Laceration within the mesencephalon and pons after traumatic brain injury: a case report.

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Citation

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Abstract

A 29-year-old male made unsuccessful suicide by hanging. Instead he fell causing brain injury. After cranial surgery and rehabilitation the patient was re-admitted with vertical gaze paresis and unsteady gait. MRI revealed a cystic formation in the mesencephalon. Due to neurological deterioration, growing of the cystic formation within mesencephalon, and signs of posttraumatic hydrocephalus, a ventriculo-peritoneal shunt-system was implanted and a dilatation of the aqueduct was performed. Despite these measures there were ongoing neurological deterioration and increasing of the cystic formation.

INTRODUCTION

The appearance of hydrocephalus as a post-traumatic sequel due to disturbances in circulation of cerebro-spinal fluid or brain atrophy is well known. The number of reports of post-traumatic hydrocephalus has increased since the first publication of such a case in 1914 by Dandy [1]. The incidence of symptomatic post-traumatic hydrocephalus is reported from 1% - 29% [23]. To our knowledge, there is no report of posttraumatic hydrocephalus combined with ongoing rhexis mesencephali.

CASE REPORT

A 29 year old patient was admitted in September 2006 after having attempted suicide. Due to schizophrenia which caused demons of the mind the patient put a rope around his neck, fixed it in a room and jumped out of a window from the 2nd floor. The rope tore and the patient fell hardly on the ground. The emergency physician found him drowsy, shouting and able to move all extremities. After sedation, intubation, artificial ventilation and recovery, the patient was admitted to our emergency department No problems in relation to circulation and oxygenation occurred during the transport.

Examination in the emergency room revealed pupils in an isochoric status with prompt reaction to light. There were no movements of extremities or eyelids as a reaction to pain, and no corneal reflex. The patient showed several grazes and lacerations, particularly to head and neck. Computed tomography (CT) of neurocranium, thorax, abdomen and extremities displayed lung-contusions (segment 6), fractures of the ribs 8 to 11 left with emphysema, a left-sided ventral pneumothorax, brain edema in right hemisphere with compression of the right lateral ventricle, galea-haematoma, blasting of the sutura coronalis and frontalis on the left side with fracture of the os parietale on both sides. As a result of these findings, a decompressive craniectomy was performed and the epidural and subdural haematoma were removed (Fig. 1).

The patient was transferred into a psychiatric and rehabilitation hospital. At that time, he was disorientated, but able to communicate without any hemi-symptoms. One month later, an autologous cranioplasty was performed. After the surgery, he has been transferred to the psychiatric and rehabilitation hospital.

Six months later the patient was presented again with soaring gait ataxia, double vision and punctual loss of sight in the left eye. At neurological examination the patient presented anisochourus pupils – the left one larger than right one - with slight elliptic pupil on the left side. Motor function, coordination, and power were within the normal range, excluding the previously mentioned gait ataxia and psycho-motoric deceleration. A new Magnet-Resonance-Imaging (MRI) of the neurocranium displayed a new formation (0.5 x 0.3 cm) inside the left pons close to the aquaeduct (Fig. 1). After drainage of 30ml cerebrospinal fluid the patient showed an improvement for three days. An ophthalmologic examination before lumbal punction did not confirm an atrophy of N. opticus or a retinal detachment, but

a left sided ophthalmoplegia was revealed.

Investigation of cerebrospinal fluid circulation revealed a stenosis of the aqueduct causing a hydrocephalus. Seven months after the traumatic brain injury, a cystic formation and progressing hydrocephalus forced us to implant a ventricular-peritoneal shunt-system from right lateral ventricle. A stabilisation of the neurological status with a slight improvement of the gait ataxia without any further deterioration of the ophthalmoplegia followed

However, further MRI investigation revealed a growth of the cystic formation with connection to the aqueduct and infiltration from the thalamus, mesencephalon to the fourth ventricle and in later from the pyramidal tract to the left lateral ventricle (Fig. 2) in first four weeks after the shunt-implantation. A special display of cerebrospinal fluid flow (CISS-MRI) showed a new jet from the fourth ventricle into the cyst deformation within mesencephalus cerebri, during the obstruction of the aqueduct. As a consequence of these findings we performed an endoscopic dilatation of the aquaeduct. A follow-up MRI five days post-operation showed a widening of the aqueduct, but also ongoing rhexis mesencephali. We therefore programmed a reduced pressure level of the ventricular-shunt system. The patient was transferred to the well-known psychiatric and rehabilitation hospital for further monitoring and therapy.

Six weeks later, the follow-up examination showed a slight reduction in gait ataxia without ophthalmoplegia. The patients' psycho-motor function had decelerated which was maybe a result of antipsychotic medication. Coordinative disorders were still detectable. One year after the trauma, a new MRI of the neurocranium showed an increased cyst formation and connection to the left lateral ventricle (Fig. 2).

The patient was able to talk and walk a few steps on his own, but he was mostly depending on assistance in everyday life. Coordinative disorders were regressive.

