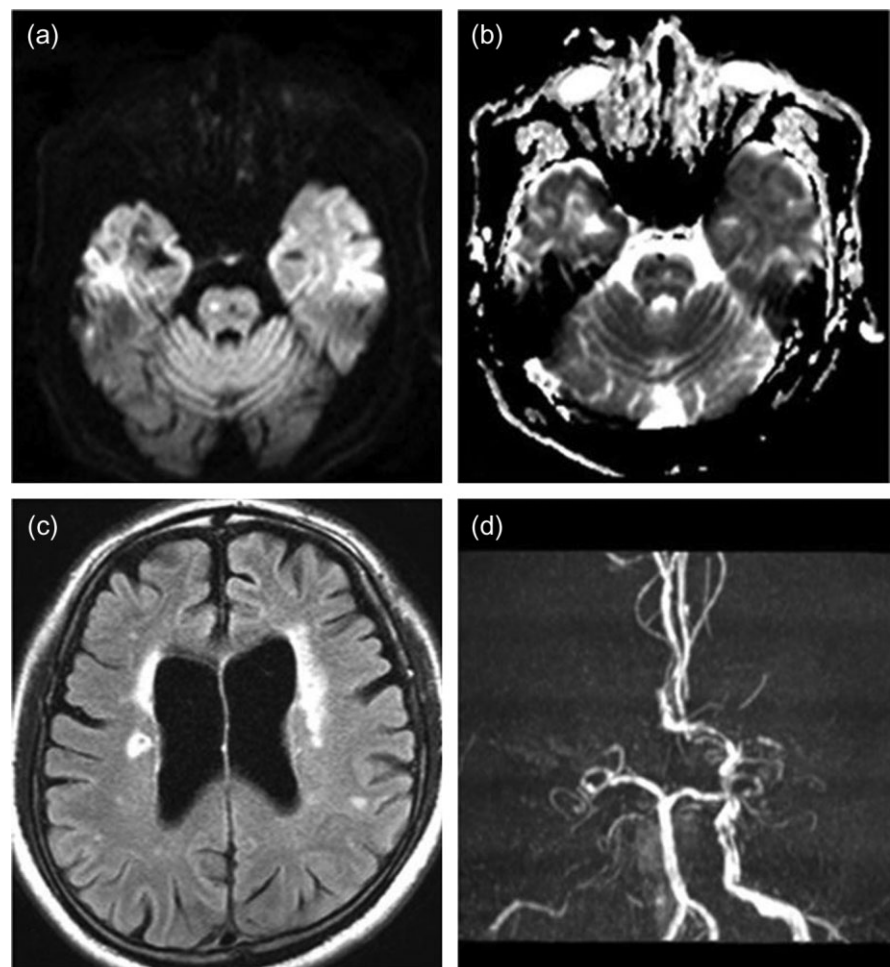


LETTERS TO THE EDITOR  
CASE REPORT**Acute pontine infarction presenting with depressive mood only**

Dear Editor,

A 65-year-old male patient presented to Chosun University hospital in Gwangju, South Korea, complaining of sudden mood swings for 4 days. He was bright and diligent before the visit. He had become suddenly curt, unfriendly and lethargic, and then 2 days after, had more progressive mood swings. Four days earlier, his usual bright and hard-working demeanor had suddenly been replaced with a curt and unfriendly attitude. He had a history of hypertension and type II diabetes for 10 years and 4 years, respectively. The patient did not drink or smoke.

Brain magnetic resonance imaging, taken 4 days after symptom onset, showed a small lesion in the right pontine tegmentum. It showed high signal intensity in the diffusion weighted image and low signal intensity in the apparent diffusion coefficient image. These results suggested an acute cerebral infarction (Fig. 1a,b). High signal intensities were observed in the right corona radiata, and no abnormal findings were detected in other sites, such as the periventricular area, deep white matter and subcortical gray matter in T2-weighted images (Fig. 1c). Magnetic resonance angiography showed occlusion of the right proximal carotid artery and left proximal middle cerebral artery (Fig. 1d). The



**Figure 1** Magnetic resonance imaging shows (a) diffusion restriction in the right pons with diffusion weighted image and (b) apparent diffusion coefficient image. (c) Fluid-attenuated inversion recovery image shows hyperintensity on the right corona radiata and bilateral periventricular white matter. (d) Magnetic resonance angiography shows right proximal carotid artery and left proximal middle cerebral artery occlusion.

