Relapsing depression in paramedian thalamic infarctions

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Depression has recently been associated with lesions in the CNS, particularly with large infarctions in the cerebral hemispheres. We report a patient in whom two episodes of acute depression were related to relapsing paramedian thalamic infarctions, which were accompanied by additional transient mild neuropsychological deficits, hypersomnia and a discrete sensory disturbance of the left face. Thalamic infarctions have been shown to mimic a variety of higher functional deficits, such as aphasias, apraxias and attentional disorders, traditionally associated with hemispheric strokes. We conjecture that the paramedian thalamic infarctions observed in our patient have in a similar manner been responsible for the transient depression.

Keywords: Depression – Infarction – Stroke – Thalamus

INTRODUCTION

Although Bleuler (1916) long ago expressed the view that “there is indubitably a primary inclination toward depression and anxious interpretations of experience, and often toward oppressive feelings, that is quite frequently a direct consequence of disturbances of blood circulation of the brain”, depression has generally been viewed as a disorder reactive to the consequences of CNS injury, than directly caused by it. Only recently has depression been found to be preferably linked to strokes in specific vascular territories of the cerebral hemispheres (e.g. Folstein et al., 1977; Robinson et al., 1984; Sinyor et al., 1986; Landis and Regard, 1988). Thalamic strokes, on the other hand, have in recent years increasingly been shown to produce neuropsychological deficits, such as aphasia, apraxia or attentional disorders, previously thought to occur only subsequent to lesions in the cerebral hemispheres at specific sites (e.g. Metter et al., 1981; Wallesch et al., 1983; Graff-Radford et al., 1985; De Renzi et al., 1986; Skyhøj et al., 1986; Perani et al., 1987; Stuss et al., 1988; Vallar et al., 1988; Schneider et al., 1991). Patients with paramedian thalamic infarction usually show impairment of consciousness, hypersomnia, neuropsychological deficits and ocular motor disorders affecting especially the vertical gaze (Castaigne et al., 1981; Gentilini et al., 1987; Bogousslavsky et al., 1988). Motor weakness, ataxia, delayed abnormal movements and hypoaesthesia involving the upper part of the body may also occur. We report a patient with recurrent paramedian thalamic infarctions, who developed a relapsing depression of acute onset.

CASE REPORT

During recovery from viral pneumonia, this 47-year old woman noted an acute change of personality with depressive mood, anxiety, phobic attacks, slowed thinking, inability to concentrate, decreased energy, withdrawal from usual activities, diminished sexual interest, reduced appetite and constipation. She also had problems with learning and memory, and an increased total sleep time of about 2 h. With the exception of a history of cigarette smoking (39 packyears) and a daily consumption of about 25 g alcohol over the last 20 years, her medical and psychiatric history were unremarkable. Three months later she was given a tetracyclic antidepressant (maprotilin hydrochloric) and a benzodiazepine (lorazepam) and 7 months later neurological examination was normal with the exception of a hypaesthetic area over the left cheek. Neuropsychological examination revealed mild disturbances in learning and recall, more marked for verbal than for figural material and mildly impaired concept identification and interference suppression. Parasagittal and axial cerebral MRI (1.5 Tesla; spin echo; slice thickness 8 mm) showed in T1- and T2-weighted images (relaxation time 510 and 2050 ms, respectively), echo time 16 and 100 ms, respectively) a lesion in the perfusion territory of the right paramedian thalamic arteries (Fig. 1a, b). The maximal infarcted volume was between 101 and 120 mm³. Pulsed-wave Doppler and Duplex sonography of the carotid, vertebral and subclavian arteries and transcranial Doppler examination of the intracranial vertebral, basilar, posterior, middle and anterior cerebral arteries and carotid...
siphons were normal. An electroencephalogram showed beta and episodic bitemporal theta activity with some interspersed alpha transients. Except for mild hypercholesterolemia, all laboratory tests including CSF examination were normal. In the following months the patient subjectively recovered completely, but 12 months later the same symptoms recurred acutely. Neurological examination showed again only an area of diminished sensation over the left face. Neuropsychological examination demonstrated unchanged mild memory disturbances for verbal and figural material, a marked reduction in figural fluency (Regard et al., 1982) but an excellent verbal fluency. Para-
sagittal and axial cerebral MRI (Fig. 1c-f; 1.5 Tesla; spin echo) disclosed an increased maximal infarcted volume of the above-mentioned paramedian thalamic lesion to 154-170 mm$^3$ in T1- and T2-weighted images (slice thickness 5 mm and 4 mm, relaxation time 500 and 2000 ms, echo time 15 and 100 ms, respectively). ECG and echocardiogram were normal, as was the coagulation status, anti-nuclear-antibody-, anti-DNA and anti-cardiolipin-titres were negative.

About 10 months after the second stroke the patient was free of any symptoms.

**DISCUSSION**


**REFERENCES**


