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Article

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Sinking skin flap syndrome in a patient with giant thrombosed posterior cerebral artery aneurysm: a case report

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Abstract: Sinking skin flap syndrome is a rare complication following decompressive craniectomy. The pathogenesis is based on disturbed cerebral autoregulation and as a consequence dicreased CBF and cerebral metabolism. This results in neurologic disturbances, i. e. mental changes and focal deficits. The authors present the patient who developed the motor trephine syndrome after decompressive craniectomy following complicated giant posterior cerebral artery aneurysm clipping.

Key words: sinking skin flap, motor trephine syndrome, syndrome of the trephined, decompressive craniectomy, cranioplasty

Introduction

The sinking skin flap syndrome, or motor trephine syndrome, is a rare complication following decompressive craniectomy [2]. This pathologic entity may be explained by athmospheric pressure gradient that is even more exaggerated by CSF hypovolemia as a result of ventriculoperitoneal shunting, excessive dehydration or position change [2]. Thus athmospheric pressure gradient exceeds intracranial pressure and as far as cerebral autoregulation is disturbed, paradoxical herniation leading to neurologic deterioration, coma and death may result [3]. Urgent cranioplasty is required to reverse the neurologic deterioration [1].

Case report

58-year old male presented with right upper limb weakness and clumsiness, resulting in inability to hold a spoon and write. Brain magnetic resonance imaging (MRI) revealed partially thrombosed giant posterior cerebral artery (PCA) aneurysm with brain stem and thalamus compression and perifocal edema (Figures 1, 2). The patient was referred to neurosurgeon for the operative treatment.

The frontotemporal transcavernous approach with extradural clinoidectomy and partial orbitotomy was performed. At the time of manipulating temporary clips intraoperative rupture of the neck of the aneurysm occured. The PCA was sacrificed after making sure that distal PCA as well as

posterior communicans artery and visible perforant arteries are flow-patent.

Three hours postoperatively computed tomography (CT) perfusion showed no perfusion restriction and symmetrical cerebral blood flow (CBF) and volume (CBV). Nevertheless, the patient woke up in the intensive care unit (ICU) having severe right side hemiparesis and sensorimotor aphasia. Subsequent CT and MRI revealed ischemia in the left PCA territory.

One week later the patient's level of consciousness deteriorated, and CT scan showed cerebral edema and midline shift to the right. The patient underwent left decompressive craniectomy. His neurologic condition improved postoperatively. He was transferred from the ICU to the department of neurosurgery. Rehabilitative treatment was initiated and the patient gradually recovered. At the end of the treatment the patient was fully oriented with slight partial sensorimotor aphasia and moderate right side hemiparesis: upper extremity (UE) 4/5, lower extremity (LE) 3/5.

After 7 weeks of rehabilitation and physiotherapy program the patient started progressively deteriorating: he became drowsy and disorientated, and right side hemiparesis increased in severity: UE 3/5, LE 2/5. Head CT scan revealed concave depression of the scalp flap with underlying cerebral hemisphere and lateral ventrical compression, midline shift of 6 mm to the contralateral side and effacement of the cortical sulci (Figure 3). The postdecompressive skin flap was deeply sunken and kept being immobilised when the patient was laid down on his left side (Figure 4). The patient underwent cranioplasty with autologous bone flap on urgent basis. Postoperative CT scan showed the re-expansion of the brain to fulfill the intracranial cavity, normal sulcal patterns and restoration of the midline (Figure 5). The postoperative period was uneventful and the patient recovered to his preoperative state.

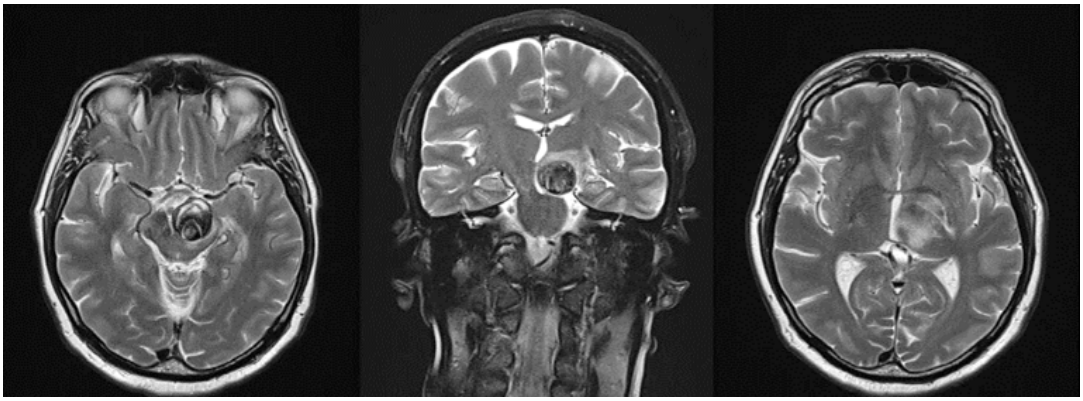

Figure 1 - Brain MRI, T2-weighted image. Partially thrombosed giant posterior cerebral artery (PCA) aneurysm with brain stem and thalamus compression and perifocal edema

