Psychiatric disturbance was alluded to in Addison’s original description of pernicious anaemia, and nowadays the disorder is recognised as being associated with depression, mania, psychosis and dementia. It is also known that psychiatric symptoms can precede the onset of anaemia or neurological features and can sometimes be seen in the absence of macrocytosis. A case report has recently added catatonia to the list of psychiatric syndromes associated with B12 deficiency. Here we present a further case, where catatonia was the presenting – and only – psychiatric feature of what turned out to be autoimmune B12 deficiency. Haematological abnormality was absent and neurological abnormality was subtle to the point of being easy to dismiss.

Method

A 27-year-old single croupier and singer of mixed race was compulsorily admitted after she was found to be living in a flat with no heating and electricity. She had stopped working around a year previously and had become withdrawn. There was no previous medical or psychiatric history. In hospital she was noted to be unkempt and vague; she would often stare blankly into space, had no spontaneous interaction with others, and at times was doubly incontinent. However, no affective or psychotic symptoms were elicited. She was treated with olanzapine with slight improvement. She was discharged by a mental health tribunal and then became lost to follow-up.

She was readmitted 6 months later, again in a state of self-neglect. She was very slow to initiate movements, took a long time to get out of bed, and would stand in the same position for long periods in the ward, often with her arms folded across her chest. She also followed members of staff around the ward repeating what they said. During an interview, she spent the whole time with her arms folded, her head down, and a right upgoing plantar. She also had a positive Luria’s (fist-edge-palm) test. Cognitively, she scored 67/100 on the Addenbrooke’s Cognitive Examination–Revised (ACE–R)3 (normal range ≥82). Digit span was 4 forwards and 4 backwards. She showed borderline poor performance on story recall (6/23 idea units on the ‘Brian Kelly’ story from the Rivermead Behavioural Memory Test,4 normal range ≥6) and poor verbal fluency (five words beginning with P in 1 min). She was in the impaired range on two executive tests: the Hayling Test,5 which measures ability to inhibit prepotent responses (error score 43, scaled score 1), and the Brixton Test,6 a rule-shifting test conceptually similar to the Wisconsin Card Sorting Test (error score 39, scaled score 1).

Computed tomography (CT) scan was reported as showing mild frontal atrophy, but according to further neuroradiological opinions this was not unequivocally outside the normal range (Fig. 1). Magnetic resonance imaging was reported as showing only increased cerebrospinal fluid spaces around the hemispheres and increased third vertebral size. Single photon emission tomography scan was normal. Electroencephalogram (EEG) showed only bilateral posterior theta wave activity.

Because of the inconclusiveness of these findings, one of the authors (S.J.) undertook a raft of further investigations, including autoantibody screen, thyroid function tests, syphilis serology, serum B12 and folate, and voltage-gated potassium channel antibodies. The only abnormal finding was a low vitamin B12 of 159 ng/l (normal range 189–883 ng/l). Around this time her haemoglobin was 12.7–13.1 g/dl on different occasions, with mean cell volume (MCV) of 86–94 fl (normal range 86–98 fl). Subsequently, antibodies to intrinsic factor were found, and the diagnosis of pernicious anaemia was made.

Results

Olanzapine, which had been recommenced shortly after admission (in addition to diazepam), did not result in obvious improvement over 4 weeks. However, less than 10 days after starting on hydroxycobalamine (intramuscularly, at a dosage of 1 mg on alternate days for 10 days), nursing staff commented on a marked improvement in her behaviour and self-care. Her Addenbrooke’s score (on parallel versions of the ACE–R) was 80/100 2 weeks after commencement of treatment and 90/100 4 weeks later. Digit span after 10 weeks of treatment was 6 forwards and 5 backwards and she had improved into the ‘moderate average’ range on the Brixton test (19 errors, scaled score 5); however, she remained in the impaired range on the Hayling Test (33 errors, scaled score 1). As she began to talk more, she described having had pins and needles in her hands and feet for approximately a year, which could have reflected spinal cord demyelination. No abnormalities were found on repeat neurological examination. A repeat CT scan 4 months after institution of B12 replacement continued to show much the same degree of frontal sulcal widening, but was reported as normal.

