Bulimia in a patient with temporal lobe epilepsy

There have been no reports of focal neuroanatomical lesions associated with bulimia nervosa. The following is a case report of a patient with bulimia, who was found to have a lesion of the left temporal lobe on MRI during evaluation for a seizure disorder.

The patient was a 33-year-old right-handed female college administrator who had complained of fainting spells since the age of six. These had occurred at least once or twice a year and, after puberty, often five to seven days premenstrually. She came to neurological attention after having had three such spells in four months. During one of these recent episodes, rhythmic twitching of the right hand had been observed. She typically experienced an aura of a strong odour of ether lasting up to one minute before each event, followed by unconsciousness for about 30 seconds. Infractually she also had experienced nocturnal bladder incontinence as a child and as an adult.

An eating disorder with onset at the age of 28 had been diagnosed as bulimia nervosa. Since that time she had been having recurrent periods of binge-eating behaviour followed by self-induced vomiting which would last for up to a year, followed by periods of remission lasting four to six months.

Despite feeling loss of control over her eating behaviour, she was always aware of her actions and had never experienced anorexia, somnolence, or syncope during the binges or purges. She generally maintained her weight in the normal range of 55-60 kg with a maximum weight fluctuation of 14 kg. The severity of the behaviour had increased during the couple of months preceding the time of presentation.

There had also been alcohol abuse up until five years ago. There was no history of depression. The family history included the father who had alcoholism and a paternal cousin with multiple sclerosis.

Her neurological and general examination were normal. Her mood was not depressed. A sleep deprived EEG showed a burst of theta slow waves in the left temporal leads during the awake state. An MRI of the brain showed a lesion in the left temporal lobe adjacent to the medial tip of the temporal horn, to the region of the hippocampus. This was felt to represent a cystic dilatation of the ventricle or a small area of infarction (figure).

The patient was treated with carbamazepine. Despite 18 months of treatment, she has had no further olfactory hallucinations, seizures, or syncopal episodes. She has had one episode of nocturnal incontinence. Her eating habits have been under good control and her weight has been stable. There have been no eating binges, purging, or feelings of compulsion to fast or eat large quantities of food.

This case demonstrates a possible pathogenic relationship of temporal lobe disease to bulimia. Although the cause and pathogenesis of bulimia remain unknown, in addition to psychological factors, biological factors may also play a role in the disorder.

Hyperparathyroidism has been described in some neurological disorders including Huntington's disease, amygdalocephaly, frontal and hypothalamic tumours, frontal lobeotomy, Pick's disease, epilepsy, Kleine-Levin syndrome, and von Economo's encephalitis. Hyperparathyroidism in such patients, however, clearly differs from the binge-purge diathesis seen in bulimia. Furthermore, bulimic patients tend to be selective in their eating behaviour, unlike the Klüver-Bucy syndrome, and maintain a normal weight. Aside from the relatively late onset of bulimia, this patient displays the behaviour, feelings, and clinical course typical of the more specific disorder of bulimia nervosa.

Studies of patients with bulimia nervosa using CT have reported ventricular dilatation and sulcal enlargement; however, focal brain disease has not been demonstrated. MRI is a more sensitive technique for the detection of temporal lobe lesions in epilepsy, which could be of potentially greater yield in showing lesions in patients with eating disorders, as was seen in this case.

Epilepsy has yet to have an integrative role in forming the brain's body image via afferent information from secondary sensory areas and the limbic system. Structural or biochemical disease of this part of the brain could thus have a more role in understanding the distorted self image which occurs in bulimia.

Reports of EEG abnormalities in bulimia range from 16-64%, depending mainly upon the diagnostic criteria used. In a series of 23 patients with compulsive eating treated by Rau and Green, a positive response to phenytoin was correlated with those patients having a combination of abnormally low EEG plus other neurological symptoms such as dizziness or rage attacks. A modest response to phenytoin was seen in a later double blind, placebo controlled study of 20 patients; however, response was not correlated with pretreatment EEG abnormality. Subsequent case studies of phenytoin treatment were less positive, and enthusiasm for anticonvulsant treatment of bulimia waned.

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Non tumoural aqueductal stenosis with intermittent course. Case report after a six year follow up.

