

Focal luxury perfusion with an early-filling vein in relation to neurological symptoms evoked by heat

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Summary. A 50-year-old man with a 1-year history of transient attacks of left total hemiparesis was admitted to hospital with a complaint of increasing frequency of attacks. Minimal or moderate left hemiparesis was elicited by elevation of environmental temperature when taking a hot bath or a hot shower. Right carotid angiography revealed an early-filling vein near the right central sulcus. An increase of focal luxury perfusion by elevation of body temperature seemed to cause relative ischaemia in this paracentral gyrus.

Key words: Transient ischaemic attack – Luxury perfusion – Body temperature

Introduction

It is well known that neurological deficit is elicited by elevation of environmental temperature in some patients with multiple sclerosis [2, 4, 7]. Such a phenomenon is rarely observed in patients with other diseases [4]. We report a patient with small focal luxury perfusion in the cortical motor area showing contralateral hemiparesis during the elevation of environmental temperature.

Case report

On 6 January 1982, a 50-year-old man first had an attack of left total hemiparesis; this neurological deficit disappeared completely 2 days later. Plain computed tomography of the brain revealed no abnormality. Six months after the first attack he was admitted with increasing frequency of transient attacks of minimal left hemiparesis, each continuing for about 2 h. His transient neurological deficit was sometimes noted three times per day and usually occurred when taking a hot bath or shower. He therefore usually took a bath or a shower with lukewarm water. Examination revealed a mild cardiac systolic murmur, suggesting a congenital atrial septal defect. His blood pressure was 160/70 mm Hg with no orthostatic hypotension and his pulse rate was 88/min and regular. Neurological examination showed slight shallowness of the left nasolabial fold. A "hot bath test" was refused by the patient. However, a "hot shower test" gave a positive result; several

minutes after taking a hot shower, he showed minimal left total hemiparesis and increased muscle stretch reflex in the left upper and lower extremities. The grip power of his left hand decreased from 55 kg to 11 kg. Dysaesthesia and hypalgesia in the left face and left upper extremity were also noted. Systemic blood pressure did not change during this test. These neurological signs disappeared several hours after the hot shower. Examination by cardiac catheterization showed passage of the catheter across the atrial defect, but no right-to-left shunt through an atrial septal defect. Echocardiogram revealed no abnormality other than the atrial septal defect. Laboratory examinations, including cerebrospinal fluid, gave no abnormal results. Electroencephalogram was normal. CT scan of the brain was also normal, but in the right carotid angiogram an early-filling vein was seen near the right paracentral sulcus (during a non-symptomatic period; Fig. 1). Anticoagulant therapy with aspirin or medication with vasodilators did not decrease the frequency of ischaemic attacks.

Discussion

In 1937, Simons [7] originally reported that heat and cold might influence certain symptoms in patients with multiple sclerosis. In 1951, Guthrie [2] reported that elevated environmental temperature may induce changes in visual and motor function in patients with multiple sclerosis, suggesting that this phenomenon may be due to changes in blood circulation in the central nervous system during heating. Many authors have reported similar phenomena in patients with multiple sclerosis [3]. In 1973, Rasminsky [6] reported that elevation of temperature caused reversible conduction block at identifiable internodes of experimentally demyelinated rat ventral root fibres. He suggested that neurological deficit in patients with multiple sclerosis during elevation of environmental temperature may be due to the direct effect of elevated temperature on the affected demyelinated fibres [4]. In a similar way, intermittent claudication during exertion is induced in some patients with arteriovenous malformation [1]. The mechanism of this phenomenon is thought to be stealing of arterial blood flow to other regions [5–7]. In this report we describe a patient with intracerebral small localized luxury perfusion in the right paracentral sulcus, showing focal neurological deficits corresponding to the affected area, after a hot shower. The mechanism that causes this phenomenon may be similar to that in



Fig. 1. Late arterial phase of the right carotid angiogram showing early-filling vein (*arrow*) in the region of the right central sulcus

patients with arteriovenous malformation. Increased luxury perfusion by elevation of environmental temperature must cause relative ischaemia of the surrounding cerebral cortex. Presumably this lesion is not congenital because no neurological deficit was found before the first attack at the age of 49 years. The reason for the first ischaemic attack is uncertain. We consider that it might have been caused by embolism because this patient had a congenital cardiac anomaly, with no arteriosclerotic change, confirmed by angiography. In general, a capillary blush and an early-filling vein have been detected by angiography in the acute phase in 36% of patients with recanalization of occluded arteries and in cerebral neoplasms [8–10]. Neither intracranial neoplasm nor new attack of cerebral infarction were present in this case. Thus we consider that this lesion may have persisted after the first hemiplegic attack in the region of the occluded and recanalized artery. There seems to have been no previous report of a patient whose neurological instability during the elevation of environmental temperature was caused by the focal luxury perfusion in the cerebral cortex.

References

1. Beraud R (1972) Vascular malformations of the spinal cord. In: Vinken PJ, Bruyn W (eds) *Handbook of clinical neurology*, vol 12. North-Holland, Amsterdam Oxford New York, pp 548–555
2. Guthrie TC (1951) Visual and motor changes in patients with multiple sclerosis. A result of induced changes in environmental temperature. *Arch Neurol Psychiatry* 65:437–451
3. Irino T, Taneda M, Minami T (1977) Angiographic manifestations on post recanalized cerebral infarction. *Neurology (Minneapolis)* 27:471–475
4. Kurtzke JF (1970) Clinical manifestations of multiple sclerosis. In: Vinken PJ, Bruyn W (eds) *Handbook of clinical neurology*, vol 9. North-Holland, Amsterdam Oxford New York, pp 161–216
5. Mossy J (1976) Vascular diseases of the spinal cord. In: Baker AB, Baker LH (eds) *Clinical neurology*, vol 3. Haper & Row, Hagerstown, p 11
6. Rasminsky M (1973) The effects of temperature of conduction in demyelinated single nerve fibres. *Arch Neurol* 28:287–292
7. Simons DJ (1937) A note on the effect of heat and cold upon certain symptoms of multiple sclerosis. *Bull Neurol Inst NY* 6:385–386
8. Taveras JM, Gilson JM, Davis DO, Kilgore B, Rumbaugh CL (1969) Angiography in cerebral infarction. *Radiology* 93:549–558
9. Taveras JM, Wood FH (1976) Carotid angiography. In: Taveras JM, Wood FH (eds) *Diagnostic neuroradiology*, 2nd edn. Williams and Wilkins, Baltimore, p 650
10. Waxman SG (1981) Clinicopathological correlations in multiple sclerosis and related diseases. *Adv Neurol* 31:169–182

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