

Frontal cognitive dysfunction with apraxia of eyelid opening after traumatic brain injury: a slowly progressive delayed complication

M.Y. Park, D. Shin, M. Kim

Neurology, Yeungnam Medical Center, South Korea

Apraxia of eyelid opening (AEO) has been suggested to be a dysfunction of the supranuclear control of the levator palpebrae superioris caused mainly by basal ganglial lesion. The hypometabolism of the medial frontal lobe may be a pathophysiologic mechanism in frontal lobe dysfunction and AEO. We report two patients who developed frontal cognitive dysfunction with AEO, as a delayed complication, 4-6 years after traumatic brain injury (TBI) progressively. Their MRI showed encephalomalacia with low signal intensity in medial frontal cortex which suggests loss of cortico-spinal tract fibers in the medial frontal cortex, which was not shown in initial brain CT scans. Delayed pathologic changes after TBI may contribute to the development of frontal cognitive dysfunction and AEO in these cases. Therefore we need to make close observation if the patients have TBI, especially severe enough to loss of consciousness even their brain imaging scans do not show abnormal finding initially. Key words: frontal cognitive dysfunction, apraxia of eyelid opening, traumatic brain injury, delayed complication