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TRAUMATIC BRAINSTEM INJURY DUE TO BLUNT FORCE TRAUMA: AN AUTOPSY CASE REPORT

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ABSTRAK

Trauma adalah penyebab utama morbiditas dan mortalitas pada pasien di bawah usia 35 tahun dan menjadi penyebab utama kematian keenam di seluruh dunia, akan tetapi cedera batang otak primer (Primary Traumatic Brainstem Injury) akibat kekerasan tumpul pada kepala sangat jarang ditemukan. Otopsi forensik pada seorang wanita berusia 35 tahun yang ditemukan tewas di sebuah apartemen dengan kedua tangan dan kaki diikat dan mulut ditutup dengan lakban. Pemeriksaan luar menunjukkan korban mengalami cedera akibat kekerasan tumpul pada kepala, wajah, dan keempat anggota gerak. Pada pemeriksaan dalam (otopsi) ditemukan adanya resapan darah pada seluruh pada kulit kepala bagian dalam, seluruh otot-otot temporal dan otot-otot leher bagian dalam. Selain itu ditemukan adanya perdarahanan subarachnoid, perdarahan intraventrikular, perdarahan dan memar pada batang otak, disertai dengan tanda-tanda asfiksia pada seluruh organ dalam.. Kekerasan tumpul pada daerah oksipital kepala tidak hanya dapat menyebabkan cedera otak besar dan kecil, namun juga dapat menyebabkan trauma langsung pada batang otak akibat terjadinya hiperfleksi akut pada leher. Perdarahan dan kontusio pada batang otak dapat menyebabkan perubahan pada regulasi sistem pernapasan, bergantung pada lokasi lesi. Penyebab kematian pada kasus ini disimpulkan sebagai kekerasan tumpul pada kepala yang menyebabkan perdarahan dan memar pada batang otak, dengan mekanisme kematian adalah asfiksia.

Kata kunci : kekerasan tumpul, injury, asphyxia

ABSTRACT

Trauma is the leading cause of morbidity and mortality in patients under 35-years of age and the sixth leading cause of death worldwide, but primary traumatic brainstem injury after blunt force trauma to the head is a rare event. A forensic autopsy was performed on a 35 years old female who was found dead in an apartment building with both her hands and feet tied together and mouth closed with duct tape. External examination showed the victim suffered from blunt force trauma to the head, face, both arms and legs. Internal examination of the body showed hematomas on the scalp, temporal muscles and deep neck muscles and organs, subarachnoid, intraventricular and brainstem haemorrhage and contusion, along with signs of asphyxiation. Trauma to the occipital region of the head not only can cause traumatic brain injury but can also cause trauma to the brainstem as a result from acute hyperflexion of the neck. Haemorrhages and contusions of the brainstem can lead to changes in the respiratory system, depending on the location of the lesions. Cause of death was concluded to be force trauma to the occipital region causing primary brainstem injury with the mechanism of death of asphyxia.

Key words: Blunt force trauma, traumatic brainstem injury, asphyxia

INTRODUCTION

Trauma is the leading cause of morbidity and mortality in patients under 35-years of age and the sixth leading cause of death worldwide. Severity is related to both the mechanism of injury as well as the underlying comorbidities of the patient. Traumatic brain injury can result from a closed head injury or a penetrating head injury. The damage from traumatic brain injury can either be focal or diffuse. Diffused trauma to the brain is frequently associated with concussion, diffuse axonal injury, while focal injury of brain consists of epidural hematoma, subdural hematoma, and subarachnoid haemorrhage. But, brainstem haemorrhage after blunt head injury is a rare event. The majority of these cases are categorized as secondary haemorrhage in the brainstem which resulted from an increase of intracranial pressure and brain herniation. This is commonly seen as a secondary event in cases such as extra or subdural haemorrhage or intracerebral bleeding following head injury. The second type of brainstem haemorrhage encountered in cases of head injury is a primary damage caused directly by trauma due to physical effect of traumatic forces damaging cells, axons and blood vessels. Primary brainstem haemorrhage is divided into 2 distinct types, which are diffuse and focal.[1,2,3]

CASE REPORT

A forensic autopsy was performed on a 35 years old female who was found dead in an apartment building faced down on the bed with both her hands and feet bound together and mouth closed with duct tape. External examination showed the victim suffered from blunt force trauma leading to bruising and swelling in the occipital region, dislocation of the right wrist, several small lacerations on the ears and lips, several small abrasions and bruises on the face, body, arms and legs. No signs of trauma were seen superficially around the neck area, but the autopsy revealed haemorrhage in the deep neck muscles (*musculus sternohyoid sinistra* and *musculus thyrohyoid sinistra*), esophagus and thyroid gland. Signs of asphyxia were seen clearly throughout the internal organs.

Autopsy findings in the head showed subgaleal hematomas, haemorrhages in the left and right temporal muscles, small but several areas of areas of subarachnoid bleedings in the parietal-occipital region and also frontal region. The brain weighed one thousand two hundred grams and showed flatted gyri and filled sulci, a faded distinction between the white and grey matter, and showed no signs of herniations. Vasodilation of blood vessels were seen throughout the brain. The dissection of the cerebrum showed minimal amount of bleeding inside the lateral ventricles. The cerebellum showed no signs of hernia. Petechial haemorrhages were seen on the floor of the fourth ventricle and there was also a small contusion, five millimeters by three millimeters, on the posterior side of the medulla oblongata (transverse dissection).

