International Journal of Radiology

Online Submissions: http://www.ghrnet.org/index./ijr/doi:10.17554/j.issn.2313-3406.2015.02.24

Int Journal of Radiology 2015 December 2(2): 53-55 ISSN 2313-3406(print)

EDITORIAL

Diagnostic Imaging of Primary Brainstem Injury Using Magnetic Resonance Imaging

Yasushi Shibata

Yasushi Shibata, Department of Neurosurgery, Mito Medical Center, University of Tsukuba, Mito Kyodo General Hospital, Miyamachi 3-2-7, Mito Ibaraki, 3100015, Japan

Correspondence to: Yasushi Shibata, MD, PhD, Department of Neurosurgery, Mito Medical Center, University of Tsukuba, Mito Kyodo General Hospital, Miyamachi 3-2-7, Mito Ibaraki, 3100015, Japan.

Email: yshibata@md.tsukuba.ac.jp Telephone: +81-29231-2371

Received: July 25, 2015 Revised: September 28, 2015

Accepted: September 30, 2015 Published online: December 10, 2015

ABSTRACT

Traumatic primary brainstem injury is brainstem injury caused by mechanical head trauma. The author herein reviews the recent clinical studies of MRI of primary brainstem injury. The importance of early MRI examination appears to be a consensus. Our study demonstrated that small superficial lesion may be detected only a few days after injury. The acute phase within a few days after a head injury may show an unstable vital sign, thus intensive monitoring of the vital sign and manual or mechanical ventilation during a MRI examination should be employed. Direct brainstem contusion has a good prognosis compared with brainstem injury associated with diffuse axonal injury (DAI). Small superficial lesions are detected only within a few days after a head injury. Recent studies of MRI in patients with head injuries could not determine the pathology. However, the differentiation of the pathophysiology must be clinically important because the prognoses are different and distinguishable using only acute stage MRI.

© 2015 ACT. All rights reserved.

Key words: Brain stem; Trauma; MRI; DAI

Shibata Y. Diagnostic Imaging of Primary Brainstem Injury Using Magnetic Resonance Imaging. *International Journal of Radiology* 2015; 2(2): 53-55 Available from: URL: http://www.ghrnet.org/index.

php/ijr/article/view/1329

EDITORIAL

Traumatic primary brain stem injury is brainstem injury caused by mechanical head trauma. Before the development of diagnostic imaging, a pathological examination has been the nearly only diagnostic method of primary brainstem injury. Because magnetic resonance imaging (MRI) has a high resolution and CT demonstrates much artifact around the brainstem, MRI became a powerful tool for investigating primary brainstem injury. There are some types of primary brainstem injury. The author herein reviews the recent clinical studies of MRI of primary brainstem injury.

Gentry et al classified traumatic brainstem injuries into primary and secondary lesions^[1] (Table 1). Primary lesions included direct superficial contusion, diffuse axonal injury (DAI), primary petechial hemorrhage, and pontomedullary rent. Secondary lesions included Duret hemorrhage, brainstem infarction, transtentorial herniation, and hypoxic/ischemic injury. Previous pathological studies showed that most types of primary brainstem injury was accompanied with DAI and only a few patients showed direct brainstem injury without DAI^[2,3]. Because DAI has a poor prognosis and high mortality, most pathological specimens showed findings of DAI. On the other hand, direct brainstem contusion has a better prognosis than brainstem injury associated with DAI, thus, the pathological investigation of this injury has been limited. The clinical introduction of MRI has brought much progress toward diagnosing brainstem injury in living human in the acute stage. Direct superficial brainstem contusion is occasionally observed at the lateral brainstem near the free tentorial edge, however, this direct contusion has not been extensively investigated.

Firsching *et al* examined the MRI findings of 61 consecutive patients within 7 days after severe head injury^[4]. Bilateral pontine lesions proved to be 100% fatal, and most patients showed concomitant lesions other than brainstem injury. In these patients, an increased intracranial pressure did not account for this high mortality, thus the most probable cause of death is DAI. Patients with no brainstem injury showed a very low mortality. They also reported isolated primary brainstem contusion.

