Abrupt onset of disturbed vigilance, bilateral third nerve palsy and masturbating behaviour: a rare presentation of stroke

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The clinical presentation of stroke usually includes sensory-motor impairment, cranial nerve palsies, or cognitive dysfunction. Disorders in behaviour are less frequently seen. The case of a patient with a very disturbing presentation, which included a disturbance in vigilance, bilateral third nerve palsy and masturbating behaviour, is presented. The topography of the lesions and its implications on the deficits observed are discussed.

A 48-year-old woman was admitted to the hospital because of an acute disturbance in vigilance. The patient's medical history included gastro-oesophageal reflux disease, Ménière's disease, breast cancer 2 years before admission, and chronic mild mood depression. Her current medications included amitriptyline (20 mg/day), tamoxifen citrate (20 mg/day), and lansoprazole (30 mg/day).

On admission her temperature was 37.5°C, blood pressure was 133/75 mm Hg and heart rate was 84 beats/min; the remaining findings of the physical examination were within normal limits. The heart sounds were normal, with no murmur, gallop, or rub. The pulmonary examination revealed no jugular venous distension and the breath sounds were equal bilaterally. The electrocardiogram was in normal sinus rhythm.

On neurological examination, the patient was in a coma (Glasgow Coma Score 7 (E1V1M5)). There was no loss of motor function and the deep tendon reflexes were present. There were no signs of pyramidal irritation. The ocular examination showed bilateral third nerve palsy with non-reactive mydriasis.

The disturbance in vigilance progressively improved during the next 7 days, although the bilateral third nerve palsy persisted. Disturbingly, the patient developed motor stereotypes concerning her upper (crumpling) and lower limbs (rubbing on the floor). In addition, she developed uncontrollable masturbating behaviour. When she was asked why she did this, she answered "I don't know why, I can't do otherwise". Although she had a slowing of her mental functions, her language was clear and the patient did not present any gross deficits in her cognitive abilities (a complete neuropsychological evaluation was impossible because of bilateral ptosis and severe oculomotor palsy).

Brain imaging (magnetic resonance imaging) showed bilateral thalamic infarction (fig 1A). No embolic source was found on cardiac, cervical and transcranial ultrasound.

DISCUSSION

We report the case of a patient who presented with disturbing sexual behaviour following coma and oculomotor

palsy, related to an embolism in paramedian arteries of the thalamus.

There are four major thalamic vascular territories, each with a predilection for supplying particular groups of nuclei: the tuberothalamic, the inferolateral, the posterior choroidal and the paramedian vessels. The paramedian arteries arise from a short portion of the posterior cerebral arteries situated between the basilar bifurcation and the junction with the posterior communicating artery (also called the P1 section). To a variable extent, they supply the thalamus which is principally constituted by the dorso- and posteromedial nuclei, the internal medullary lamina, and the intralaminar nuclei. In some cases, a single paramedian artery supplies both sides. When this anatomical variation is present, stroke results in a bilateral paramedian thalamic infarct (fig 1A), the classical features of which include deep coma, abnormal eye movements, and cognitive and behavioural impairment.¹ Coma results from the discontinuance of ascending fibres originating in the reticular formation which is involved in arousal mechanisms. Abnormal eye movements result from infarction in the midbrain nucleus which is usually associated (fig 1B). When the decrease in the level of consciousness resolves, cognitive and behavioural changes become more apparent: disorientation, Korsakoff-like amnesia, apathy, loss of psychic self-activation, dysinhibited behaviour, and aggressiveness. Amnesia results from injury to the Papez circuit, which includes the mamillothalamic tract and the anterior nucleus of the thalamus. Behavioural disturbances are explained by discontinuance of the thalamocortical fibres, especially when the dorsomedian nucleus, which massively projects onto the frontal cortex, is involved.²

Disorders of sexual behaviour after injury to the brain are infrequent and usually occur following lesions in the

Figure 1 Magnetic resonance imaging-T2 axial slice through the thalami (A) and the midbrain (B) and a diffusion-weighted MRI (not shown) showing a hypersignal compatible with a recent infarct in the territory of the paramedian arteries.

amygdala, the hypothalamus or the temporal or frontal circuits. They have rarely been described after bilateral thalamic infarction and usually occur in hypersexuality.³ Masturbating stereotypes have occasionally been reported and are associated with frontal hypoperfusion.⁴

This case report involving the prevalence of behavioural disorders underscores the possible heterogeneity which can be seen in this syndrome. Since patients often initially present with the abrupt onset of coma, bilateral thalamic infarcts are usually referred to the intensive care unit and early diagnosis is

difficult. Recognising these behavioural disorders as symptoms of focal brain infarction is important in terms of the diagnostic management and prognosis.

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Images in emergency medicine

Traumatic diaphragmatic rupture: a diagnostic challenge in the emergency department

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A 24-year-old man presented to the emergency department after falling down from the fourth floor of a building on a construction site. Upon arrival he was alert, but dyspnoeic. The breath sounds over the left

side of the chest were decreased. A chest radiograph was taken and revealed elevation of the left side of the hemidiaphragm (fig 1). Computed tomography of the chest was arranged to determine the severity of the lung injury (fig 2).

The incidence rates of traumatic diaphragm rupture is between 0.8–1.6% of patients admitted because of blunt trauma.¹ Left-sided rupture is more common than right-sided rupture (68.5% vs 24.2%).² Hepatic protection of the right side, increased strength of the right hemidiaphragm, and weakness of the left hemidiaphragm at points of embryonic fusion all contribute to the predominance of left-sided diaphragmatic rupture.³ A delayed diagnosis of diaphragm rupture and its associated injuries to the chest wall, abdominal cavity, and pelvic cavity can result in life-threatening sequelae. Surgery should be performed as soon as the clinical diagnosis is made, either through the transabdominal approach or via thoracotomy.

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Figure 2 CT scan showing herniation of intra-abdominal organs into the left thoracic cage.

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Figure 1 Chest radiograph showing an arch-like curvilinear density indicating elevation of the left hemidiaphragm.