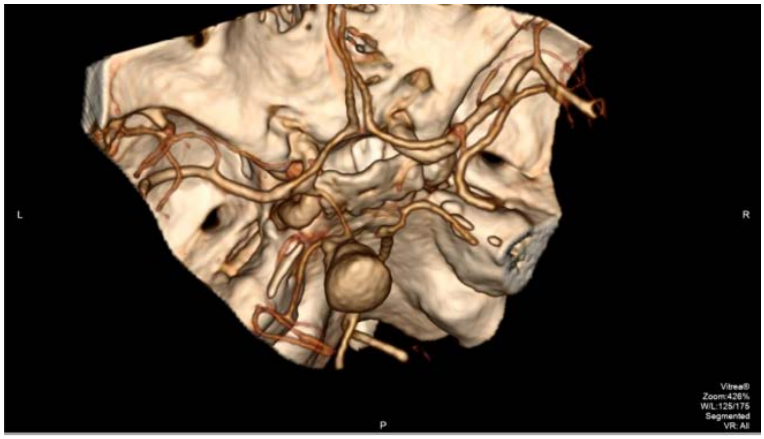

Figure 2 - Brain CT angiography. Giant posterior cerebral artery (PCA) aneurysm

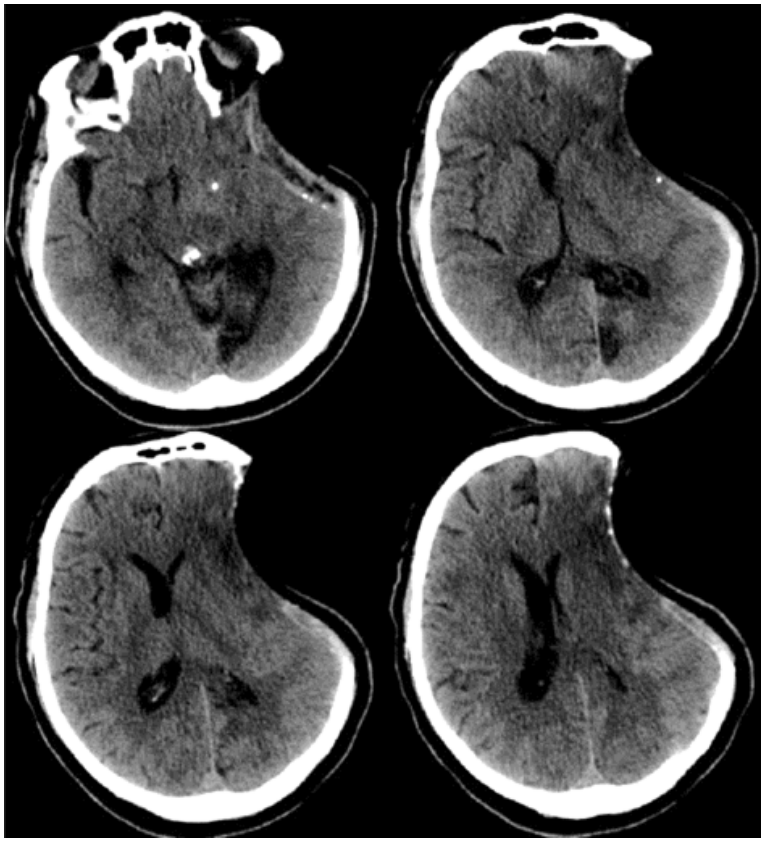

Figure 3 - CT scan. Concave depression of the scalp with underlying cerebral hemisphere and lateral ventrical compression, midline shift of 6 mm to the contralateral side and effacement of cortical sulci

