

Atypical presentation of thalamic post-stroke pain

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Central post-stroke pain (CPSP) is a neuropathic pain syndrome occurring after a cerebrovascular accident. CPSP following a thalamic stroke (“thalamic pain”) is characterized by severe, persistent, paroxysmal, often intolerable pain on the hemiplegic side, unresponsive to any analgesic treatment [1].

We describe a patient in whom thalamic pain, which was the main presenting symptom of hemorrhagic stroke, was initially considered as peripheral neuropathic in nature, due to an atypical distribution of pain and the presence of muscular weakness.

A 76-year-old man came to the Emergency Room for pain in his left thigh begun abruptly 3 days before and progressively increased in intensity, becoming intolerable. He had a history of hypertension, diabetes and chronic low back pain. On admission, neurological examination revealed moderate weakness in left hip flexion (Medical Research Council (MRC) 4-/5), absent deep tendon reflexes in lower limbs, allodynia and hyperpathia in the left anteromedial thigh, associated with a mild decrease in superficial sensation over the same region; sensation in response to pinprick, light touch and temperature, as well as vibratory sensation, was symmetrically decreased in feet and legs with a stocking distribution and a distal–proximal gradient (Fig. 1a). Subjective numbness associated with pain (described as severe, persistent, intolerable, deep and

sometimes burning) over the left anteromedial thigh was reported.

Brain computed tomography (CT) performed exclusively on the unfavorable vascular risk factor profile of the patient (diabetes and hypertension and acute onset of symptoms) showed a small hemorrhage in the right lateral thalamus ruptured in the homolateral ventricle (Fig. 1b). Neurophysiological study ruled out the coexistence of a lumbar radiculopathy/plexopathy or femoral mononeuropathy, showing only a sensory polyneuropathy.

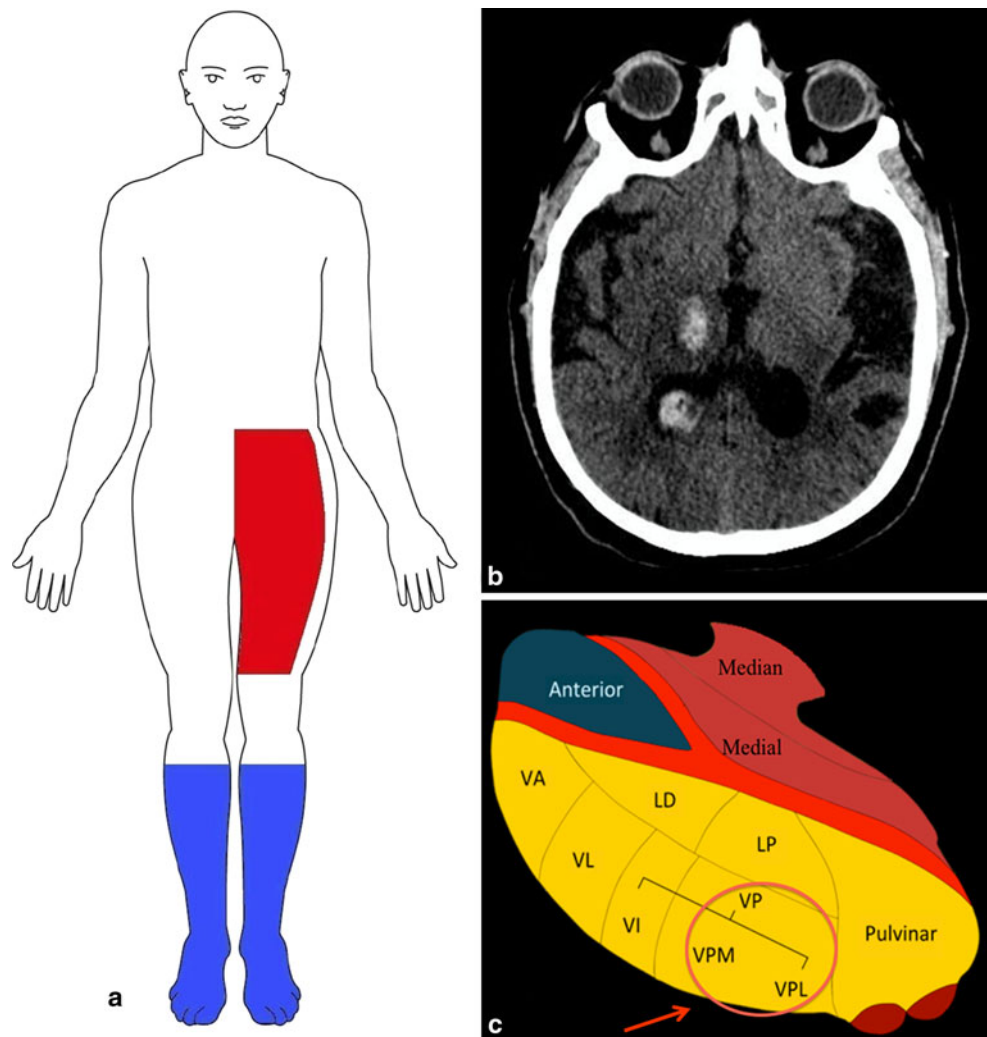
During the hospitalization, the pain progressively and spontaneously reduced, and completely disappeared within 1 week, without requiring pharmacological treatment. According to the literature [2], we did not use mannitol since there were no signs of increased intracranial pressure, and mean blood pressure was maintained below 130 mm Hg.

