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Hypersomnia due to injury of the lower ventral ascending reticular activating system in a patient with intraventricular hemorrhage

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Running title: Hypersomnia following intraventricular hemorrhage

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Disclosures

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Key words

Hypersomnia, Intraventricular haemorrhage, Diffusion tensor tractography, Ascending reticular activating system

1. Introduction to the case

A 74-year-old female underwent extraventricular drainage for an intraventricular haemorrhage (IVH) at the neurosurgery department of a university hospital (Fig. 1-A). She exhibited severe hypersomnia following IVH onset. At four weeks after onset, she was transferred to the rehabilitation department of the same hospital to undergo rehabilitation. Except for approximately three hours during daytime, she remained asleep. Her sleep abnormality was indicated as severe hypersomnia on the Epworth Sleepiness Scale (score = 23; maximum score = 24; cut off for severe hypersomnia = 10) [1].

2. Image analysis

Diffusion tensor imaging data were acquired at four weeks after IVH onset by using a 1.5 T Philips Gyroscan Intera (Philips, Best, Netherlands). Two portions of the ascending reticular activating system (ARAS) were reconstructed by selecting fibres passing through regions of interest (ROIs). For analysis of the lower dorsal ARAS, the seed ROI was placed on the pontine reticular formation (RF), and the target ROI with the option of termination was placed on the intralaminar thalamic nucleus (ILN) [2]. For reconstruction of the lower ventral ARAS the seed ROI was placed on the pontine RF and the target ROI with the option of termination was placed on the hypothalamus [2]. Out of 5000 samples generated from a seed voxel, results were visualized at a threshold of 2 streamlined through each voxel for analysis. The lower ventral ARAS was not reconstructed on either sides, whereas the lower dorsal ARAS was well-reconstructed on both sides.

Figure 1

3. Discussion

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Since Moruzzi and Magoun's study in 1949, the ARAS has been considered as a crossroad of arousal regulation [3]. Using diffusion tensor tractography, we evaluated the patient's two lower portions of the ARAS: the lower dorsal ARAS between the pontine RF, the thalamic ILN, and the lower ventral ARAS between the pontine RF and the hypothalamus. We observed that the lower ventral ARAS was not reconstructed on either side, indicating severe injuries of both lower ventral ARAS. Based on our results and several studies which reported a close association between the hypothalamus and hypersomnia, as well as that injuries of the lower ventral ARAS are closely related to hypersonnia; the hypersomnia in this patient appears to be mainly ascribable to injury of the lower ventral ARAS [2,4,5]. Furthermore, the pathophysiological mechanism of injury of the lower portion of the ARAS in this patient appears to be related to the pathogenic mechanism of the neural injury resulting from the hematomas in the intraventricles (third and fourth ventricles). Previous studies have suggested that injury to periventricular white matter could occur through mechanical (increased intracranial pressure or direct mass) or chemical injury mechanisms of hematomas (as a blood clot itself can cause extensive damage). In this study, considering the normal configuration of the lower dorsal ARAS, which passes the pontine tegmentum along with the lower ventral ARAS close to the fourth ventricle, the injury of the patient's lower ventral ARAS appears to be mainly ascribed to the hematoma in the third ventricle adjacent to the hypothalamus.

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Figure Legend

Fig. 1.

A. Brain computed tomography images at onset show haematomas in the intraventricles of the third and fourth ventricles. T2-weighted brain magnetic resonance images at four weeks after onset reveal no abnormal lesion around the third and fourth ventricles.

B. Results of diffusion tensor tractography (DTT) for the patient: normal configuration of the lower dorsal ascending reticular activating system and non-reconstruction (arrows) of both the lower ventral ascending reticular activating system compared with a normal subject (67-year-old female).

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