Case report

Hydrocephalus and bipolar affective disorder

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Abstract

Bipolar disorder can emerge in the context of organic brain pathology. In the case presented, long-standing hydrocephalus was diagnosed in a man with relatively late-onset bipolar illness who presented initially with somewhat atypical, treatment-resistant depressive symptoms. Hypomania, followed by a rapid-cycling bipolar course, subsequently developed. This report reviews the association between bipolar disorder and hydrocephalus, and examines possible neurobiological mechanisms implicated in both conditions.

Key words: Bipolar affective disorder; Hydrocephalus; Organic mood disorder; Neuroimaging.

Case report

A 46-year-old man was referred to a psychiatric outpatient clinic by his GP with a two-year history of low mood, decreased drive, emotional indifference, poor memory and attention, lack of motivation and reduced interest in previously enjoyed activities. Social withdrawal was prominent. There was no history of other biological features of depression.

Of note there were no depressive cognitions such as hopelessness, worthlessness, guilt, or suicidal thoughts. His functioning deteriorated and he was asked to take time off work. Possible identifiable precipitant factors appeared to be a number of bereavements in the family in the last couple of years.

Past history was notable for a brief admission to a psychiatric hospital for alcohol misuse 18 years previously and a subsequent episode of depression managed by his GP with lofepramine. He has been abstinent from alcohol for many years, and has no drug use history. He smokes 30 cigarettes a day.

Family history was notable for an uncle having been admitted for alcohol misuse in the past.

Developmental history was significant – he was born one month prematurely; treatment included oxygenation, and he was nursed in a neonatal unit for three months. He was a slow learner in school and had been diagnosed as having a mild intellectual disability. A history of rheumatic fever in late childhood, and mild hypertension in adulthood, are the only other significant aspects of his medical history.

His atypical depressive symptoms were resistant to several antidepressants of different classes, namely venlafaxine, citalopram, and mirtazapine (each for a sufficient duration of time and dosage).

As a result of the apparent treatment-resistant nature of his symptoms, and also the prominence of features such as bradyphrenia, abulia and apathy, his work-up was extended to include neuroimaging. A computed tomography (CT) brain scan showed long-standing marked diffuse enlargement of the lateral and third ventricles with a normal appearing fourth ventricle. There was no evidence on CT of raised intraventricular pressure. Clinical neurological examination was unremarkable. An extensive battery of blood tests which included full blood count, liver function tests, urea, creatinine, electrolytes, thyroid function tests, vitamin B12 and folic acid levels, revealed no abnormalities. A neurology opinion was sought, and the diagnostic impression was of old, established hydrocephalus, possibly as a result of infectious brain injury in the postnatal period (thought likely either encephalitis or meningitis). The hydrocephalus was judged unsuitable for surgical intervention.

Seven months after referral, he suddenly developed symptoms consistent with hypomania, including mildly elated mood, increased energy, overtalkativeness, overactivity, excessive spending and somewhat overfamiliar and disinhibited behaviour. Antidepressant therapy was stopped and his condition stabilised. However he subsequently developed a depressive illness lasting five months; this was followed by a sudden return to a brief (10-day) hypomanic episode. He has been euthymic for a number of months; current medications are quetiapine 300mg at night time, lamotrigine 50mg twice daily and aripiprazole 10mg once daily. He currently attends a day hospital and is planning to return to work.

Discussion

Hydrocephalus, or dilatation of the cerebral ventricular system, arises as a consequence of disturbance of the formation, flow, or absorption of cerebrospinal fluid (CSF). These changes lead to an increase in volume occupied by this fluid in the central nervous system.[1] Hydrocephalus is said to be 'communicating' if the obstruction is outside the ventricular system (usually in the basal cisterns). [1,2] In the case presented, the hydrocephalus is postulated to have arisen in infancy, possibly due to infectious brain injury resulting in damage to the choroid villi with subsequent impaired CSF re-absorption.

Normal-pressure hydrocephalus (NPH) was described in

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1964 and defined by the symptom triad: dementia, motor disturbance, and sphincter disturbance.[3] Enlarged ventricles and normal CSF pressure at lumbar puncture in the absence of papilloedema led to the term NPH. However, intermittent intracranial hypertension has been noted during monitoring of patients in whom NPH is suspected. Headache is not a typical symptom in NPH.[1] While the neurosurgical insertion of a ventricular shunt is the treatment of choice for hydrocephalus associated with raised intracranial pressure, there are no effective surgical or medical treatments for NPH.

A small number of case reports to date have described an association between hydrocephalus arising post head injury and rapid cycling bipolar affective disorder.[4-8] The available literature is suggestive of more likely a rapid cycling course of mood disorder of organic aetiology with a periodicity of days rather than weeks.[9] Some reports have suggested that resolution of the affective symptoms may follow successful surgical intervention.[10]

The condition was incidently found in relatively late onset bipolar affective disorder in an individual with no family history of bipolar illness. In a study of 27 patients, no demographical variables such as, age, sex, race and year of education were found to be associated with lateral ventricular enlargement in bipolar disorder. However, measures such as, more frequent hospitalisation and histories of persistent unemployment were found to be associated with lateral ventricular enlargement in bipolar disorder.[11]

The pathophysiology of psychiatric symptoms in hydrocephalus remains unclear. Changes in intracerebral pressure may have a role in precipitating neuronal damage and affective symptoms. This could arise either through localised pressure on structures adjacent to ventricles or pressure on other structures affected by generalised raised in intracranial pressure.[12]

Of note, white matter hyperintensities and lateral ventricular enlargement (reflecting reduced thalamic and hypothalamic volumes) are among the structural brain abnormalities frequently reported in bipolar disorder.[13] Abnormal structure or functioning of prefrontal and anterior cingulate cortical area and subcortical structures such as amygdala, thalamus, and striatum have been found to be associated with depressive disorder and bipolar affective disorder.[14,15]

Where bipolar disorder emerges with hydrocephalus, the damage to afore-mentioned structures could conceivably result from a localised or generalised rise of intracranial pressure.

Although it is tempting to ascribe causality of the affective illness course to the identified organic brain pathology, the possibility remains that hydrocephalus may represent an incidental finding with no causal role in psychiatric presentation. On the other hand it is also possible that underlying cerebral pathology could have contributed to the failure of conventional treatment and thus prompted further investigations. Another possibility, albeit unlikely, is that an underlying and undiagnosed bipolar disorder may give rise or contribute to the development of hydrocephalus (through an undetermined mechanism).

As such, this case also illustrates the value of neuroimaging in first-episode bipolar cases, particularly where an atypical presentation or treatment-resistance is evident. Although the hydrocephalus in the presented case was longstanding, nevertheless psychiatric symptoms associated with hydrocephalus may represent early signs of cerebral pathology and neuronal damage, and delay in diagnosis in such cases may lead to inappropriate treatment and potential brain damage.

Conclusion

This report adds to the literature on the association between hydrocephalus and bipolar disorder. It remains unclear whether the observed association between the two conditions reflects a causal relationship or simply represents an incidental finding. Nevertheless, this case highlights the importance of considering an organic aetiology, and neuroimaging studies, in first-onset bipolar cases.

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