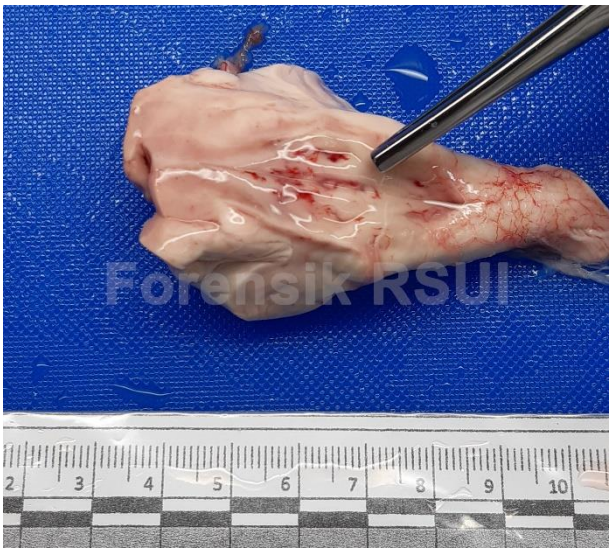

Figure 1. Petechial haemorrhages on the floor of the fourth ventricle.

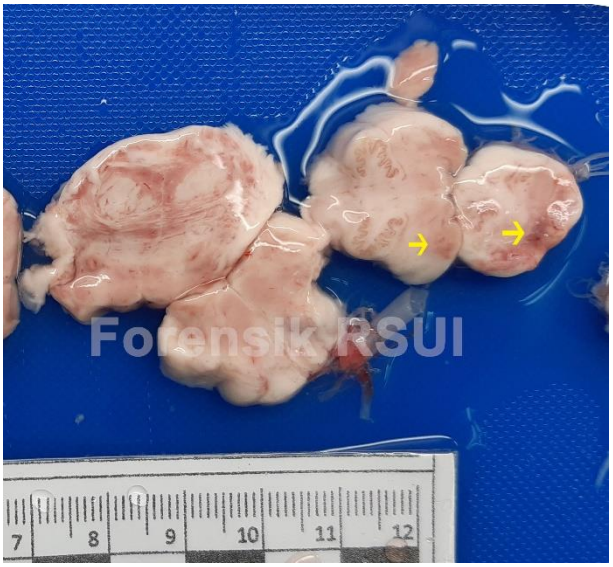

Figure 2. Transverse dissection of the medulla showed constusions on the posterior side (arrows).

DISCUSSION

Traumatic brainstem haemorrhage is an uncommon finding in patients with blunt head injury. Traumatic brainstem haemorrhage typically results in coma, decerebrate posturing, and autonomic nervous system dysfunction. There are two known types of traumatic brainstem haemorrhage, primary and secondary. Primary type is the result of direct mechanical distortion of the brain stem. Secondary type results from diffuse cerebral edema, hypoxia, posttraumatic vasospasm, and transtentorial herniation.[2]

Primary brainstem haemorrhages are usually associated with occipital impacts and the victim is often unconscious from the time of the injury, as opposed to the lucid interval and gradual decline of those who suffer secondary stem lesions because of a developing cerebral oedema or

space-occupying haematoma. It is commonly believed that brainstem haemorrhage is associated with the worst prognosis. Previous studies have indicated mortality rates of 83%, with up to one-half of the surviving patients remained in a persistent vegetative state. Previous studies have also shown that the most frequent site of haemorrhages, were the ventral rostral midbrain adjacent to the interpeduncular cistern. Disruption of axonal tracts in the brainstem injured patients occurs as part of the widespread white matter damage. This lesion often coexists with other brain lesions such as acute epidural hematoma, acute subdural hematoma, and haemorrhagic contusion.[2,4]

This autopsy cased helps understand the cause and effects of blunt force trauma to the occipital region of the head. Though blotches of subarachnoid haemorrhages in the cerebrum, minimal haemorrhage inside the lateral ventricles, vasodilation throughout the the brain, and signs of a slightly oedematous brain was seen during the autopsy, there were no gross signs of herniations found and the brain weighed was still in normal range. Trauma towards the brainstem in this case is categorized into the focal primary brainstem injury with petechial haemorrhages on the floor of the fourth ventricle and contusions in the medullary.

