Short Report

Catatonia with frontal lobe atrophy

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Summary A middle-aged woman with a four year history of behavioural change including episodes of catatonia is described. Despite a persistently abnormal electroencephalogram, the patient was diagnosed repeatedly as suffering from a primary psychiatric disorder. Neurological examination and psychological testing suggested frontal lobe dysfunction, which was confirmed by the finding of isolated frontal lobe atrophy on computed axial tomography.

Frontal lobe lesions can present with significant psychiatric symptoms which overshadow the neurological signs, which may lead to the initial misdiagnosis of a primary psychiatric disorder. This has been reported in cases in which frontal lobe dysfunction resulted from tumour, infarction or haemorrhage, trauma, or epilepsy. This paper describes a middle-aged woman with a four year history of behaviour change characterised by mood fluctuations, impaired judgement, and recurrent episodes of catatonia. The neurological examination, electroencephalogram (EEG), and psychological tests suggested frontal lobe disease which was confirmed by the findings of isolated frontal lobe atrophy on computed axial tomography (CAT).

Case report

Mrs A, a 53 year old college educated mother, had been active in community affairs until 5 years ago, 1 year after the onset of her menopause. She began an extramarital affair at that time, and her family members reported that her disposition changed from being reserved to voluble. She complained of frequent, sharp frontal headaches. During the next two months, there was a marked deterioration in her personal hygiene and housekeeping skills. Her sleep and activity patterns became irregular and she had increasing difficulty in making decisions. Medical evaluation revealed her to have a depressed affect without formal thought disorder or amnesia. Sleep and waking EEGs revealed bilateral frontal sharp waves and excess theta activity. An EEG obtained 5 years prior had been normal. Radioactive nucleotide brain scan and flow study were normal. She was diagnosed as having a psychotic depressive reaction and was started on chlorpromazine, with improvement in her affect and personal hygiene. As an outpatient, however, she discontinued her medication and refused further follow-up care. During the ensuing 4 years, the patient was hospitalised for episodes characterised by the patient sitting or standing mute in a public place for many hours. At times the patient spent prodigious amounts of money, and she became increasingly withdrawn from her family and friends. Her diagnosis included paranoid or catatonic schizophrenia, manic-depressive illness, and involutional melancholia. She always left the hospital against medical advice and refused follow-up care. The EEG remained abnormal on four separate occasions.

On transfer from a state hospital to our facility, she appeared unkempt, sad, and had little psychomotor activity. Her eye contact was poor. She had no insight into her problems, stating that her past problems were the result of a bad marriage. Neurological examination showed kinesthetic apraxia, and snout and grasp reflexes. An EEG showed bilateral frontal sharp waves, rare spikes, and excess theta activity. A CAT scan showed isolated bilateral frontal lobe atrophy without ventricular enlargement (fig).
Lumbar puncture revealed clear, colourless fluid with normal opening pressure, cell count, protein and glucose levels, and negative serology. On the Wisconsin Card Sorting Test, the patient failed four of six categories, due to perseveration. The patient did poorly on the picture completion and picture arrangement sections of the Weschler Adult Intelligence Scale, despite a verbal IQ of 130 and performance IQ of 127. The results of the Minnesota Multiphasic Personality Inventory and Rorschach tests did not suggest a primary psychiatric disorder.

On the 6th hospital day, she became mute, stared blankly and was unresponsive to her surroundings. She stood, shifting from foot to foot, or lay on the floor. Examination showed waxy flexibility, but normal cold calories and flexor plantar responses. She had a tachycardia (90-110/min.) and was hypertensive (150-165/95-100/mm Hg). The EEG was unchanged. A clinical diagnosis of a catatonic state was made, and she was treated with chlorpromazine intramuscularly, with excellent response. She was able to recall in great detail all that had transpired during her catatonic episode, and described this as a period in which she was "deep in thought and couldn't decide what to do". She was maintained on haloperidol 1 mg orally twice a day with continued improvement in her social interactions and personal hygiene. During the following year, the patient did not have any catatonic episodes; repeat EEG and CAT scan were unchanged.

Discussion

This patient's neurological examination, EEG, and psychological test results suggested that the patient had frontal lobe dysfunction, which was confirmed by the CAT scan finding of isolated frontal lobe atrophy. In addition, the EEG changed from being normal prior to the onset of her illness, to showing abnormalities indicating frontal lobe injury. Although various types of frontal lobe lesions have been reported to produce catatonia, catatonia has not been associated previously with isolated frontal lobe atrophy.

As noted by Leigh et al, catatonia can respond to standard therapy despite the presence of structural brain disease. Patients with frontal lobe atrophy have been noted to have paroxysmal periods of thought fixation, as this patient described during her period of catatonia.

The aetiology of this patient's frontal lobe atrophy is not clear. Patients with Pick's disease frequently present with behavioural changes, poor judgement, lack of insight and resistance to testing, and may go many years before memory and language deficits develop. In Van Mansvelt's review, 166 of 172 cases of Pick's disease showed a definite predominance of atrophy in either the frontal lobe, temporal lobe, or both. However, it would be unusual for a patient with Pick's disease of five years' duration to have as little deficit as this patient. McGeachie et al emphasise that the CAT scan in patients with Pick's disease shows enlargement of the frontal lobe atrophy. The ventricular system in our patient was normal in size. Infarction in the distribution of the anterior cerebral arteries could produce the frontal atrophy and kinesthetic apraxia seen in this patient, but it would be unusual without at least transient motor deficit.

This case illustrates that isolated frontal lobe dysfunction can produce profound psychiatric signs and symptoms, including catatonia, and that unless a careful neurological examination and the appropriate tests are performed, the diagnosis of frontal lobe disease may be missed. This case also emphasises the importance of pursuing the aetiology of focally abnormal EEGs in psychiatric patients.

References

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*J Neurol Neurosurg Psychiatry* 1980 43: 185-187
doi: 10.1136/jnnp.43.2.185

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