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A Case of Schizophrenia Complicated in Subacute Combined Degeneration: How Nutrition Affects Physical Health in Psychotic Patients

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ABSTRACT

Vitamin deficiencies are not very common in western high-income countries. However, psychiatric patients, especially those affected by Schizophrenia, frequently do not follow a healthy lifestyle. This can cause deficiencies.

We present the case of a 35-year old woman suffering from treatment-resistant disorganized Schizophrenia. The clinical picture, initially identified as a possible resurgence of the underlying pathology, was further complicated, leading clinicians to suspect first Neuroleptic Malignant Syndrome, and subsequently encephalomyelitis. Improvement of the clinical picture through deeper investigation highlighted a condition of sub-acute myelin degeneration on a dysmetabolic-deficiency basis; thus modifying the initial diagnosis from psychotic decompensation to the more correct diagnosis of neuro-psychiatric symptomatology due to lack of vitamin B-12.

In our case, vitamin deficiency mostly gave rise to neuropsychiatric symptoms, such as altered state of consciousness, agitation and accentuation of somatic delusions. These symptoms exacerbated the severe food restrictions in the patient, which worsened the deficiency and psychopathological situation. Vitamin deficits often remain silent for a long time, progressing in pathologies that are generally reversible but which, if not caught in time, can face further complications, making it difficult to trace their origin. It is therefore essential, in psychotic patients, to constantly investigate eating habits, and also to remember not to exclude a constant blood-chemical monitoring of the parameters that account for the nutritional status of the patient.

Keywords

Hypovitaminosis, Schizophrenia, Subacute Combined Degeneration.

Introduction

In order to function properly, the Central and Peripheral Nervous System need an appropriate and constant intake of nutrients: an important role is played by Vitamin B12, also known as Cobalamin [1]. Deficiency of this vitamin is responsible for multiple neurological syndromes; some of which are milder, characterized by headache, exhaustion, drowsiness, reduced concentration, and others more severe, such as dementia, peripheral neuropathy and sub-acute combined degeneration. Psychiatric manifestations related to this deficit are also frequent, precocious, and generally non-specific. They include mood changes accompanied by depressive symptoms, apathy and irritability; psychomotor changes with agitation or catatonia; involvement of consciousness and the ideological-perceptive sphere, possibly with delirium, delusions and hallucinations [2]. The neuropsychiatric symptoms described above may precede the most characteristic hematological changes of the hypovitaminosis under examination (such as megaloblastic anemia) even by many years [3]. However, mental symptoms could be undervalued.

In western high-income countries, this type of deficit may be common among the elderly, in post-gastric resection surgery patients, and in people with intrinsic factor deficiency with vitamin B12 malabsorption [4]. However, the literature shows that psychiatric patients may also have low levels of Cobalamin [5], although other studies indicate a lower prevalence in psychiatric patients than in the general than healty controls [6]. Psychiatric

patients, especially those suffering from Schizophrenia and other psychoses of the Schizophrenia spectrum [7], frequently do not follow a healthy lifestyle and are more likely to be sedentary, to consume tobacco, caffeine and high-calorie foods excessively, at the expenses of adequate food requirements, a higher vitamin and trace elements intake; this predisposes them to clinical conditions, even serious ones, based on deficiency [8]. In this kind of patients, therefore, we need to differentiate the exacerbation of psychopathology from any symptom found in an innate pathology attributable to a wrong diet. Hence, we present the case of a young schizophrenic woman who silently developed a Sub-acute Combined Degeneration.

