospital.

Discussion

Due to high mortality and mortality rate, PTCI is destructive complication of trauma (1,2,14). Overall mortality rate is 75%. A GCS score between 3 to 5 is a high risk factor for mortality. Traumatic herniation of the brain may cause malignant high ICP which causes brain death. Despite modern medicine, CT scans, drugs, intensive care and monitoring, PTCI's mortality and morbidity rate is still high (2).

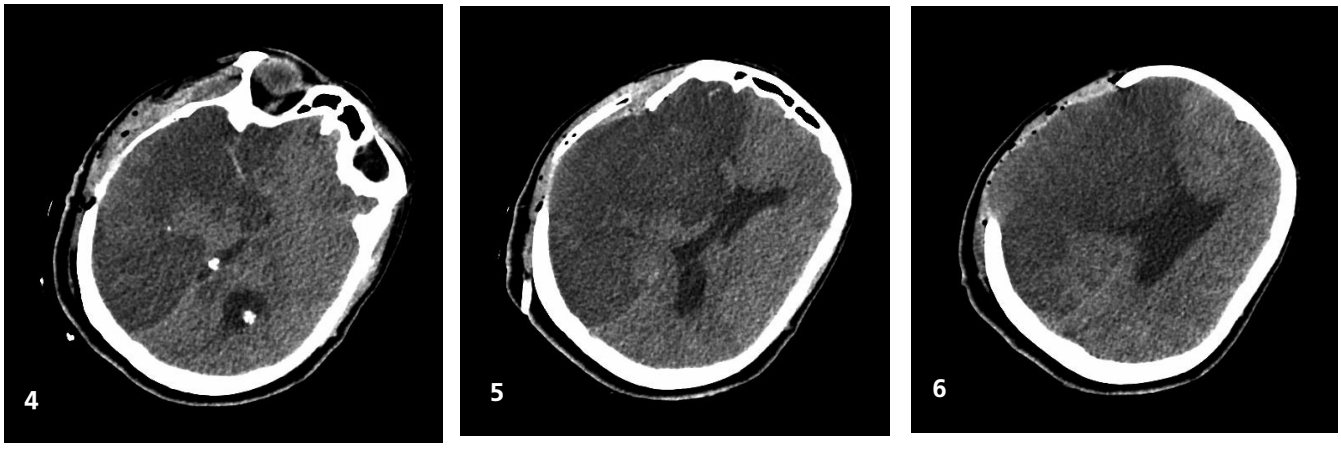
Low GCS (3-8), hypotension, and head trauma with vascular damage are main risk factors for brain shift and herniation with occurrence of fatal brain edema (2,4). The radiological factors for midline shift and postsurgical affected-to-contralateral side ratio are important clues of predicting clinical outcomes (15-18).

Regarding neurological status, the GCS score at admission and prior to surgery was 8 and 6, respectively. In addition, there was hypotension (60 mmHg-30 mmHg). On CT, it was observed that the midline shift was 10 mm. The time interval between injury and surgery was 12 hours. Hypotension is lustily associated with cerebral infarctions. Primarily, head trauma is complicated with craniofacial and limb fracture, chest trauma, and hemothorax.

Hypotension and hypoxia are significant predictors of mortality (4). If arterial perfusion is limitted with direct effect over cerebral cortex, infarction may occur. Intracranial vasospasm is also likely to occur in patients with severe injury (8). Rotational forces following trauma are secondary to the arterial wall intimal dissection, with primary thrombosis and vasospasm as less likely causes (1). Spasm may be due to direct vascular trauma or the effect of adjacent contusion and hemorrhage or may be mediated by release of a vasoactive humoral factor (6). The reported incidence of intracranial arterial spasm following moderate to severe head injury is in the range of 5% to 10% (1,19).

Massive hemispheric infarctions cause poor clinical outcome. If not treated surgically, case fatality is 80% (5). In several studies, there was a dramatic effect of surgery, with a highly significant absolute reduction of 52.8% in the death rate in the surgery group compared with the no-surgery group (9,10,15,20).

Anticoagulation using orally-administered low-molecular-weight heparin and thrombolysis using a recombinant tissue plasminogen activator have been found to be effective in adult patients when given within three hours of the thrombotic event. We could not use heparin and acetylsalicylic acid therapy because of high INR. Head trauma may cause coagulation abnormalities with poor clinical outcome. Elevated admission INR, elevated INR at 24 hours, and overall trend in INR strongly predict mortality in the trauma population (22,23). After giving the patient frozen plasma and vitamin K and, decreasing the level of INR, DC operation was started.



Figures 4-6. Postoperative computed tomography scans show the infarction area and still the midline shift

Unless medical treatments are succesful, DC is an established modality. Fastest way in order to reduce ICP, early DC and duraplasty are needed (2,12,14,21). In patients with a GCS less than 5, the risk of mortality increases. At this point, irreversible ischemic damage has no chance of recovery (2,15).

In the presence of bilaterally fixed or dilated pupils, a GSC of 3 or haemorrhagic transformation of the infarct, known coagulopathy or systemic bleeding disorder, contraindication for anesthesia, pregnancy, or contralateral ischemia, surgery should be avoided (5).

Death from brain herniation is not the only outcome. After a cerebral infarction, prolonged intensive care is needed, and worse outcomes, such as pneumonia, venous thromboembolic complications, seizures, depression, urinary tract infection, cerebral abscess, tracheostomy, gastric ulcer, etc., may occur (10).

Early diagnosis and successful management of traumatic cerebral infarction require a high degree of clinical suspicion. Close monitoring of the patient's neurologic condition is necessary for early diagnosis and succesful management of PTCI The decision about whether to perform DC or not is based on the patient's age, medical history, laboratory findings, neurological condition (GCS, clinical examination), imaging (CT or MRI) findings, in case of failure of conservative treatment and clinical deterioration. According to the literature, in the first 12 to 48 hours, early DC confers a survival benefit.

If PTCI occurs earlier with poor clinical status, this may cause high mortality and morbidity. Delayed surgery may cause irreversible ischemic infarcts. Even though we used medical and surgical treatment, the patient died. Malignant MCA infarction treatment is so difficult. In our patient, the presence of multiple trauma, including chest trauma, hemothorax, pneumothorax, craniofacial, and limb fractures, coagulation problems, and hypotension made it more difficult.

Decompressive craniectomy is widely accepted and applied as a rescue therapy in patients with refractory elevated ICP. DC provides the fastest relief by immediate reduction in raised ICP and resolution of midline shift.

Authorship Contributions

Design: A.A., U.E. Data Collection or Processing: A.A., U.E. Analysis or Interpretation: A.A., E.K., U.E., B.M.K. Literature Search: A.A., E.K. Writing: A.A., E.K., B.M.K.

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