Traumatic Isolated Brain Stem Hematoma: A Case Report Presenting with Hemiparesis

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Abstract

Isolated brain stem hemorrhages are rare but have a high mortality rate after blunt head trauma. A 56-year-old male admitted to the emergency room with weakness on the right side of his body, one day after the blunt head trauma. Right hemiparesis which is near to plegia (lower extremity motor weakness level was higher than upper extremity) was found on his neurological examination. T2-weighted images and susceptibility weighted images (SWI) of the magnetic resonance revealed heterogenous hyperintense lesion with hemorrhage at the left side of the pons. The patient was managed with conservative treatment; at three-month follow-up, near total recovery was observed on his neurological examination. As a conclusion, it could be said that brain stem contusion with hemorrhage could be kept in mind in patient with hemiparesis if the neurological deficit of the patient cannot be explained by supratentorial or spinal cord lesions.

Keywords: Brain stem, contusion, hemiparesis, hemorrhage

INTRODUCTION

Isolated brain stem hemorrhages which have a high mortality rate (67–83% of the cases) is rarely seen after blunt head trauma (0.75–3.6% of the cases) (1–7). Previous studies showed that half of the remainder of the patients with brain stem hemorrhage could live in the vegetative state (6).

In this case report, a patient with isolated brain stem hemorrhage developed after blunt head trauma was presented.

CASE PRESENTATION

56-year-old male patient with weakness on his right side who had a solid blow to his occipital region one day ago was admitted to the emergency room. At his admission, Glasgow Coma Scale (GCS) score was found 15/15 points and his pupils were equal in size and reactive to light on his neurological examination. Of 2/5 weakness in his upper limb and 1/5 weakness in his lower limb was detected. The remaining of the neurological examination did not reveal any abnormal finding. Because of no pathological finding on cerebral computerized tomography (CT) images, magnetic resonance (MR) scan was performed. In the left side of the pons, a heterogenous hyperintense lesion with a hemorrhagic spot located in its center was detected on T1 and T2-weighted images (WI), and susceptibility weighted images (SWI) of MR, but MR spectroscopy, perfusion imaging, and MR-tractography demonstrated no finding that suggest neoplasia (Figure 1–3). According to these radiological findings, conservative treatment consisted of anti-edema drug administration was considered rather than surgical intervention. One week after trauma, hemiparesis of the patient began to improve significantly (of 4/5 weakness in upper limb and 3/5 weakness in lower limb). At third month follow-up, near total recovery was observed in his neurological examination (of 5/5 weakness in upper limb and 4/5 weakness in lower limb).

A written consent form has been signed informing the patient that his medical data will be sent for publication.
Figure 1. Axial T1 (A), T2-weighted images (B) and susceptibility weighted images (C) MR scan showed heterogenous hyperintense lesion with hemorrhage at the left site of the pons.

Figure 2. MR-spectroscopy, and perfusion images excluded the presence of any neoplasia.

Figure 3. MR-tractography showed continuity of the axonal transmission.
DISCUSSION

Traumatic brain stem hemorrhages could be classified as primary and secondary. Secondary brain stem hemorrhages are usually related to the venous stasis in the venous branches of the pons. This venous stasis usually developed by tentorial herniation secondary to increasing of the intracranial pressure (8). On the other hand, the mechanism of the primary traumatic brain stem hemorrhages has been summarized by Hashimo et al. by four different mechanisms: 1) diffuse axonal injury during acceleration/deceleration, 2) tentorium shear strain at the midbrain as a result of the damage caused by the cutting effect from the rigid nature of the tentorium, 3) injury of the neurovascular structures due to edge of tentorium, and 4) hyperextension of the cervical vertebrae injuring the lower brain stem (9). However, it is uncertain to define the exact mechanism because of conflicting neuropathological evidences in brain stem hemorrhage. Some authors suggested that acceleration/deceleration injury is the most common reason of traumatic brain stem hemorrhages (10). Others considered the hyperextension at the rostrocaudal axis as the basis of the traumatic brain injury (4, 9, 11). Since there was no etiology like supratentorial lesions or brain edema that could increase the intracranial pressure in our patient, we evaluated the pontin lesion of our patient as primary traumatic brain stem contusion and hemorrhage. We thought that the left side pontin contusion could be produced by edge of tentorium which injures the neurovascular structures. Therefore, we accepted that acceleration/deceleration injury developed by blowing to the occipital region with a solid object was considered to be the etiological agent.

The diagnostic evaluation of the traumatic brain stem hemorrhage is quite challenging. Cerebral CT gives false positive results in 1/3 of patients because of irregular bone formations of the skull base (12). So, brain MR is still the best diagnostic modality in diagnosis of these patients (13). In our patient, although the CT scan could not demonstrate the brain stem injury, T1 and T2 WI and SWI MR scan easily revealed a heterogenous hyperintense lesion with punctate hemorrhagic focus at the center of the pons. Furthermore, MR-spectroscopy, and perfusion images excluded the presence of any neoplasia, while MR-tractography showed continuity of the axonal transmission. On the other hand, it has been demonstrated that brain stem contusion with hemorrhage is generally associated with poor prognosis (1–3). Our patient admitted only with the complaints of right hemiparesis, and after the conservative treatment, his hemiparesis was healed almost completely at long term follow-up. We believe that the favorable outcome of our patient depends on the good initial neurological state at the time of admission, and the absence of additional brain injury.

CONCLUSION

In conclusion, in a patient with hemiparesis developed following a blunt head trauma, detailed investigation of brain stem contusion and/or hemorrhage could be recommended if the neurological loss could not be explained by supratentorial or spinal cord lesions.