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Posterior Cerebral Artery Territory Infarct Presenting as Acute Psychosis

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ABSTRACT

An 82-year-old man with a past medical history of hypertension was admitted to a psychiatric hospital for sudden onset of acute psychosis. He was then transferred to an acute geriatric unit for further evaluation. During the admission the patient was noted to be very restless, agitated and noisy and was shouting and screaming incessantly. This was interspersed with occasional short periods of calm and quiet. Clinically, no obvious focal neurological deficits were detected. A CT scan of the brain was performed and it revealed an acute infarct involving the area supplied by the left posterior cerebral artery. This was a rather atypical presentation for an infarct involving this area.

Keywords: acute confusional state, elderly, stroke, organic, psychiatry

INTRODUCTION

The association between cerebral infarcts and acute confusional state has been observed since the 1980s[1,2]. Infarctions in the territory of any of the three large cerebral vessels can cause an acute confusional state with or without focal neurological deficits.

Involvement of the anterior cerebral artery can present with abulia, akinetic mutism, agitation or delirium[3,4], while a middle cerebral artery territory infarct can present with sudden agitation, inattentiveness, incoherent speech and disorientation[5].

Infarcts of the posterior cerebral artery (PCA) territory commonly present with visual or sensory deficits; there may also be ataxia, hemiplegia, hemiballismus and other brainstem signs if the occlusion is more proximal. Reports of altered mentation associated with PCA infarcts first surfaced in 1893 when Dejerine and Vialet reported a patient who presented with paranoid behaviour, hallucination and denial of blindness[6]. Since then, several authors have reported cases of PCA territory infarcts presenting as acute psychosis[7-10].

We present the case of an elderly man who developed acute psychosis after a left PCA territory infarct.

CASE HISTORY

An 82-year-old man who was a known hypertensive, was transferred to our unit from a psychiatric ward for acute confusional state. His only significant past medical history was that of a possible stroke in 1995 which was never investigated. He had presented with weakness of his right hand which resolved spontaneously after seeing a general practitioner.

His pre-morbid functional status was good. Up to the time of this admission, he had been working as a hawker and was totally independent in his activities of daily living. He lived alone in a one-room flat.

One week prior to admission, he was found by his son to be confused, restless and severely agitated. He was noted to be shouting and screaming at the top of his voice almost throughout the day and night, and he hardly slept. He was taken to a psychiatric hospital for admission by his son. Because of a low grade fever, he was transferred to our unit for further evaluation.

In the geriatric ward, his behaviour was observed to be very labile, swinging from being docile and quiet occasionally to being severely agitated, restless and noisy. These swings occurred rather suddenly and abruptly. He could be answering questions rationally at one moment but in the next moment he would suddenly break out loudly in a string of abusive words. He also displayed disinhibition and kept wandering about in the ward. The patient kept yelling for his daughter and a Chinese deity throughout the day and stayed awake on most nights. There were times when he appeared anxious and frightened and held on to whoever was standing nearby.

Physical examination was difficult. We did not detect any focal neurological weakness. Visual field examination could not be carried out. There was no clouding of consciousness but the patient was disorientated to time, place and person. There were no signs or focus of sepsis and all laboratory investigations were essentially normal except for a low serum vitamin B12 level (112 pmol/L). CT scan of the brain (Fig 1) showed a hypodense lesion in the left occipital lobe extending into the posterior part of the parietal lobe consistent with an acute PCA territory infarct. There were small and old infarcts seen in the left caudate nucleus, lentiform nucleus and the right external capsular region.

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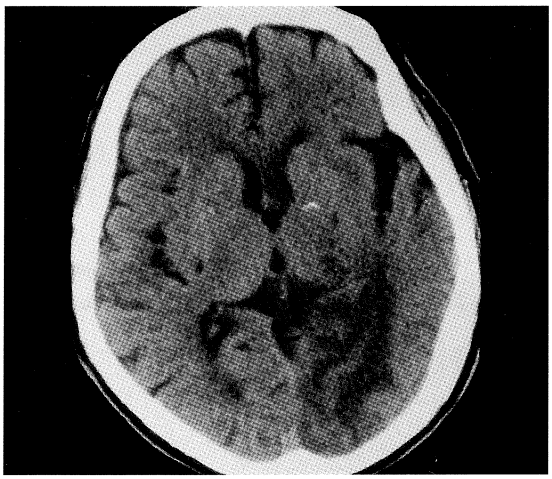

Fig 1 – CT scan of the head showing a hypodense lesion in the territory of the left posterior cerebral artery.

The patient was subsequently transferred back to the referring psychiatric hospital for further management of his disturbed behaviour, which was not controlled with haloperidol or thioridazine. He developed generalised tonic-clonic seizures and was transferred back to our department. These seizures were controlled with phenytoin and he was subsequently discharged with a follow-up appointment with the psychiatrist. His family had refused to have him transferred again to the psychiatric hospital despite his uncontrolled behaviour.

While at home, the patient continued to be agitated, restless and noisy. His family was unable to cope and finally agreed to readmit him to the psychiatric hospital. He stayed there for more than a month and was finally discharged with Risperidone, Lorazepam and Zudopenthixol.

His behaviour was more subdued although he still had intermittent episodes of yelling and screaming and remained disorientated to time, place and person.

DISCUSSION

In 1967, Horenstein et al reported 9 patients with PCA territory infarctions presenting with agitation, forced crying out and extreme distractibility with exaggerated response to any visual, auditory and tactile stimulus[11]. In 1974, Medina et al also reported a man who became blind, agitated, used foul language and struck, bit or spat on others after suffering a similar infarct[12]. In 1977, Medina et al again reported 3 patients who presented with similar traits[8].

Our patient's presentation is similar to that reported by the above authors. It is interesting to note that our patient's lesion was on the left side, corresponding to the same side as those reported by the above authors. The latter have observed that while lesions of the middle cerebral artery presenting with disordered attention were most often on the right side, it was more likely to be on the left side in PCA lesions. It is postulated that the aggressive behaviour results from disruption of the anterior limbic structures from the cortical and limbic inhibitory input[6].

A striking feature of this man's problem is the intractable nature of his behaviour change and the difficulty in controlling it. In the reports quoted above, it was noted that some of the patients with PCA territory infarcts developed psychiatric symptoms which were difficult to control and which persisted till death while others developed severe dementia.

Dunne et al, in their series, observed that 3% of patients with 'inobvious' cerebrovascular lesions actually presented with delirium, an organic delusional state, acute dementia or mania, mimicking psychiatric illnesses[13]. Our patient presented with an acute change of behaviour mimicking a psychiatric illness. There were no obvious neurological deficits detected and thus a stroke could have easily been missed.

The lesson that can be drawn from the above discussion is that a PCA territory infarct can present as an acute psychiatric problem especially in the elderly. Consequently, when an elderly patient presents with acute psychosis, even with absent or minimal neurological deficits, a CT scan of the brain should be considered in the work-up, not the least because of the possibility of a PCA territory infarct, but also because of the possibility of the many other intracranial pathology that can present in this manner. Moreover, an inadvertent discovery of a cerebrovascular lesion has implications with regard to prognosis and management.

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