The mechanism of injury can be hypothesized considering the dynamics of trauma. Brainstem haemorrhage can be caused by primary lesion resulting from either rotational forces, or from transient acceleration-deceleration forces that cause contusion against the tentorium, or secondary to the brainstem compression. However, it is said that the acceleration-deceleration injury model in a sagittal plane is the most frequent cause of traumatic brainstem hemorrhage.[2] Nervous and/or vascular compression against the tentorial notch mostly occurs at its lateral portion due to the shortest distance to the brain stem and near the level of ponto-mesencephalic junction. They supposedly result from shearing strains at the craniocervical junction due to fixation by the edge of the tentorium and odontoid peg which played any part on medulla. The primary damage to the brain stem occurs mostly in the tegmentum of brain, more frequently than those lesions in cerebral peduncles or basis pontis.[4,5]

Frequent site of haemorrhages or contusion site of the brain stem are the dorsal side of midbrain, cranial nerves, whole brain stem, cerebellum, combined with upper cervical spinal injuries. In terms of cranial impact site, literature reviews addressed there is an association between occipital blows and primary cerebellar and brain stem lesions. primary lesions are present in an undistorted brain stem and they are lateral, tegmental, and often unilateral microhemorrhages. Impacts to the neck, although few in numbers, have been known to give primary brain stem lesions.[5]

Autopsy findings in this case showed haemorrhages in the deep neck muscles, esophagus and thyroid gland without any signs of superficial wounds on the neck and fractures. Not only were there petechial haemorrhage on dorsal brainstem (floor of fourth ventricle) but contusions were seen on the dorsal area of the medulla.

Injury of lower brain stem could be caused by hyperextension of the cervical vertebrae or reciprocal actions of fracture of the clivus and the direct effect on the brain stem by acceleration or rotational forces.[5] Biomechanics of brainstem contusions are damage by direct impact, flexion and distortion and vascular involvement or hyperextension. A study conducted by Cooper et al. showed that acute flexion is the main recognized factor for brainstem contusion, contrary to hyperextension. Blunt force trauma to the back of the head can result to an acute hyperflexion of the neck.[6,7]

Usually, brainstem contusions are associated with a decrease of consciousness (low Glasgow Coma Scale) and systemic changes like pulmonary complications and cardiological changes, depending on the focal location of the lesion. Automatic, unconscious cycle of breathing is controlled by three pair of respiratory centers situated in the reticular formation of the medulla

oblongata and the pons. Control of ventilation depends on a brainstem neuronal network that controls activity of the motor neurons innervating the respiratory muscles. This network includes the pontine respiratory group and the dorsal and ventral respiratory groups in the medulla.[7,8]

The dorsal respiratory group (DRG) has the most fundamental role in the control of respiration, initiating inspiration (inhalation). It is composed mainly of inspiratory neurons located bilaterally in the medulla, the DRG controls the basic rhythm of breathing by triggering inspiratory impulses. The DRG is a collection of neurons forming an elongated mass that extends most of the length of the dorsal medulla. They are near to the central canal of the spinal cord, and just behind the ventral group. They set and maintain the rate of respiration.[9,10]

Most of the neurons are located in the nucleus of the solitary tract. Other important neurons are found in the adjacent areas including the reticular substance of the medulla. The solitary nucleus is the end-point for sensory information arriving from the pontine respiratory group, and from two cranial nerves – the vagus nerve, and the glossopharyngeal nerve. The solitary nucleus sends signals to the respiratory center from peripheral chemoreceptors, baroreceptors, and other types of receptors in the lungs in particular the stretch receptors. The dorsal respiratory group is thus seen as an integrating center that gives output to the ventral respiratory group to modify the breathing rhythm.[9,10]

These neurons send impulses to the motor nerves of diaphragm and external intercostal muscles. DRG nerves extend into the Ventral Repiratory Group (VRG), but the VRG neurons do not extend into the DRG. Vagus and glossopharyngeal nerves bring sensory impulses to the DRG from the lungs, airways, peripheral chemoreceptors, and joint proprioceptors.[12,13,14]

Chemoreceptors detect the levels of carbon dioxide in the blood by monitoring the concentrations of hydrogen ions in the blood. Chemoreceptor regulation of breathing is a form of negative feedback. The main chemoreceptors involved in respiratory feedback are: Central chemoreceptors and Perpheral chemoreceptors. Central chemoreceptors are located on the ventrolateral surface of medulla oblongata and detect changes in the pH of spinal fluid. They can be desensitized over time from chronic hypoxia (oxygen deficiency) and increased carbon dioxide. While peripheral chemoreceptors include the aortic body, which detects changes in blood oxygen and carbon dioxide, but not pH, and the carotid body which detects all three. They do not desensitize, and have less of an impact on the respiratory rate compared to the central chemoreceptors. Negative feedback responses have three main components: the sensor, the integrating sensor, and the effector. For the respiratory rate, the chemoreceptors are the sensors for blood pH, the medulla and pons form the integrating center, and the respiratory muscles are the effector. Chemoreceptor feedback also adjusts for oxygen levels to prevent hypoxia, though only the peripheral chemoreceptors sense oxygen levels. In cases where oxygen intake is too low, feedback increases ventilation to increase oxygen intake. Hence trauma to the area will modify the breathing pattern. This is consistent with autopsy findings of signs of asphyxia (trias asphyxia) found throughout the internal organs. [12,13,14]

CONCLUSION

Traumatic brainstem injury is an uncommon finding in victims with blunt head injury but has a high mortality rate. Cause of death concluded in this autopsy case report was blunt force trauma to the occipital region causing primary brainstem injury with the mechanism of death of asphyxia.

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