

Pusher syndrome improved by secondary newly developed stroke

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Abstract

Pusher syndrome is a neurological disorder where the patient actively pushes away from the non-hemiparetic side following brain damage. We experienced a case in whom intractable pusher syndrome associated with thalamic hemorrhage improved following a newly developed pontine infarction. A 63-year-old man showed severe pusher syndrome after an initial thalamic hemorrhage. After approximately 2 years, a pontine infarction developed and improved the persistent pusher syndrome. We postulate that it resulted from involvement of the medial lemniscus with interruption of the distorted upward proprioceptive signal of body orientation.

Keywords: Pusher Syndrome

INTRODUCTION

Pusher syndrome is a neurological disorder where the patient actively pushes away from the non-hemiparetic side following brain damage. The disorder impedes rehabilitation following brain injury and provokes a tendency to fall.^{1,2} It can also increase the length of hospital stay.² However, it is generally known to have a good prognosis and does not influence the outcome of rehabilitation. Pathologic pusher syndrome usually recovers within 6 months of the brain injury.³

We managed a case in whom pusher syndrome associated with thalamic hemorrhage did not improve until about 2 years after the injury; where he improved following a new brainstem infarct. To our knowledge, there has been no previous similar report in the literature. We report here the case and discuss the possible pathological mechanism.

CASE REPORT

A 63-year-old man was admitted to the neurosurgery department of a university hospital after presenting with a reduced level of consciousness on March 14, 2013. His brain computed tomography showed a large intracranial hemorrhage in the left basal ganglia and thalamus, in addition to an intraventricular hemorrhage (Figure 1). He underwent emergency extraventricular drainage.

He was transferred to a rehabilitation center on April 19, 2013. He had drowsiness and right hemiplegia with Medical Research Council grade 0/5. A more detailed neurologic and physical examination were not possible because of the poor cooperation due to cognitive impairment and drowsiness. His medical history was unremarkable; however, he had a history of significant alcohol abuse. With intensive rehabilitation, his motor weakness improved and he became more alert.

As his muscle power improved, sitting and standing training was performed. His posture while sitting and standing was severely tilted towards the right side, with a tendency to fall. When he was sitting, the left upper extremity was abducted forcefully from his body and the left elbow was extended at rest. When he was standing, the left lower extremity was abducted forcefully, and the left hip and knee were extended at rest. He actively resisted correction of the right tilted posture when he was sitting, as well as standing. Based on these findings, a diagnosis of pusher syndrome was made.¹ The score of the Scale for Contraversive Pushing was 6/6.⁴

After about 3 months, a manual muscle test revealed Medical Research Council grade 4/5. However, he could not sit alone because of the severe pusher syndrome. The score of the Scale for Contraversive Pushing remained 6/6. His pusher

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Figure 1. Brain computed tomography showed a large intracranial hemorrhage in the left basal ganglia and thalamus, with intraventricular hemorrhage.

syndrome and functional level showed no signs of improvement despite intensive rehabilitation until the next neurological event about two years following the initial illness.

On February 25, 2015, he suddenly started drooling. His neurologic exam revealed a deteriorated right facial palsy, hemiplegia and dysarthria. The manual muscle test showed Medical Research Council grade 2/5 in the right upper and lower limbs. Sensory testing was not performed because of cognitive impairment. The score of his Mini-Mental Status Examination was 7/30. Magnetic resonance imaging was performed and showed a left pontine infarction (Figure 2). Antiplatelet therapy was started. A week after acute care, rehabilitation was resumed. Despite

deterioration of his right hemiplegia, he could sit alone because of the improvement in his previous pusher behaviors. The deteriorated neurologic signs recovered a short time later. The manual muscle test revealed Medical Research Council grade 4/5 in the right upper and lower limbs. Gait training with one person assistance was now possible. His posture while sitting was mildly tilted and his posture while standing was severely tilted, towards the right side, however without a tendency to fall. When he was sitting, the left upper extremity was not abducted from his body and the left elbow was not extended until his position was changed. When he was standing, the left lower extremity was not abducted, and the left hip and knee were not extended until his position was changed. Resistance to passive correction of the right tilted posture was not demonstrated while sitting, but was shown while standing. The score of the Scale for Contraversive Pushing was 3/6.

DISCUSSION

Despite many studies related to pusher syndrome, the mechanism underlying the disorder and its related anatomy have not been clarified.² To date, lesions of the posterior thalamus, insula and postcentral gyrus are known to be associated with pusher syndrome. These neural structures process the afferent somatosensory signals mediating gravioceptive information on upright body posture. Thus, it is thought that misperception of body orientation in relation to gravity induced by these lesions produce pusher syndrome.^{2,5}

It is interesting that a newly developed pontine infarction inhibited pusher behaviors in our case. The lesion was located in the pyramidal tract in the pons, which was consistent with his worsening of motor weakness. We do not believe

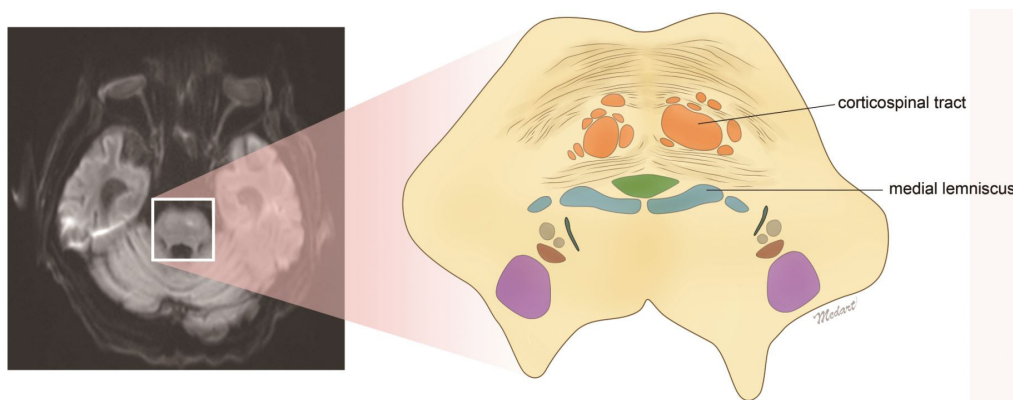


Figure 2. Magnetic resonance imaging showed a left pontine infarction located in the pyramidal tract. Right neuroimaging illustration showed the location of medial lemniscus dorsal to the pyramidal tract.

that dysfunction of the pyramidal tract improved the pusher syndrome, because the improvement in the symptoms were still present after restoration to the previous state from the motor weakness. We postulate that the improvement in pusher syndrome resulted from a dysfunction of medial lemniscus located dorsal to the pyramidal tract (Figure 2). The medial lemniscus is responsible for proprioception, namely the awareness of body position.⁶ We presume that dysfunction of the upward proprioceptive signal reduced the distorted processing of body orientation. Demonstrating proprioceptive impairment could provide support for these hypotheses. However, sensory testing was impossible in this case due to his cognitive impairment. The behavioral verticals, such as the visual vertical, haptic vertical, and postural vertical, were not testable because of his cognitive impairment. Our case suggests that afferent sensory signals *per se* can affect pusher behaviors. This finding can help the study of the mechanisms of this disorder.

DISCLOSURE

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Conflicts of interest: None

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