Table 1 Classification of Traumatic Brain Stem Lesions ^[1] .	
Lesion	Injury
Primary	Primary direct superficial laceration or contusion
	Diffuse axonal (shear) injury
	Multiple primary petechial hemorrhages
	Pontomedullary rent or separation
Secondary	Secondary (Duret) hemorrhage
	Focal brain stem infarcts from vascular compromise
	Distortion, compression, and rotation of brain stem
	Pressure necrosis from transtentorial herniation
	Diffuse hypoxic/anoxic/ischemic injury

Kampfl *et al* reported the MRI findings of 42 patients in a posttraumatic persistent vegetative state^[5,6]. MRI was examined between 42 to 56 days after the injury. Most patients showed injury to the corpus callosum, dorsolateral rostral midbrain and supratentorial white matter, which is evidence of DAI.

We evaluated the MRI findings from 17 patients with primary brainstem injury in the acute stage, mostly within 2 days after injury^[7]. Continuous monitoring of the electrocardiograph, blood pressure, oxygen saturation and manual or mechanical ventilation was continued during the MRI examination^[8]. The good prognosis group showed ventral brainstem lesions or dorsal superficial brainstem lesions. These lesions appeared to be a direct focal brainstem injury caused by an impact against the free tentorial edge. The poor prognosis group showed deep dorsal brainstem lesions. These lesions were considered to be due to DAI. These acute stage findings, especially superficial small lesions, are seen only temporally in many cases, thus MRI should be taken in the acute stage, namely the first few days after injury.

Moen *et al* evaluated a longitudinal MRI study in patients with traumatic brain injury^[9]. They concluded that many brainstem lesions were detected only in the acute phase, and only early MRI findings predicted the clinical outcome. Their results showed that the number and volume of the lesions detected on early MRI predicted the clinical outcome, however, they did not analyze the location or depth of the lesions.

Chew et al retrospectively analyzed the MRI and clinical records of 36 patients with traumatic brainstem injury and found that "crossing midline" bilateral injuries to the pons and medulla are associated with poor outcomes, although their two patients with "midline-crossing" ventral pontine lesions both achieved a functional recovery^[10]. In addition, although they analyzed the volume of the brainstem lesions, they did not analyze the location or depth of the brainstem lesions. In our paper, as Dr. Chew described, superficial and ventral brainstem lesions were associated with good outcomes. Even though continuous lesions were observed at the bilateral brainstem, we did not use the term "midline-crossing" to describe these lesions because we did not understand the mechanism of injury. If unilateral lesions progress to another side, "midline-crossing" injury may be an appropriate name; however, concurrent bilateral injuries do not cross, but rather are attached to each other. Therefore, we analyzed only unilateral and bilateral injuries^[7]. I believe that the brainstem lesion volume is not the single prognostic factor; however, it is reasonable to suspect that patients with larger brainstem lesions will exhibit poorer prognoses than patients with small brainstem lesions. In Dr. Chew's result, the total volume of brainstem injury did not correlate with the outcome. This result may be due to some problems in the study design and confounding factors. In their study, patients who did not recover underwent MRI an average of 0.8 days after admission, whereas those who recovered underwent MRI an average of 4.2 days after admission. The authors reported that lesion size did not exhibit a linear regression with the time to scan. The time after injury with maximum

lesion size depends on various factors. Small lesions may disappear in a few days after injury. The contusion lesion size sometimes increases over a few days after injury due to secondary injuries. We cannot know the exact timing of the maximum lesion size without conducting a sequential image acquisition study.

Hilario et al retrospectively analyzed the MRI data from the first 30 days of 108 patients with severe head trauma, and found that the presence of posterior and bilateral brainstem injuries were poor prognostic signs^[11]. Although they analyzed the location of the brainstem lesions, they did not analyze the volume or depth of the brainstem lesions. In their study, the median time between trauma and MRI was 17 days, with a maximum 30 days. In our study, MRI was carried out within 6 days, generally 2 days after the injury [7]. Superficial dorsal brainstem injury was found to be an indicator of a good prognosis, while only deep dorsal brainstem injury was related to a poor prognosis. Small lesions may disappear in a few days after injury. Most of the injuries in Dr. Hilario's series of patients had affected the posterior dorsolateral aspect of the midbrain. Dr. Hilario's study may have underestimated the lesion size and presence of brainstem injury. They did not extensively discuss the cause of unilateral injuries with a good prognosis, except for the possibility of supratentorial herniation, and they did not discuss the clinical and MRI findings of supratentorial herniation. Our study excluded secondary brainstem injury associated with cerebral herniation and evaluated only "primary" brainstem injur^[7].