She discontinued olanzapine 2 months after discharge from hospital. Four months later, after a 2-week delay in administration of her hydroxycobalamine injection, she had a brief, 2-week...
B12 deficiency can cause cognitive impairment up to and including reversibility.\(^7\) Brain changes associated with B12 deficiency are less variable, with 1 year having been suggested as a watershed for cerebral atrophy or leukoencephalopathy, again with varying levels of documentation, with different studies finding no abnormality, which then improved. This is inconsistent with schizophrenic psychosis for more than 2 years of illness. Second, she originally showed a substantial degree of cognitive impairment, which then resolved promptly to re-initiation of olanzapine. Two periods of catatonia, which resolved promptly to re-initiation of olanzapine. Currently, she is continuing to receive olanzapine and vitamin B\(_{12}\) injections, is out of hospital, lives independently in her own flat and is planning to enrol in a music course.

**Discussion**

This individual presented with marked social decline, self-neglect and later developed full-blown catatonic symptoms without evidence of psychotic or affective symptoms. Her neurological abnormalities were minimal, and whether there was any abnormality on brain imaging became the subject of debate among the clinicians and neuroradiologists involved in her case. Olanzapine brought about no improvement over 4 weeks but after its discontinuation, the patient went into a period of catatonia, which resolved promptly to re-initiation of olanzapine. Currently, she is continuing to receive olanzapine and vitamin B\(_{12}\) injections, is out of hospital, lives independently in her own flat and is planning to enrol in a music course.

This person's lack of anaemia and her (at most) subtle neurological signs meant that vitamin B\(_{12}\) deficiency was not originally screened for. In 1988, Lindenbaum *et al* questioned the existing orthodoxy that B\(_{12}\) deficiency only presented with neurological disorder when anaemia was also present.\(^9\) They reviewed 141 people who were given a final diagnosis of clinical B\(_{12}\)-deficiency-related neurological symptoms and found that 40 (28%) had either no anaemia or no macrocytosis at initial presentation, and that in 19 (13%) both haematological indices were normal. Six further individuals had anaemia, but with an MCV that was normal or low (in four of these there were independent causes for this). In some of these people diagnosis was delayed for several months or years.

This woman's case adds to the literature documenting the occurrence of catatonia in a wide range of systemic diseases, and supports the view that it is an independent psychiatric entity, not one that develops only in the context of a schizophrenic or affective syndrome.\(^6\) It also provides an argument that vitamin B\(_{12}\) deficiency should be considered in any person with an unusual psychiatric presentation, not only when this is 'organic', and regardless of the absence of macrocytosis or anaemia. This is particularly important since delayed diagnosis can be associated with lack of reversibility.

---

**Fig. 1**: Computed tomography scan.

---

**References**


---

**Acknowledgements**

We thank the patient for giving permission to publish this case report.

---

**Funding**

P.J.M. is supported by the Instituto de Salud Carlos III, Centro de Investigación en Red de Salud Mental, CIBERSAM, Intramural Research Program.

---

**Correspondence**

S. Jauhar, Hairmyres Hospital, East Kilbride, Lanarkshire, G75 8RG, UK. Email: sameerjauhar@yahoo.co.uk

**First received 12 May 2008, final revision 17 Feb 2010, accepted 28 Feb 2010**
Pernicious anaemia presenting as catatonia without signs of anaemia or macrocytosis
Sameer Jauhar, Allison Blackett, Pavan Srireddy and Peter J. McKenna
BJP 2010, 197:244-245.
Access the most recent version at DOI: 10.1192/bjp.bp.108.054072

This article cites 7 articles, 1 of which you can access for free at:
http://bjp.rcpsych.org/content/197/3/244#BIBL

To obtain reprints or permission to reproduce material from this paper, please write to permissions@rcpsych.ac.uk

You can respond to this article at /letters/submit/bjprcpsych;197/3/244

Published by The Royal College of Psychiatrists