The aetiology of non tumoural aqueductal stenosis presenting in adults remains unknown in most cases.\(^1\)

Rarely it can occur either as a sequela of neonatal or infantile meningitis, congenital toxoplasmosis or viral infection,\(^2\) in a genetically determined pattern with an x-linked recessive genetic trait as part of a malformative syndrome.\(^3,^4\) It can also occur in a congenital form coexisting with other CNS malformations.\(^5\)

The clinical picture is extremely varied ranging from symptom-free cases, with hydrocephalus and aqueductal stenosis being an accidental necropsy finding,\(^6\) to a sudden onset of progressive neurological deterioration.\(^7\)

A minor skull trauma, a febrile illness, a subarachnoid haemorrhage, or even a lumbar puncture can precipitate an acute disturbance of cerebrospinal fluid dynamics with a complex clinical picture.\(^8\)

On 22 May 1981 a 12 year old male suffered a skull trauma without loss of consciousness or fractures and presented with a severe headache with nausea and vomiting lasting one week. CT scan showed a slight ventricular dilatation.

He presented with a parental migraine and a past history characterised by recurrent attacks of migraine-like headaches without aura. From the age of 13 the patient experienced episodes characterised by the sudden onset of headache, nausea, vomiting, sleepiness and gaze abnormality. These episodes lasted two to five days and occurred up to once a month. He had experienced nine episodes before admission.

On 30 March 1983 when he was 14 years he was admitted to the Neurological Department of Bologna University with headache, vomiting and stupor.

Neurological examination revealed defective upward gaze, skew-deviation and very sluggish-reacting mydriatic pupils. Routine blood and cerebrospinal fluid tests were normal. EEG showed a spindle coma pattern. CT scan with contrast enhancement demonstrated a conspicuous three-ventricular hydrocephalus (fig 1a).

On the fourth day pneumoencephalography confirmed the hydrocephalus and a subsequent ventriculography showed a hypertrophy of the massa intermedia in the third ventricle, which was dilated. Bilateral carotid angiography was negative. Sabin-Feldman dye test and serological test for cisticercosis were negative. Eight days after admission the patient was asymptomatic and the EEG was normal.

One month later, a CT scan demonstrated a slightly shrunken hydrocephalus (fig 1b). After being discharged, he experienced an initial three month symptom-free period followed by episodes at first once a month, then once every three months.

On 13 November 1984, MRI scans were performed during the acute event showing a remarkable three-ventricular hydrocephalus with an important aqueductal stenosis. Two months later the patient was asymptomatic.

Over the past four years the episodes have occurred as infrequently as once a year with a spontaneous remission. Currently, neurological examination and neuroradiological tests are normal. The last CT, performed on 12 March 1989, showed a slight ventricular dilatation.

Our patient showed transient signs and symptoms of aqueductal stenosis such as Parinaud's syndrome and disturbance of consciousness, directly related to an intermittent three-ventricular hydrocephalus.

After a minor skull trauma suffered at the age of 12 years, an intermittent clinical course started with recurring episodes with gradual spontaneous remission.

Neuroradiological studies over a period of six years performed during the acute phase and interictically, revealed an intermittent aqueductal stenosis.

The clinical course of non neoplastic aqueductal stenosis in adulthood is described as a chronic process with a slow or fast evolution. Cases with obstructive hydrocephalus and an intermittent clinical course were only seen with intermittent obstruction of the ventriculoperitoneal or ventriculo-atrial shunt,\(^7\) in spontaneous ventriculostomy\(^8\) and in Arnold Chiari malformation.\(^9\)

We agree with Williams\(^10\) who analysed the pathogenesis of benign aqueductal stenosis and stressed that hydrocephalus precedes and is usually the most important cause of the narrowing or blockage of the aqueduct.

Our patient had a cranial trauma precipitating an acute disturbance of cerebrospinal fluid dynamics. This factor together with slight physiological increases in intracranial pressure, usually well compensated, might cause a progressive enlargement of the ventricular system. As hydrocephalus progresses the third ventricle shows a tendency to enlarge progressively and the massa intermedia becomes stretched.\(^11\) A further increase in cerebrospinal fluid pressure in our patient could force open the aqueduct with the restoration of normal flow and rapid remission of the clinical symptoms.

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