Neurology Asia 2021; 26(1) : 145 – 148

CASE REPORTS

Acute onset post-stroke pain from a paramedian pontine infarct

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Abstract

Acute post-stroke pain can be a late complication; rarely it may occur in the acute phase of a stroke. However, pain following acute pontine infarcts are exceedingly rare and all such cases described had pain and paresthesia involving only the face. There are no reports of pontine strokes causing pain or abnormal sensory involvement in the limbs. Here we describe a patient where following an acute paramedian pontine infarct; one of the main presenting symptoms was acute episodic very severe burning paresthesia on the side of hemiparesis. This post-stroke pain was also associated with remarkably high blood pressure surges.

Keywords: Acute post stroke pain, pontine pain, acute pontine infarction, paresthesia, hemiparesis, dysthesias

INTRODUCTION

Central post-stroke pain (CPSP) is a term used to describe the symptom of pain arising after a stroke. The classical example is pain following thalamic strokes (Dejerine-Roussy syndrome).[1] Radiologic studies shown that lesions located at any point of the pain processing pathways in the central nervous system including the brain stem can cause CPSP.[2] CPSP is commonly seen as a long-term sequel of stroke; rarely it can occur in the setting of acute strokes involving the thalamus and the sensory cortices.[3] However pontine infarcts with CPSP are extremely rare. There are a few case previous reports of acute pontine strokes associated with pain and dysesthesias limited to the face area.[4,5]

Here we describe an exceedingly rare case where following an acute paramedian pontine infarct, the main presenting symptoms was acute episodic very severe burning paresthesia on the side of hemiparesis which was also associated with remarkably high blood pressure surges.

CASE REPORT

A 42 year-old lady who was a known hypertensive was admitted with history of weakness on the right side of the body and intermittent severe burning pain on the same side. Her symptoms started 24 hours prior to the admission and her weakness which was initially mild has progressed. On examination she was fully conscious and orientated. The cranial examination showed a right sided upper motor neuron type of facial weakness and mild dysarthria. The tone was mildly increased in the right side with power in the upper limb of 0/5 and in the lower limb of 2/5. The deep tendon reflexes were exaggerated on the right side with extensor plantar response. The sensory examination including, touch, pain, joint position, proprioception, and cortical sensations were normal.

The brain MRI showed acute left paramedian lower pontine infarct. There was no feature of any basilar artery occlusion or pathology (Figure 1).

The patient had intermittent bouts of very severe burning paresthesia involving the right side of the body. The attacks were described by the patient as the worst possible pain graded 10/10 on the numeric rating scale (NRS). These pain attacks were not associated with any triggers. Additionally, there were no other associated phenomena such as allodynia or hyperalgesia neither during nor in between the attacks. The

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Date of Submission: 3 June 2020; Date of Acceptance: 3 November 2020

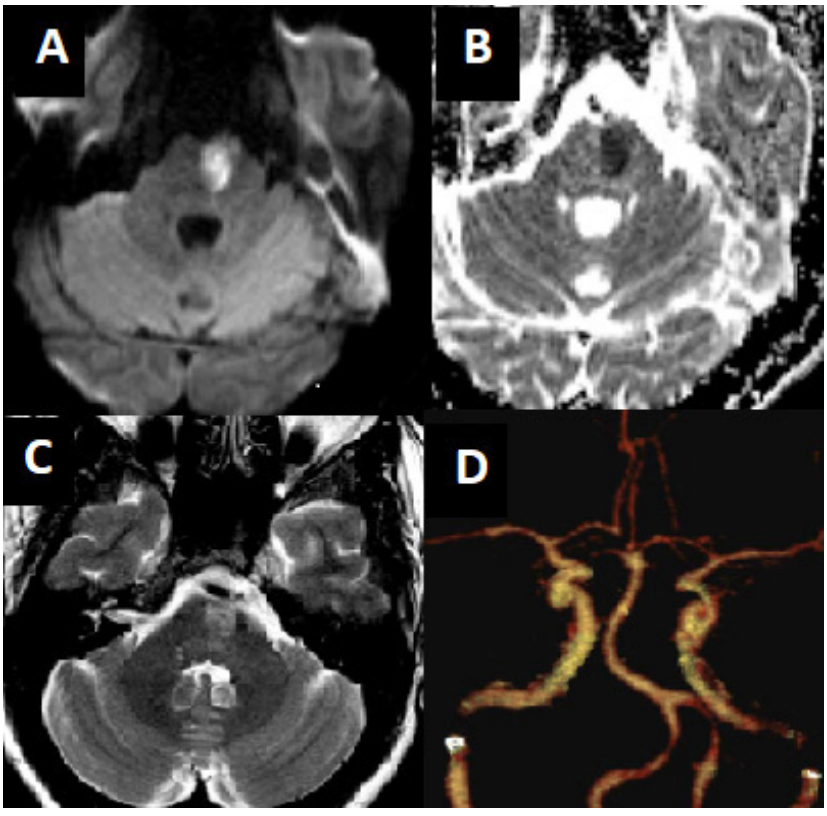

Figure 1. Brain MRI showed acute left paramedian lower pontine infarct. A. Diffusion Weighted Imaging (DWI) showing hyperintense paramedian lower pontine lesion. B. Apparent Diffusion Coefficient (ADC) showing hypointense paramedian lower pontine lesion. C. FLAIR showing hyperintense paramedian lower pontine lesion. D. CT angiogram with reconstruction showing normal basilar artery.

burning paresthesia was mainly involving the medial parts of the arm above and below the cubital fossa /elbow joint and also the lower limbs medially above and below the knee joint/ popliteal fossa (Figure 2).