Case Presentation

The case presented concerns a 35-year old Caucasian woman suffering from treatment-resistant disorganized Schizophrenia (ICD-9 code: 295.10), treated with Clozapine 250 mg/day for more than two years. No known drug allergies, nor internal comorbidities were reported. In June 2018, the patient was admitted to a Low-intensity Psychiatric Unit, following the worsening of her basic psychopathological condition. Figure 1 [9] describes briefly patient's symptoms and diagnostic tests over the time. Her clinical features were social withdrawal, gear changes with falls, and a somatic delusion for which the patient believed she had insects and cockroaches inside her stomach. Mild normo-cytic and normo-chromic anemia resulted from the blood chemistry tests, while haemocoagulation, liver and kidney function were normal; Clozapinemia was also evaluated, it was 1,220 ng/mL (range: 150-300 ng/mL). In consideration of the high level of Clozapinemia and the state of the patient, Clozapine was gradually stopped and replaced with Aripiprazole. In the following days, however, a worsening of psychiatric symptoms was observed, with the appearance of auditory hallucinations, memory alterations, imbalance and worsening of gait alterations. There was also a significant increase in Creatine phosphokinase (CPK: 1,431 U/L, range: 69-190 U/L) and Myoglobin (MB 380 ng/mL, range: 0-85 ng/mL), without additional suggestions of Neuroleptic Malignant Syndrome (NMS). The antipsychotic therapy was therefore suspended; subsequently, we observed the worsening of the psychiatric symptomatology with appearance of psychomotor agitation. It was therefore decided to transfer the patient to a Hospital-intensive Psychiatric Unit. Blood chemistry tests showed an increase in CPK to 25.769 U/L, without fever or muscle stiffness; in a few hours the clinical features worsened rapidly, with moments of alternating agitation and a reduction in the state of consciousness, up to progressive enticement and a state of coma. The patient was therefore transferred to a Semi-intensive care Unit, where she was stabilized and monitored in vital parameters and subjected to a brain CT, which resulted negative for ischemic and hemorrhagic lesions, and Electroencephalogram (EEG), which showed diffuse delta activity. After the improvement of the state of consciousness and the autonomic control and laboratory values (CPK 400 U/L and Myoglobin in range), the patient was finally transferred to a Neurological Unit, for diagnostic analysis and specialist evaluation. In suspicion of an autoimmune or infectious encephalitic form, a lumbar puncture was performed with cytochemical examination, as well as Link index, oligoclonal bands analysis, bacteriological and virological examination for neurotropic viruses, with negative results. Additionally, an autoimmune profile was performed on serum and liquor of the antibodies antiGAD, antiVGCK, NMDA, AMPA and GABA, which were also negative. Moreover, a full blood examination was performed, which showed a deficiency of vit. B9 (2.1 ng / mL, range: 3-17 ng/mL) and vit. B12 (120 ng/L, range: 180-914 ng/L). The deficiency status was investigated through the patient's mother (caregiver), who reported a diet based almost exclusively on refined carbohydrates for years. Finally, RMN brain, without and with contrast, was performed (Figure 2) [10]: the latter revealed a widespread hyperintensity of signal in the T2 weighted sequences, extended in the posterior cordon from C1 to the first viewable back metamers. This finding was compatible with a condition of sub-acute myelin degeneration on a dysmetabolic-deficient basis: therefore, a therapy was started with Thiamine 500 mg/day, Cyanocobalamin 1000 mg/day, Folina 5 mg/day, and, in consideration of the protracted immobility of the patient, Enoxaparin 4000 IU/day. Following the introduction of the therapy, a partial resolution of clinical features was found, with an improvement in the state of consciousness and gradual recovery of mobility, which allowed to detect a stenic and proprioceptive deficit in the four limbs, in line with the signal alteration highlighted cervical MRI.

