benefit in the treatment of bulimia and deserves consideration for future placebo controlled study.

BRIAN R OTT
Department of Neurology,
Roger Williams General Hospital,
Brown University,
Providence, RI, USA


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 sequella of neonatal or infantile meningitis, congenital toxoplasmosis or viral infection, in a genetically determined pattern with an x-linked recessive pattern of inheritance as part of a malformative syndrome.2,3 It can also occur in a congenital form coexisting with other CNS malformation.4

The clinical picture is extremely varied ranging from symptom-free cases, with hydrocephalus and aqueductal stenosis being an accidental necropsy finding, to a sudden onset of progressive neurological deterioration.5

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.6

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 sluggishly reacting mydriatic pupils. Routine biochemical and imagiological 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 hypotrophy 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 every one or two 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 neuropsychological 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. Neurological 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 fast or slow evolution. Cases with obstructive hydrocephalus and an intermittent clinical course were only seen with intermittent obstruction of the ventricular system or ventriculo-atrial shunt, in spontaneous ventriculoloméy6 and in Arnold Chiari malformation.7

We agree with Williams8 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.9 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.

Correspondence to: Dr Sacquegna.