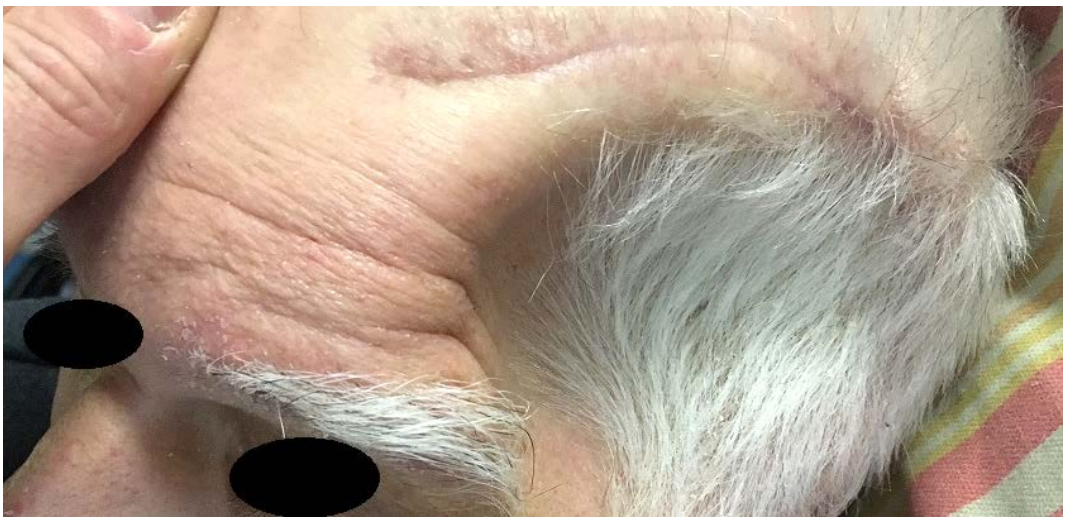

Figure 4 - Sunken skin flap

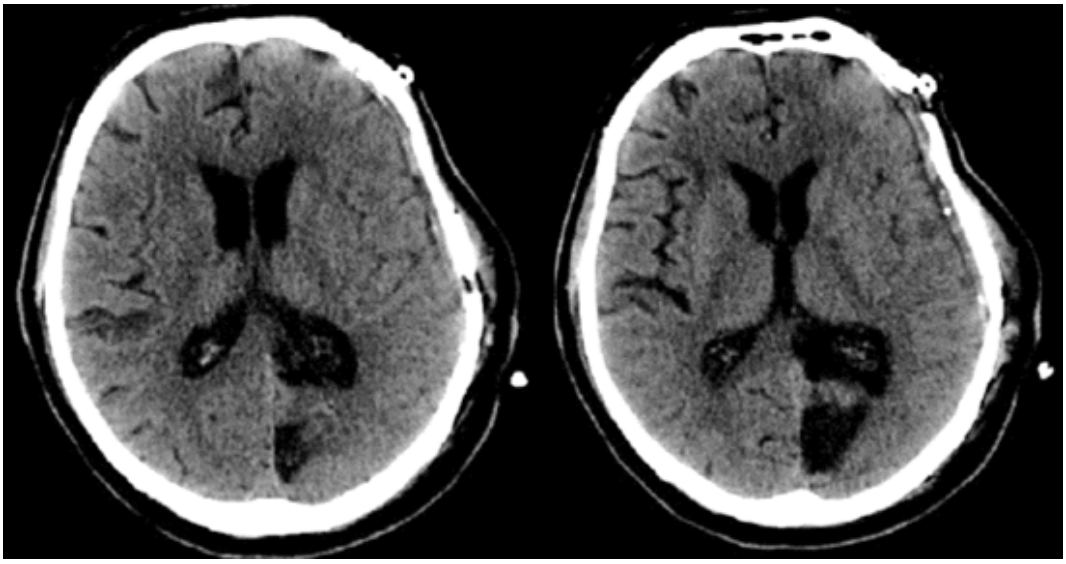

Figure 5 - Post-cranioplasty CT scan. The brain re-expanded and fully occluded epidural dead-space. Normal sulcal patterns and restoration of the midline. Chronic ischemic cerebral malacia in the left PCA territory

Discussion

Neurologic symptoms such as headaches, mental changes, language and motor disorders has been in reported in sinking skin flap syndrome, or the syndrome of the trephined [3, 4]. Motor trephine syndrome has been used by some authors to highlight delayed motor dysfunction following craniectomy [2]. Often the symptoms arise in upright position and diminishes in supine position [2, 4]. The

causative factors are local cortical compression along with the local ischemia and CSF circulation disturbances [2].

A variety of issues arrise following decompressive craniectomy. Cerebrospinal fluid dynamics alterations may result in hydrocephalus, subdural or subgaleal fluid collections [1]. Cerebral perfusion pressuse and blood flow is reduced. The impaired venous return and obliteration of the subarachnoidal space due to the direct atmospheric pressure on the brain may be the cause of It [1]. The atmospheric pressure is resisted by two mechanisms: the elastance of the brain that resists deformation and ventricular CSF that keeps the brain expanded. The parenchymal injury with tissue loss is a predictive factor for the development of the sinking skin flap syndrome [2]. Prolonged dehydration and upright position often precipitates this condition [4].

Paradoxical herniation is a potentially devastating event. The effect of atmospheric pressure along with gravitational forces lead to herniation syndrome [1]. Patients are more susceptible to this syndrome after the CSF drainage procedures, i. e. external ventriculostomy, ventriculoperitoneal (VP) shunting and lumbar drainage [1]. The CSF hypovolemia along with atmospheric pressure potentiate the brain being pushed down through the incisural notch [1]. The traditional treament for brain herniation such as mannitol, CSF drainage, and hyperventilation reduce intracranial contents and exacerbate paradoxical herniation due to increasing pressure gradient across the craniectomy defect [1]. Paradoxic measures are applied to counteract the atmospheric pressure, i.e. Trendelenburg position, excessive hydration, closing the CSF drainage (clamping or re-programming the VP shunt) and discontinuation of the hyperosmolar treatment [1]. The definitive treatment is performing cranioplasty on urgent basis [1].

If clinical and neurologic deterioration occurs after initial improvement, the skin flap over the craniectomy defect is concave and CT scans is characteristic of the syndrome of the trephined [2], early cranioplasty must be considered once other causes are excluded.

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