Figure 1

Figure 1: Initial computed tomography with acute subdural haematoma and midline shift due to brain edema (left). MRI five months after trauma (DWI): Midbrain lesion within the mesencephalon; only displayable in diffusion weighted imaging (right).

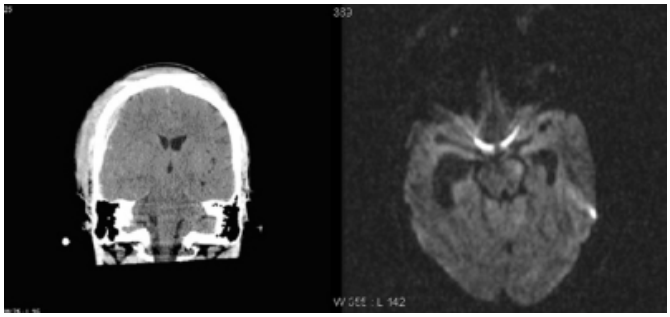
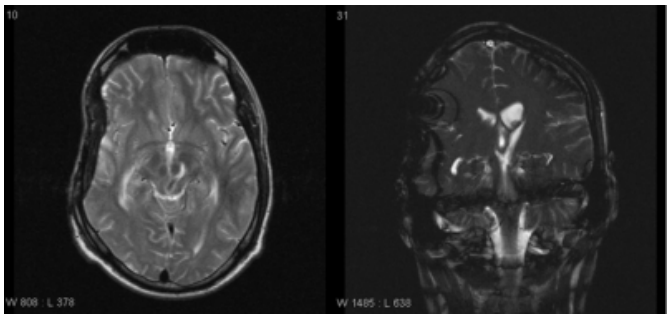


Figure 2

Figure 2: MRI seven months after trauma (T2): Laceration within the mesencephalon; By- finding the cranioplastic bone in resorption (left). MRI one year after trauma (T2): the cystic formation connected to the left lateral ventricle (right).



DISCUSSION

The diagnosis, incidence, mechanism, and pathophysiology of post-traumatic hydrocephalus have been reported and investigated in various reports and studies [2,3,4,5,6,7,8,9,10,11]. However, the majority of reports recommends to differ between post-traumatic atrophy-related ventriculomegaly and active, symptomatic ventricular dilatation. Treatment with endoscopic surgery and ventriculo-peritoneal shunting or ventriculo-atrial shunting depends on causation [1,2].

Czosnyka et al. reported a changing pattern of cerebro-spinal fluid circulation after a cranioplasty resulting from a previous decompressive craniectomy for refractory intracranial hypertension after traumatic brain injury. They state that the resistance to cerebro-spinal fluid outflow after craniectomy decreases twofold and brain compliance increases. After development of an acute hydrocephalus, craniectomy allows compensation of cerebro-spinal fluid circulation in the early stages. This process may be reversed

after cranioplasty [4]. Another possible explanation was made by Hochwald et al. A large craniectomy may facilitate irreversible ventricular enlargement over weeks or months. After cranioplasty the expanded ventricles may obstruct the lumen of the cortical subarachnoid space - via the cerebral mantle -, and increase the resistance to cerebro-spinal fluid outflow [13].

These may be the causes for hydrocephalus. But the mentioned hypothesis may not explain the origin and expansion of the lesion and the rhexis inside the mesencephalon. Retrospective investigation of computed tomography and MRI revealed a lesion inside the mesencephalon, which could not be detected in the initial computed tomography. Assumedly, a lesion inside the midbrain occurred due to the trauma mechanism of combined traumatic brain injury caused by the fall and hypoxia caused by cervical strangulation. Maybe the traumatic brain injury only may have caused a lesion as contusion like on the right side (Fig. 1), but the lesion on the left side remained occult. The lesion lacerated and formed the rhexis mesencephali due to tissue transformation in a light cell structure and the close connection to the third ventricle and his pulsation. That grew due to pulsation of cerebro-spinal fluid and even more due to the stenosis of the aqueduct.

We cannot evidence the hypothesis of structural changes, but this can explain the morphological findings in magnet-resonance tomography and their absence in computed tomography.

We know from former studies that post-traumatic lesion in the brain and particularly in the midbrain might appear differently in magnet-resonance imaging. We have furthermore knowledge of the possibility of post-traumatic changes of cerebro-spinal fluid volume and total brain volume due to changes and degeneration of white matter after traumatic brain injury [1415]. These are indications for tissue transformation after traumatic brain injury.

We cannot detect a regression of rhexis mesencephali after surgical treatment with shunt system and aqueduct dilatation. Neurological symptoms remained stable.

Even though there are numerous publications about post-traumatic hydrocephalus, the report of rhexis mesencephalon is new. If post-traumatic tissue lesions, particularly in the midbrain, appear close to the cerebro-spinal fluid pathways, and the disease course reveals a post-traumatic hydrocephalus, particularly in combination with an aquaeduct stenosis, a rhexis mesencephali may occur.

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