The patient that changed my practice: A rare case of organic psychosis

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This article presents a patient with a history of depression, progressive impairment of brain functions, diarrhoea and faecal incontinence. A cobalamin deficiency, without megaloblastic anaemia was detected, and an abnormal Schilling test not due to intrinsic factor deficiency was obtained. Once other causes of cobalamin deficiency were ruled out, it was considered caused by blind-loop syndrome. Treatment with vitamin B complex and long-term oral antibiotic therapy led to a complete resolution of neurological and digestive symptoms. This case illustrates B12 deficiency as a possible cause of reversible dementia, as well as the importance of considering B12 screening in a patient with a history of gastric surgery. Given the depressive history of the patient, the case also emphasizes the relevance of distinguishing a dementia from the cognitive symptoms of depression. (Int J Psych Clin Pract 2003; 7: 147–150)

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INTRODUCTION

Approximately 5–10% of dementias are wholly or partially reversible. This case illustrates the importance of B12 screening in a psychiatric patient with a history of gastric surgery, known to have suffered from recurrent depressive disorder in the last 12 years, presenting with mixed neurological and psychiatric symptoms (affective symptoms and symptoms of dementia).

CLINICAL CASE

The case of a 64-year-old single man, the elder of two brothers is described. The patient, a Spanish farmer, turned to the emergency service, accompanied by a member of his family, who reported a cognitive deterioration that had started approximately 1 year ago. Disorientation, a staggering walk and stereotyped movements had been observed, especially during the past 4 months. He occasionally showed episodes of temporary disconnection (10–15 min) characterized by an inability to undertake normal activities, word repetition, mutism and a fixed look. After these episodes, the patient presented with retrograde amnesia, although he succeeded in connecting with the previous conversation. He was a heavy smoker suffering from hypertension, and had no history of substance abuse.

Depressive symptoms had been first observed 40 years ago. Six years later, the patient had been admitted to hospital suffering from a serious depressive episode. He had undergone several treatments with intravenous clomipramine during the past 12 years showing a slight, although sustained, improvement. His relatives reported spells of “scrupulousness and obsessions”. Furthermore, we know that as a child he had lost consciousness and rapidly recovered; in addition, we identified an affective disorder in a second-degree relative.

The patient had undergone a partial Billroth II gastrectomy for a duodenal ulcer with incipient penetration into the pancreas, approximately 10 years ago. From then on he had noticed changes in his intestinal habit. At the time of observation, he presented with chronic diarrhoea. During the previous year he had been suffering from faecal incontinence.

Psychopathological exploration made upon arrival at the hospital documented a spatially disoriented patient. Slight response latency and a coherent, although digressive, impoverished and sometimes persevering, speech was observed. He looked worried, anxious and lacked insight about his general condition. He suffered from amnesia of recent and remote events, and had behaviour disorders (running

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away from home, taking off his clothes). He experienced low mood (without daily fluctuations), thoughts of disability, hopelessness, anxiety and affective lability. He presented with little appetite and suffered from initial insomnia. No obsessive or phobic symptoms, or sensoperceptive disorders, were evidenced.

Neurological exploration highlighted a discreet asymmetry in the finger–nose test, slight left ptosis, facial hypomimia, slight grasping and moderate axial and distal bradykinesia. Throughout the physical exploration he looked perplexed for a few seconds, became mute and did not react to simple orders. Some minutes later he started babbling a few words, regained consciousness and went on with the examination. Since these episodes (similar to a complex partial crisis) occurred repeatedly, dialogue with the patient turned out to be very difficult in later interviews.

NEUROPSYCHOLOGICAL TESTS

MINI-MENTAL STATE EXAMINATION (MMSE):
Score of 23/30 (cognitive deterioration).

BENTON:
Five correct answers expected and three obtained D-2; eight errors expected and 12 obtained D-4.

Figure 3 is complete; in Figure 7 there is a perseverance of the right peripheral; in Figure 8 there is an omission of right peripheral, and in Figure 9 there is an error in the size of the left peripheral. Therefore, we can appreciate an unacquired deficit in short-term memory.

RAVEN:
Score 87 (normal-low).

MMPI:
Within normality with signs of meticulousness, perfectionism and scrupulousness.

COMPLEMENTARY EXAMINATIONS

EEG: No anomalies at rest or under activation were screened.

CT: CT exam demonstrated a hypodense area in the protuberantial region without signs indicating the presence of any expansive process in the posterior fossa. The supratentorial exam revealed a moderate enlargement of the ventricular system, ruling out other signs of an expansive process or even the possibility of a subdural collection.

MRI: The MRI exam confirmed the lesion determined in the CT study. A high-intensity signal lesion was demonstrated affecting the protuberance, internal capsule and globus pallidus. No signs indicating mass effects were observed.

The supratentorial exam only revealed the presence of cortical atrophy. The differential diagnosis of the infratentorial lesion should include central pontine myelinolysis (CPM) or even an ischaemic injury (Figure 1).

