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Clinical Note

Unilateral Thermal Anaesthesia

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Summary

A patient is described who had a deep parieto-temporal haemorrhage. Following resolution, the patient exhibited contralateral hemi-anaesthesia limited to the temperature sense. The possible mechanisms are discussed.

Introduction

The somatosensory deficits resulting from lesions of the parietal lobe are manifested usually as contralateral anaesthesia, particularly involving discriminative aspects. Disturbances of pain and temperature sensation, if they occur, are minimal, and selective involvement of the thermal sensitivity was to our knowledge exceptionally mentioned [3]. This paper describes a patient in whom somatosensory loss resulted from a parieto-temporal haemorrhagic infarction. After its resolution, the lesion manifested with contralateral thermal anaesthesia alone.

Case

This male was hospitalized at the age of 55 for a sudden left hemiparesis. The patient had no history of cardiovascular disease and the general examination was normal. He was mildly disoriented with left hemiparesis including the face, left hemianopsia and hemihypaesthesia for deep and superficial sensitivities.

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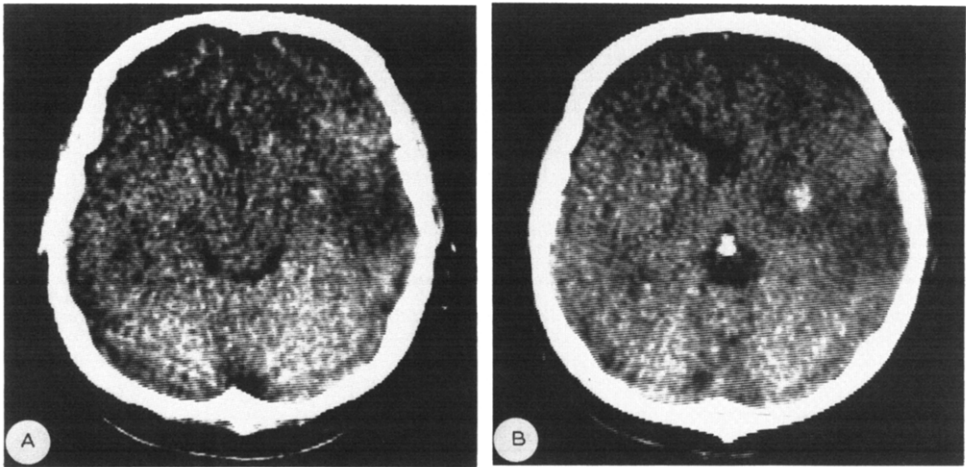


Fig. 1. Non-enhanced brain CT performed 24 h after onset of symptoms. Note high density area (probably blood), with more peripheral hypodense area in the right parietal lobe, and compression of the lateral ventricle.

The EEG showed mild slowing in the anterior and central right hemisphere. Computed tomography (Fig. 1) revealed a haemorrhagic infarction mainly affecting the right parietal lobe.

The evolution was uneventful and after 2 weeks the patient was transferred to a rehabilitation department, where his improvement continued. The patient was discharged 1 month after the acute event and was repeatedly seen in the outpatient clinic. He reported continued motor improvement and resumed his work as an engineer. However, he noted difficulty in appreciating temperature with his left limbs, for example when touching cold objects or getting into a hot tub with his left foot.

Repeated examinations several months to 3 years following the acute lesion revealed a patient who was well oriented with a relative left hemianopsia, relative neglect of the left hand and hypoexpressivity of the lower half of the left side of the face; brisk deep reflexes and a Babinski response on the left side. His speech was normal, but aprosodic. There was no constructional apraxia.

Sensory testing in the following modalities was normal and symmetric: tactile sensitivity with the cotton wool, including topognosia; superficial and deep pain; vibration sense; joint position sense and joint directional kinaesthesia of the fingers and the toes; two point discrimination on the finger tips and hands; stereognosis; graphaesthesia on the palms; directional cutaneous kinaesthesia; unilateral tactile extinction to bilateral simultaneous stimulation with non-painful and painful stimuli.