These episodes occurred one to two times in a day mainly in the late evenings and night often waking her up from sleep. Each episode lasted 3-4 hours. There was a concomitant rise in the blood pressure during these episodes. Some of these blood pressure surges were high enough to warrant boluses of IV labetalol. Otherwise the blood pressure readings normalize once these episodes subside. This blood pressure rises most likely were due to severe pain, since the blood pressure readings remains normal otherwise. The patient was fully conscious and orientated at the time of these episodes and there was no change in her motor power or any involuntary movements.

An EEG done at the time of one of her painful episodes did not show any epileptiform activity. The somatosensory evoked potentials from the tibial nerve was normal. MRI of the spine was

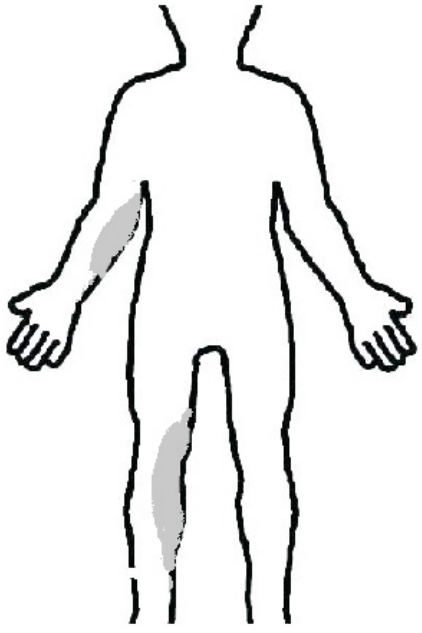

Figure 2. A cartoon illustration of patient's distribution of pain.

unremarkable except for mild cervical and lumbar disc protrusions.

While in the ward her power gradually improved over a 6-day period to a grade of 3/5 in the upper limb and 4/5 in the lower limb. The facial weakness also improved. For her episodic pain she was initially started on pregabalin and subsequently amitriptyline was added. However, she had only minimal relief of her symptoms. Her blood pressure remainedwithin the normal range except for sudden surges during her painful episodes.

Workup towards pheochromocytoma and renal artery stenosis and other endocrine causes were normal. Workup towards young stroke including assays for Protein S, C, antithrombin 3 were negative.

Since her episodic pain was not controlled; carbamazepine was added, and the dose was gradually titrated up until she has improvement in her symptoms. She was subsequently discharged.

At three months follow up; her motor functions had improved to 4+/5 power in both upper and lower limbs. Her burning paresthesia had improved considerably. Pregabalin and amitriptyline were stopped. She was continued on a tapering schedule of carbamazepine.

DISCUSSION

Pain can occur as an important non motor symptom in strokes. Multiple pain mechanisms both central or peripheral often coexist. CPSP is one of the causes; others being post-stroke spasticity, shoulder pain, and complex regional pain syndrome (CRPS).

In patients with CPSP, sensation of pain is triggered with very minimal or no stimulation of the peripheral pain receptors. The patient may describe the pain in a variety of terms ranging from a dull ache to an excruciating lancinating pain.[6] Majority CPSP occurs by 6 months following the stroke.[7]

Tasker[8] described three types of pain components in CPSP which were a constant component, an intermittent component, and a hyperalgesia/ allodynia component. Often these components could occur in combination.

In the Helsinki Stroke Study[9], young stroke patients were more at risk for developing CPSP. Even though CPSP is well-known to occur in the post stroke period, such central pain occurring in the acute setting is rare. Bayat *et al*.[10] described a patient with stroke in the insular region with acute onset lateralized neuropathic pain.

Many brainstem nuclei are involved in pain processing and the lateral medullary syndrome is the commonest brainstem syndrome associated with CPSP.[7] In pons, pontine reticular formation, periaqueductal gray, and raphe nuclei in the rostral pons are involved with pain processing.[11] Facial pain and dysesthesias have been documented with acute pontine strokes and can often be the heralding symptom.[5,12] Caplan[13] described 3 patients with acute pontine infarcts with "salt and pepper "dysesthesia over the face. Lateral pontine infarcts presenting with facial pain similar to trigeminal neuralgic has also been reported.[4] Chen *et al*.[14] reported a case of pontine tegmental infarct presenting with acute episodic burning pain in a cheiro-oral distribution. Their patient also had associated sudden elevations in blood pressure with the pain. They hypothesized the episodic component of the paraesthesia to a relapsing expansion of focal cerebral oedema. Our patient is similar with pain and blood pressure elevations occurring in episodes. However, our patient had the burning pain over the paretic upper and lower limbs sparing the face.

In conclusion, we present a unique case of acute CPSP associated with acute paramedian pontine infarction involving the limbs.

DISCLOSURE

Financial support: None

Conflict of interest: None

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