Disappearance of haemorrhagic stroke-induced thalamic (central) pain following a further (contralateral ischaemic) stroke

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Summary

We report the case of a patient who, following a right thalamic haemorrhage, developed thalamic syndrome characterised by burning pain and hyperalgesia in the left side of the body. Three years later, following a further (contralateral ischaemic) stroke, she reported the complete disappearance of the pain and hyperalgesia. To our knowledge, this is the first described case of disappearance of thalamic syndrome following a second stroke, different in nature from and contralateral to the first. Various hypotheses, based on the nervous tracts and nuclei involved in pain processing, may be advanced to explain this occurrence.

KEY WORDS: spino-thalamic pathways, stroke, thalamic (central) pain, thalamo-parietal radiations.

Introduction

Thalamic syndrome occurring after stroke, first fully described by Déjérine and Roussy in 1906 (1), includes diffuse, paroxysmal, often burning pain, contralateral to the lesion(s), hyperalgesia (overreaction to noxious stimuli), and allodynia or dysesthesia (painful sensation in response to non-noxious stimuli). Cerebrovascular disease is the most common cause of thalamic syndrome, other causes being tumours, multiple sclerosis and traumatisms. We report the case of a woman affected by thalamic syndrome after a right thalamic haemorrhage who reported the disappearance of the pain following a left, ischaemic stroke.

Clinical case

F.M., a hypertensive woman aged 66, had an acute left hemiparesis with mild-moderate motor impairment, hypoesthesia and paraesthesias (tingling sensation). Brain CT scan showed a right thalamic haemorrhage with surrounding oedema (Fig. 1). Ectopic beats and atrial fibrillation were observed on ECG, which disappeared after a few days (stroke-induced arrhythmias). After admission to a neurological ward she remained fully alert and cooperative, reporting only an increase of the left paraesthesias. She was discharged after 7 days. Once at home, she began to complain of spontaneous or touch-induced pain in her left limbs, sometimes reported as burning and excruciating. She also began to experience non-noxious stimulus-induced pain. Carbamazepine was administered up to 800 mg/die, producing only partial relief of symptoms. At subsequent examinations over the next three years, she continued to complain of the same type of pain, reporting a reduction only of the hypoesthesia. She had some hypertensive episodes, which were accompanied by a worsening of the pain. Then she suddenly presented a speech disturbance with poor fluency, and mixed (but predominantly motor) aphasia was diagnosed. Brain CT scan showed a fronto-parietal ischaemic lesion and bilateral lacunar infarcts (Fig. 2, see over). After some months, complex partial seizures occurred, which were stopped by the addition of phenobarbital (100 mg/day). Following this second stroke, and right up to her death (from myocardial infarction) three years later, the patient no longer reported either spontaneous or induced pain, or painful paraesthesias.

Figure 1 - Brain CT scan showing a right thalamic haemorrhage surrounded by hypodense area.
tral pain, a hypothesis that its disappearance following the structures mainly involved in the genesis of cerebral cortex around the central sulcus appear to be the primary sensory cortex (10), which may be hyperactive, so affecting pain processing. The increased activity in this cortical area, combined with decreased activity in the sensory thalamus, as observed in an rCMRGlu study (9), seems to be a distinctive feature of central pain. When hyperactive nociceptive neurons within the sensory cortex are damaged by lesions to the parietal cortex, contralateral to damaged thalamus (as in our case), these lesions could, given the bilateral connections of the spino-thalamic tract (11), at least partially explain the disappearance of central pain in our patient.

Discussion

In patients with stroke-related thalamic syndrome, lesions commonly affect the posterior part of the ventral medial nucleus (the area specific for pain and temperature) involving the territory of the geniculo-thalamic artery, more frequently in the right hemisphere (2). Several hypotheses have been proposed to explain the mechanism(s) underlying thalamic (central) pain: “irritation” of sensory fibres ending in the ventro-posterior nucleus (1), loss of the modulatory gate of sensory afferents following lesions of the thalamic ventro-basal complex (3,4), spino-thalamic deafferentation or a loss of lemniscal control over afferences (5), sensory spino-thalamic deficit (6), lemniscal deficit (7). The literature contains only a few reports of patients with stroke-induced central pain whose pain disappeared following a second stroke. The case reported by Soria and Fine (8) describes a patient, aged 62, affected by right hemiparesis and hypesthesia following a left lacunar infarct; after 7 years he had a second ischemic stroke affecting his left parietal region and resulting in right hemiparesis and aphasia. His pain disappeared after the second stroke, and the authors attributed this to destruction of the thalamo-parietal radiations. A further case was highlighted by Hirato et al. (9), who described 9 patients with central pain after stroke, submitted to CT, MRI and PET investigations. In one of them, who had a putaminal lesion, a small subcortical haemorrhage near the cerebral cortex around the central sulcus accidentally occurred during the recording operation. The subsequent disappearance of the pain in this patient was attributed, by the authors, to secondary cortical damage.

In these cases, thalamo-parietal radiations and areas of the cerebral cortex around the central sulcus appear to be the structures mainly involved in the genesis of central pain, a hypothesis that its disappearance following damage to cortical areas and their radiations to the thalamus, would seem to confirm, while also indicating a practice that might be applied in order to improve or remove the pain. But in our patient, in whom it was the contralateral parieto-thalamic pathways that were damaged, the disappearance of the central pain is not easy to explain. A pain-inhibiting mechanism driven by non-noxious information, which usually exists at cortical level, is lost in patients with central pain; non-noxious information also facilitates the activity of nociceptive neurons in the primary sensory cortex (10), which may be hyperactive, so affecting pain processing. The increased activity in this cortical area, combined with decreased activity in the sensory thalamus, as observed in an rCMRGlu study (9), seems to be a distinctive feature of central pain. When hyperactive nociceptive neurons within the sensory cortex are damaged by lesions to the parietal cortex, contralateral to damaged thalamus (as in our case), these lesions could, given the bilateral connections of the spino-thalamic tract (11), at least partially explain the disappearance of central pain in our patient.

References


Figure 2 - Brain CT scan showing hypodense area in the left rolandic region. Old, small lacunar infarcts in ganglionic area of both hemispheres.