In this patient, some neurological findings (absent deep reflexes in lower limbs, vibration and superficial sensation symmetrically decreased with a stocking distribution) were indicative of a distal, symmetrical sensory polyneuropathy, probably secondary to diabetes. Other findings were suggestive of a possible lumbar radiculopathy (history of chronic low back pain; weakness in left hip flexion; pain and mild decrease in superficial sensation over the left antero-medial thigh; absent knee reflexes). Overall, these findings could be compatible with an L2/L3 radiculopathy causing hip flexors’ weakness; alternatively, although less probable, given the muscular weakness confined to hip flexion and the sensory disturbance distribution, a diagnosis of partial lumbar plexopathy/femoral nerve mononeuropathy secondary to diabetes could be taken into consideration.

The case we describe represents an atypical presentation of CPSP.

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Fig. 1 **a** Distribution of sensory abnormalities related to length-dependent polyneuropathy (*blue*) and to CPSP (*red*), colors available in the online version, **b** brain CT scan shows a small hemorrhage in the *right* lateral thalamus (maximal diameter 38 mm) ruptured in the homolateral ventricle, **c** site of the thalamus involved in the onset of pain (*arrow*). *LD* lateral dorsal nucleus, *LP* lateral posterior nucleus, *VA* ventral anterior nucleus, *VL* ventral lateral nucleus, *VPL* ventral posterolateral nucleus, *VPM* ventral posteromedial nucleus (color figure online)



Neuropathic pain is defined as “pain arising as a direct consequence of a lesion or disease affecting the somatosensory system” [3]. Diagnosis relies on a detailed history, accurate general and neurological examinations (focusing on an accurate sensory evaluation) and imaging of lesions [3]: in the presence of history suggestive of stroke, confirmed by imaging, with pain developing at the moment or after the onset of stroke, and within an area of the body corresponding to the central nervous system lesion, a diagnosis of CPSP could be performed, after having excluded other possible causes of pain. Conversely, in case of coexistence of several type of pains, concomitant diseases or delayed onset of pain following stroke, CPSP may be misdiagnosed [4]. CPSP occurs after lesions at any level of the somatosensory pathways of the brain including medulla, thalamus and cerebral cortex [1]. CPSP can be due to an ischemic/hemorrhagic stroke involving the thalamus. The posterolateral region of the thalamus is the most common location of thalamic hematoma, and is caused by the rupture of the thalamogeniculate arteries that often involves the posterior horn of lateral ventricle [5]. Pain due

to intrathalamic location of the hematoma usually develops between 3 and 15 days after the onset and it is associated with decreased touch and pain sensation at the examination [5]. Pain may be spontaneous or evoked, often fluctuates and it is described in many ways such as “lacerating” “burning” or “pricking”, although there are no pathognomonic features or uniform signs with regard to onset, presentation and intensity [1]. Even if hemibody pain is more common in patients with thalamic stroke, the distribution can range from small to large areas [1].

According to the international grading system [3] in our patient a diagnosis of definite CPSP could be made (pain distribution, history, relevant lesion or disease, negative or positive sensory signs confined in the innervations territory of the lesioned nervous structure, diagnostic test confirming lesion or disease explaining neuropathic pain).

Although it was impossible to obtain a proven anatomic-clinic correlation since we did not perform an MRI, the CT scan pointed to a hematoma involving the posterolateral thalamic region (Fig. 1c). The pain ceased within 1 week from the onset possibly because of a prompt resolution of

the hemorrhage (as demonstrated by further CT), before thalamic changes occurred.

Our case demonstrates that pain may be the presenting and main symptom of a cerebrovascular accident; when not appropriately taken into account, it may be misdiagnosed or erroneously attributed to concomitant diseases or conditions. In the diagnostic workup of pain, it is important to always consider CPSP in particular in presence of significant risk factors for cerebrovascular disease when other explanations have been ruled out. A high level of suspicion is necessary for the identification of cerebral lesions in those cases in which pain is the only clinical presentation.

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