Thermal anaesthesia was noted on the left part of the face, in the left limbs, and on the left side of the body with sensory loss diminishing gradually over the midline. Cold stimuli were better appreciated than warm ones and the painful character of excessive thermal stimuli was correctly perceived. A repeat CT demonstrated absorp-

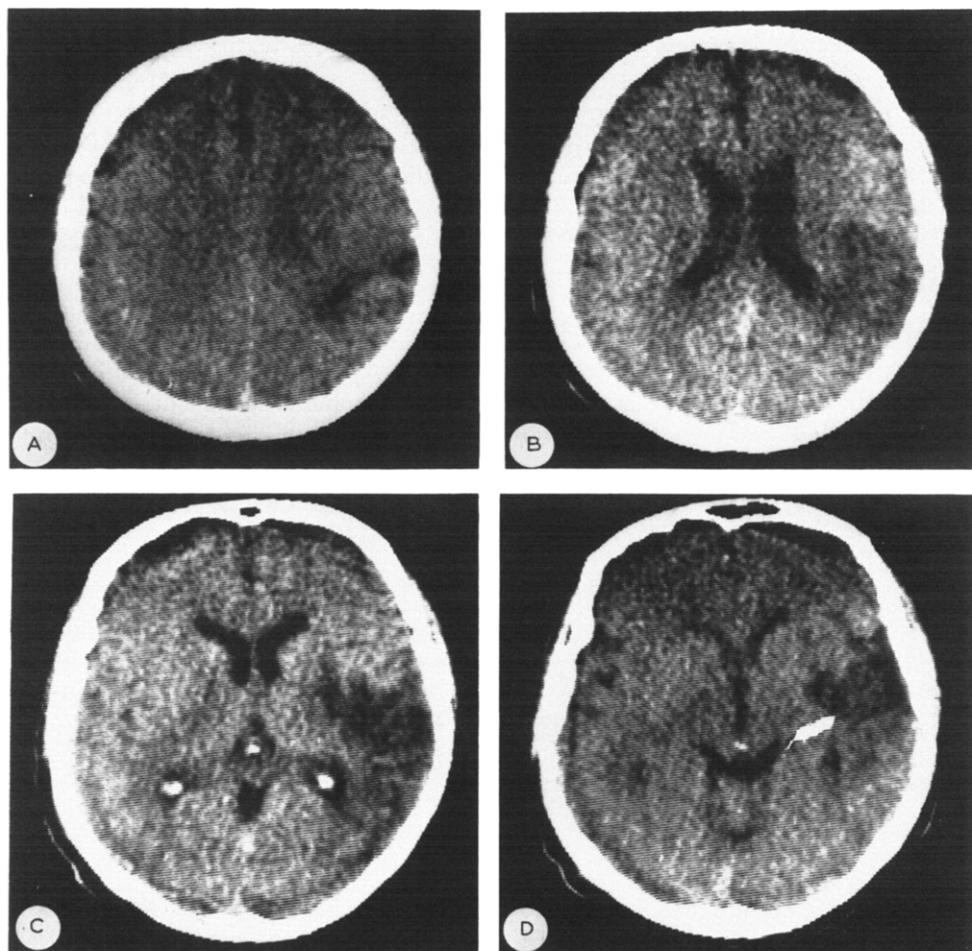


Fig. 2. Non-enhanced brain CT repeated 8 months after the stroke. Note disappearance of high density, with extension of the hypodense area, showing the infarction to involve peripheral and deep structures of the parietal and temporal lobes on the right. The lateral ventricle is widened on the affected side. The lesion was thought to be due to occlusion of the middle cerebral artery.

tion of the blood with an extensive area of damage in the depth of the right hemisphere, including the parietal and temporal lobes, and impinging on the thalamus (Fig. 2).

Discussion

The restriction of the somatosensory loss to the thermal sense in our patient is unusual and deserves some consideration. We could find just one previous reference

to selective loss of thermal sensation, in a careful study by Marshall [3] of a subject with 3 parietal wounds, two of them superficial. When tested years after the trauma, this patient manifested almost total loss of the thermal sense contralateral to the lesion, while other modalities, including superficial and deep pain, were intact.

The pathways mediating temperature and pain sensation travel together in the spinothalamic tract. It is unclear, however, whether projection to the parietal cortex is obligatory for nociceptive sensation. Most authors consider the thalamus as the site where appreciation of pain occurs. On the other hand, temperature sensation is a discriminatory function, necessitating relatively fine comparisons and gradings. It is thus expected to be represented at the post-central gyrus. Consistent with this analysis is the fact that our patient correctly identified nociceptive qualities of extreme thermal stimuli. Some evidence exists to suggest close, but separate representation of touch and position sense as opposed to temperature sensation, the latter being projected into the post-central gyrus in the depth of the central sulcus [2]. Since in our patient there is no evidence of selective cortical damage, a lesion of the afferent thalamo-cortical pathway is a more likely site of the lesion, but pertinent anatomic data on the pathways mediating temperature as opposed to touch and position are not available.

While this analysis emphasizes anatomic considerations, it should be recalled that following acute lesions involving the post-central gyrus, the improvement of the sensory deficit is said to have a temporal sequence, in which pain sensitivity is the first to return, followed by the tactile, with temperature sense being the last to reappear [1]. The basis of this functional difference is unknown. Our patient may represent an extreme example of this phenomenon where temperature sensation failed to recover.

References

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