LABORATORY FINDINGS

VDRL and VIH were negative. Thyroid hormone (TSH 0.49), folic acid level (5.9), glycaemia on empty stomach (88), haemogram (leukocyte 8.800, erythrocyte 4 610 000, haemoglobin 15.1, haematocrit 44.6, mcv 96.8, mch 32.7, platelets 342 000) urine test (urine density 1025 and ph 6.5, unaltered sedimentation rate), VSG (5–11), creatinine (1.1), urea (0.3), hepatic functioning tests (SGOT 37, SGPT 34, alkaline phosphatase 153, gamma GT 28, indirect bilirubin 0.55, direct bilirubin 0.15, total bilirubin 0.70), electrolyte balance (sodium 138 mmol/l, potassium 4 mmol/l, chlorine 103 mmol/l, calcium 9.5 mmol/l, magnesium 1.8 mmol/l), osmolality (285) and lumbar puncture were all within normal rate.

Vitamin B12: 182 (normal > 210).
No megaloblastic anaemia or reticulocytes were found in the haemogram.

GASTROENTEROLOGICAL OPINION

Several tests were applied, as a diagnosis of a malabsorptive pattern was considered. The D-xylose test revealed malabsorption, and the Schilling test excluded intrinsic factor deficiency. A bacterial overgrowth syndrome was suspected, as a post-surgical complication of the duodenal ulcer operation.
DIAGNOSIS
The diagnosis was dementia caused by B12 deficiency (ICD-10: F02.8) due to bacterial overgrowth by blind-loop syndrome after gastrojejunostomy.

EVOLUTION
Once the clinical diagnosis was confirmed, a substitutive treatment with vitamin B12 complex and parenteral thiamine was started, and tetracyclines were later added to the treatment. A dramatic improvement in orientation, memory and praxia was observed from the very first day. Difficulties in calculations and memory persisted. Three months later, the patient regained sphincter control and could lead an autonomous life. Psychopathological exploration showed better behaviour, coherent speech and better orientation. Restitutio ad integrum without after-effects was achieved in 6 months.

DISCUSSION
This case is considered to be of an extreme didactic relevance for two main reasons. Firstly, it highlights the importance of recognizing and distinguishing the manifestations of dementia from the cognitive symptoms of depression. Especially when, as is the case here, we are studying a reversible dementia. On the one hand, complaints of cognitive loss, progressive cognitive deterioration for over 6 months, disorientation, amnesic disorders, low general executive functioning, and anomalous MRI in a patient who is more than 60 years old lead us to think of a dementia diagnosis. However, the depressive mood of the patient, his family and personal history, the rapid progress of the disease, fluctuations in the deterioration and clinical symptoms seemed to fit a depressive pseudodementia pattern. Secondly, the case illustrates the clinical characteristics of dementia caused by vitamin B12 deficiency. It is known that vitamin B12 deficiency may be manifested as dementia, depression or anergia before the onset of pernicious anaemia. It has been estimated that 70–75% of pernicious anaemia patients have objective memory impairment and that 60% have abnormal electroencephalograms.

Even if there is vitamin B12 deficiency, the determined values were close to the lowest value within the range of normality. However, the low B12 levels should not be considered a datum against a neurological affection due to cobalamin deficiency diagnosis, even if there are no other haematological alterations. Although the illness is somewhat unusual, we should bear it in mind in patients having undergone surgical digestive interventions.

Central pontine myelinolysis is frequently associated with deficiencies of thiamine (B1), riboflavin (B2) or with a rapid correction of hyponatraemia. No hyperaemia or oedema of nasopharyngeal mucosa was observed, nor cheilosis, seborrhoic dermatitis or angular stomatitis, which could point to a riboflavin deficiency. Sodium levels were within the normal range (138 mmol/l). No nystagmus, ophthalmoplegia or fever was observed. The patient had no heart disease, which is also common in a wet beriberi. Since he did not have cardiovascular involvement (such as peripheral vasodilatation, oedema or biventricular myocardial failure) and the common neurological features in beriberi (peripheral neuropathy, Wernicke’s encephalopathy or Kosakoff syndrome) were not found when he was examined, a vitamin B1 deficiency does not seem to be the case in this patient. The pattern was resolved without vitamin B2 administration to the patient. Therefore, we consider that the cause of the deterioration was not the lack of this vitamin but the lack of vitamin B12.

This is a case of special interest, as it illustrates the importance of considering the organic aetiology in a patient with a clinical history of depression. In the case described, this consideration was useful to make us pay thorough attention to somatic symptoms (diarrhoea and faecal incontinence), apply the suitable tests (the importance of checking serum B12 levels), reach an accurate diagnosis, administer the appropriate treatment, and achieve a total recovery of the patient.

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KEY POINTS
- Blind-loop syndrome after gastrojejunostomy is a possible cause of B12 deficiency
- Vitamin B12 deficiency can be mainly manifested as psychiatric symptoms before the onset of pernicious anaemia
REFERENCES