Korean version of the Geriatric Depression Scale was carried out to measure the severity of depression on hospital day 3. He scored 26 points from a total of 30, and was diagnosed with severe depression. We prescribed 75 mg of clopidogrel and 30 mg of duloxetine, to no effect. Duloxetine was replaced with 10 mg of escitalopram, a selective serotonin reuptake inhibitor. Afterwards, dysphagia improved completely and depression symptoms also gradually improved. Two weeks later, his conditions did not require specific attention and he was discharged.

In this case, a patient with a small pontine infarction experienced depression only, without other neurological symptoms. It has been reported that in the early stage of post-stroke depression (PSD) with a left anterior lesion, in the majority of cases, depression is accompanied by other neurological symptoms. There has been a previous report of a pontine infarction exhibiting emotional incontinence, but there have not been any reports of a case manifesting depression only.<sup>1</sup> The prevalence of PSD is approximately 30–50%, 3–6 months after a stroke has occurred.<sup>2</sup> PSD occurring within 2 weeks has been associated with the left cerebral hemisphere, although these associations were not observed after 2 weeks to 6 months had passed. Paradoxically, after 6 months, PSD were associated with the right cerebral hemisphere.<sup>3</sup> Recently, it was reported that PSD is not related with stroke size and location.<sup>4</sup>

Of the many explanations regarding the pathophysiology of depression, the serotonin theory and norepinephrine theory are currently receiving the most attention. Serotonin controls emotions, and decreased levels can cause depression, aggression, anxiety and bulimia. The increased levels of serotonin, otherwise, can cause mood elevation and have analgesic effects. Serotonin is secreted from the raphe nucleus located in the pontine tegmentum. Serotonin neurons originate from the raphe nucleus, and relay signals to the frontal lobe and basal ganglia. Lesions in the former pathway might cause problems in mood control, whereas lesions in the latter pathway might result in problems with motor functions, and cause obsessive-compulsive thoughts and behavior. The pontine lesion of this patient might have been located just over the pathway leading to the frontal lobe, with minimal impact on the other pathway, resulting in mood symptoms with largely preserved motor functions. Furthermore, norepinephrine signals originate from the locus coeruleus, which is also located in the pons. As reduced adrenergic levels can result in lethargic and avolitic attitudes, the lesion of this patient might have interfered with adrenergic signaling.

Because of its complex structure, pontine tegmentum infarctions could cause various neurological symptoms.

The fifth, sixth, seventh and eighth cranial nuclei are located nearby and can be damaged, causing various symptoms. Damage to the fifth cranial nerve can cause ipsilateral loss of facial pain sense and paralysis of facial muscles. Damage to the sixth, seventh and eighth cranial nerves can cause diplopia, ipsilateral facial nerve palsy, vertigo and disequilibrium, respectively. Medial lemniscus damage might cause loss of facial touch and press sense, and medial longitudinal fasciculus damage could cause diplopia with eyeball movement disorder. Damage to the trapezoid body and lateral lemniscus might cause auditory dysfunction, and if the middle cerebellar peduncle is damaged, ataxia might occur.

In the present case, an infarction occurred in the central tegmentum of the mid-pons. The medial longitudinal fasciculus passes to the medial side of the tegmentum, and the fifth cranial nerve and middle cerebellar peduncle pass to the lateral side of the tegmentum. Therefore, related symptoms did not occur in the present case. The sixth cranial nerve and medial lemniscus passes through the central tegmentum in the caudal pons, but in this case the lesion was located in the mid-pons, and as the medial lemniscus is located on the dorsal side of the tegmentum, and the sixth nerve passes at a lower level than the lesion, related neurological symptoms also did not occur. We have experienced and reported that a pontine cerebral infarction occurred showing mainly symptoms of depression, without other concomitant neurological symptoms.

## Disclosure statement

No potential conflicts of interest were disclosed.

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