

Catatonia following biparietal infarction with spontaneous recovery

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Summary: We describe a case of catatonic stupor following simultaneous biparietal infarction. The patient recovered, a result not previously described in catatonia caused by this pattern of cerebral infarct.

Introduction

Catatonia is well recognized in association with organic brain conditions as well as with schizophrenia and the affective disorders. We report a patient displaying all the features of catatonia, namely waxy flexibility, bizarre posturing, stupor, mutism, and negativism, following spontaneous biparietal infarction. A previous report¹ has described a patient who, following biparietal infarction, developed incomplete features of catatonia which did not recover. Our patient made a spontaneous recovery from his catatonic state, suggesting that the prognosis for this condition following biparietal infarction may be better than reported.

Case report

A man aged 72 years returned home from a short visit to the launderette in a state of confusion and agitation. Perplexed, abusive, and shouting for his wife, who was present, he was admitted to a psychiatric hospital. There he refused to answer any questions, but his wife and general practitioner indicated that he had no past medical or psychiatric history, was on no medication, and had sustained no head injury. He was noted to be in uncontrolled atrial fibrillation, and the blood pressure was 160/100 mmHg. While he resisted systematic neurological examination, no abnormality of limbs, eyes, or cranial nerves was apparent.

He refused all food and drink, and a week later had developed a right middle lobe pneumonia. Transferred to a general hospital, he gave a superficial impression of unconsciousness, but resisted purposefully any attempt to feed or examine him. A computed tomographic brain scan (Figure 1) showed areas of recent



Figure 1 Computed tomography of head showing bilateral parietal occipital infarction.

infarction in both posterior parieto-occipital regions, principally in the watershed areas between the middle and posterior cerebral artery supply areas. Electroencephalography gave only modest non-specific abnormalities, while visual evoked responses showed intact visual pathways.

The pneumonia responded to intravenous antibiotics within 5 days, and he returned to sinus rhythm, but remained mute apart from incomprehensible muttering. He continued to resist feeding, and pulled out his intravenous and nasogastric tubes. He refused to open his eyes or mouth when asked, and attempts to move his arms or legs, or open his eyes and mouth, were forcibly resisted. Waxy flexibility of his arms was present. He adopted strange postures, sitting upright

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in bed holding his arms up like flexed birds' wings. Over the following 6 weeks he slowly improved to the point where he would speak rationally to his wife, but his behaviour was disinhibited. He would grab at the nurses, refuse to comply with instructions, and showed wide swings of mood and emotion. At the end of 3 months he was eating and drinking normally, and no longer showed signs of catatonia. His personality remained infantile, and neuropsychological assessment revealed amnesic confusion combined with both expressive and receptive forms of dysphasia. He returned home to be nursed by his wife.

Discussion

Catatonia has both organic and psychiatric causes. In a series of 25 cases of catatonia,² 36% were found to have schizophrenic or affective illness, 20% had a probable organic cause, and in 40% no organic or psychiatric cause was found. Cerebrovascular disease is a well described precipitant: subarachnoid haemorrhage,³ cortical venous thrombosis,⁴ thrombotic thrombocytopenia purpura,⁵ subdural haematoma,⁶ and cerebral infarction¹ have been implicated.

Tippin and Dunner described a 45 year old man with catatonia following biparietal infarction. He was withdrawn and mute and maintained a bizarre posture, sitting rigidly in a chair leaning towards the right. This patient showed no 'waxy flexibility'; that is to say it was not possible to place his limbs and body in positions which he would then maintain. The catatonia did not improve despite treatment with electroconvulsive therapy and haloperidol. Most of the patients described by Barnes *et al.*² either recovered spontaneously or following electroconvulsive therapy. They found those with an acute and rapidly progressive course, 'acute lethal catatonia', to have the worst prognosis, 3 out of 4 dying of acute renal failure. Persistence of the catatonic state as described by Tippin and Dunner may, however, be atypical. The recovery demonstrated by our patient is more in accord with the result of Barnes' series, and suggests that the prognosis for recovery from catatonia following biparietal infarction may be good.

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