We discussed 2 mechanisms of brainstem injury: primary brain stem injury that occurs following a direct impact of the brainstem against the tentorial free edge, and brainstem injury associated with DAI. Direct focal brainstem injuries caused by an impact against the free tentorial edge have been pathologically and radiologically recognized^[2,3]. It is sometimes difficult to differentiate these 2 mechanisms of injury; however, the brainstem lesion size and the location and supratentorial lesion findings are helpful. Recent studies^[9,10,11] did not discuss the mechanism of brainstem injury. I believe that differentiating these 2 mechanisms of brainstem injury is therefore critical for accurately diagnosing and understanding traumatic brainstem injuries. In Dr. Hilario's study, non-hemorrhagic injuries showed the highest positive predictive value for a good outcome, although no explanation was discussed. Obviously, the presence of no injuries would be most predictive of a good outcome.

The importance of early MRI examination appears to be a consensus. Our study demonstrated that small superficial lesion may be detected only a few days after injury. The acute phase within a few days after a head injury may show an unstable vital sign, thus intensive monitoring of the vital sign and manual or mechanical ventilation during a MRI examination should be employed. Direct brain stem contusion has a good prognosis compared with brainstem injury associated with DAI. Small superficial lesions are detected only within a few days after a head injury. Recent studies of MRI in patients with head injuries could not determine the pathology. However, the differentiation of the pathophysiology must be clinically important because the prognoses are different and distinguishable using only acute stage MRI.

CONFLICT OF INTEREST

There are no conflicts of interest with regard to the present study.

REFERENCES

 Gentry L, Godersky J, Thompson B. Traumatic Brain Stem Injury: MR imaging. Neuroradiology. 1989;171:177-87.

- Oppenheimer DR. Microscopic lesions in the brain following head injury. J Neurol Neurosurg Psychiatry. 1968;31:299-306.
- Clifton G, McCormick W, Grossman R. Neuropathology of early and late deaths after head injury. Neurosurgery. 1981;8:309-14.
- 4. Firsching R, Woischneck D, Diedrich M, Klein S, Ruckert A, Wittig H, *et al* Early magnetic resonance imaging of brainstem lesions after severe head injury. J Neurosurg. 1998;89:707-12.
- Andreas Kampfl, Gerhard Franz, Franz Aichner, Bettina Pfausler, Hans-Peter Haring, Stefan Felber, et al The persistent vegetative state after closed head injury: clinical and magnetic resonance imaging findings in 42 patients. Journal of Neurosurgery. 1998;88:809-16.
- Kampfl A, Schmutzhard E, Franz G, Pfausler B, Haring H-P, Ulmer H, et al Prediction of recovery from post-traumatic vegetative state with cerebral magnetic-resonance imaging. The Lancet. 1998;351:1763-7.
- Shibata Y, Matsumura A, Meguro K, Narushima K. Differentiation of mechanism and prognosis of traumatic brain stem lesions detected by magnetic resonance imaging in the acute stage. Clin Neurol Neurosurg. 2000;102:124-8.

- Matsumura A, Meguro K, Mizutani T, Tsurushima H, Satoh N, Tsunoda T, et al Cardiorespiratory monitoring and support during MRI examination in critically ill patients. Japanese Journal of Magnetic Resonance in Medicine. 1989;8:268-74.
- Moen KG, Skandsen T, Folvik M, Brezova V, Kvistad KA, Rydland J, et al A longitudinal MRI study of traumatic axonal injury in patients with moderate and severe traumatic brain injury. Journal of Neurology, Neurosurgery & Psychiatry. 2012;83:1193-200.
- Chew BG, Spearman CM, Quigley MR, Wilberger JE. The prognostic significance of traumatic brainstem injury detected on T2weighted MRI. J Neurosurg. 2012;117:722-8.
- Hilario A, Ramos A, Millan JM, Salvador E, Gomez PA, Cicuendez M, et al Severe Traumatic Head Injury: Prognostic Value of Brain Stem Injuries Detected at MRI. American Journal of Neuroradiology. 2012;33:1925-31.

Peer reviewer: Jürgen Hänggi, PhD, Division Neuropsychology Department of Psychology, University of Zurich, Binzmuehlestrasse 14 / P.O. 25, CH-8050 Zurich, Switzerland.