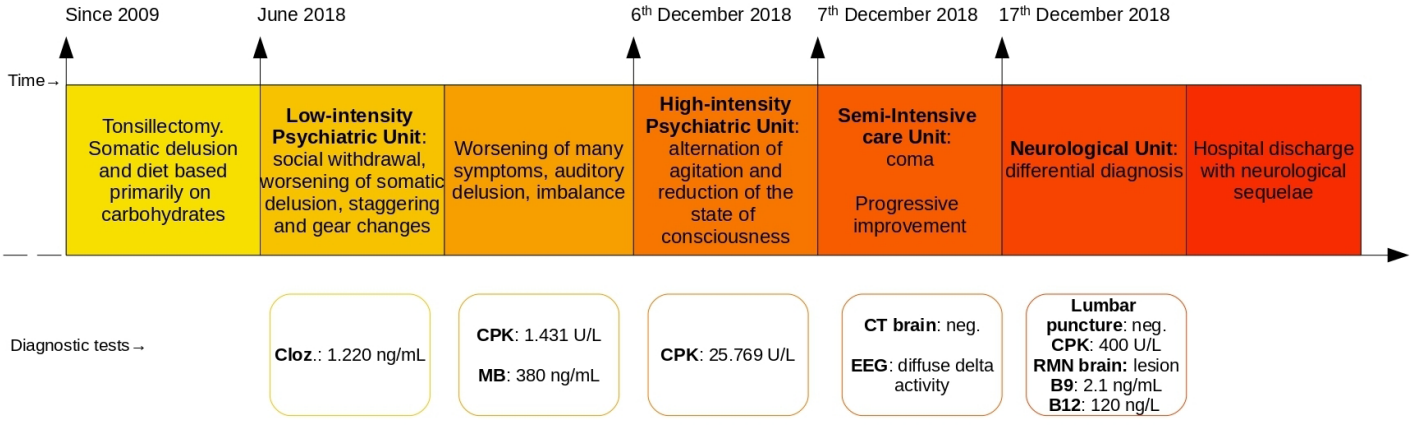

Figure 1

Figure 2

Discussion

We have described the case of an encephalopathy developed as a consequence of vitamin B12 deficiency in a patient with known disorganized Schizophrenia (ICD-9 code: 295.10). The patient's information is adequately de-identified. In this case the differential diagnosis was complicated and included neurological conditions, such as NMS, autoimmune or infectious encephalitis, deficiency diseases and resurgence of an already known psychiatric pathology. Several morbid pictures concerning psychiatric patients are frequently underdiagnosed, because erroneously traced back to a known psychiatric pathology [11]. In this case, the patient had presented gear changes with falls, social withdrawal and somatic delusion for months: these symptoms caused the suspicion of an exacerbation of the basic psychopathological picture, which was the reason why the patient was admitted to a Low-intensity Psychiatric Unit. The anamnestic connection verified with the patient's mother, who is also her caregiver, showed that somatic delusion had accompanied the patient for several years, ever since she underwent a tonsillectomy (2009). Since then, the woman had become convinced of emanating bad smell from her throat and her mouth. Indeed, the delusion added further somatic elements, mainly related to the gastrointestinal system. In particular, the belief of having cockroaches inside her stomach affected her food selectivity. In fact, the patient for years had chosen to feed almost exclusively on carbohydrates and immediate high-gratification foods, thus severely limiting the consumption of foods with vitamin content, such as vegetables, fruits and fish. In the Psychiatric Unit, clinicians evaluated the diagnostic hypothesis of NMS because they found some compatible elements. For example, they considered the reduction of motility, the worsening of agitation and alterations to her state of consciousness, and detected frequent falls, autonomic alterations such as tremors and lower blood pressure, as well as a noticeable increase in the CPK level (up to 25.769 U/L). However, she did not show some typical features, albeit not necessary ones, such as hyperthermia and muscle stiffness [12]. The suspicion of encephalitis depended precisely on the fact that frequently it manifests with changes in the level of consciousness and are associated with fever, dizziness, changes in gear or focal neurological deficits. Encephalitis is diagnosed through lumbar puncture, but imaging and electroencephalography can help. The real challenge for clinicians is to differentiate the causes of encephalopathy, including septic, metabolic and toxic ones [13].

The extension of the blood chemistry test to the vitamin structure highlighted an important deficiency of vit. B12 (120 ng/L) and vit. B9 (2.1 ng/mL), attributable to the patient's strong food selectivity. Vit. B12 deficiency generally has a slow and silent onset, however, the clinical consequences can be multiple and serious, affecting different systems, including the blood and lymphatic system, the neurological system and the gastrointestinal system. Macrocytosis, neutrophil hypersegmentation, anemia, leukopenia and thrombocytopenia might occur; the nervous system, on the other hand, is mainly affected by myelination disorders that can, in the long run, generate irreversible neurological damage; at gastric level, Vitamin B12 deficiency can generate neoplastic lesions [14].

There is no univocal chronology of clinical manifestations. In the case described above, vitamin deficiency mostly gave rise to neuropsychiatric symptoms such as alterations of the state of consciousness, agitation and accentuation of the somatic delusions, which worsened the severe food restrictions in the patient, limited to refined carbohydrates only.

Finally, what emerged from the RMN brain was primary, as it showed a widespread signal hyperintensity in the T2 weighted sequences, extended from C1 posterior cordon to the first visible back metamers. This finding was compatible with a myelin degeneration on a dysmetabolic-deficient basis. Sub-acute Combined Degeneration of the spinal cord is a progressive and potentially reversible degeneration of the white matter of the spinal cord posterior and lateral cords, due to a vit. B12 deficiency [15]. It mainly involves the posterior spinal cord and lateral tracts: the main clinical manifestations are neuropathies and peripheral paresthesia, proprioception alterations, gear changes and cognitive deficits (16). Rarely, dementia might develop. Healing is more likely if Sub-acute Combined Degeneration is treated promptly. If treated within a few weeks since symptoms onset, most people recover completely. If treatment is delayed, the progression of symptoms may be slowed down or stopped, but the complete recovery of lost functionalities is less likely. Massive oral doses of vit. B12 can be administered in case of mild deficiency and whereby specific symptoms of neurological damage do not appear.

In the case described, unfortunately, the vitamin replenishment and the resolution of the deficiency framework occurred late; in fact, after the improvement of the state of consciousness and the gradual resumption of mobility, a stenic and proprioceptive deficit in the four limbs was found. This corresponds clinically to the altered neuroradiological picture shown in RMN cervical.

Conclusion

This case allows stressing an important concept: patients affected by psychiatric pathology are often underdiagnosed, as their comorbidities are traced to their basic psychopathology. Also, patients often follow an unhealthy diet and clinicians tend to investigate it only when deficiency states are already established. Generally, vitamin deficits remain silent for a long time, gradually giving rise to pathologies that are generally reversible but which, if not caught in time, might lead to complex clinical conditions, whose origin can be difficult to trace back. In fact, vitamin B12 deficiency is easily concealed in a patient with a psychiatric history, since this vitamin deficiency can generate a vague syndromic picture that can be confused with a basic psychopathological re-exacerbation. It is therefore critical not to neglect the anamnestic evaluation of the patient's eating habits, which must be consistently investigated by the attending clinician. It is also important to remember to include in the blood-chemical monitoring those parameters that may be relevant to highlighting a deficient picture. Finally, clinicians may consider preventive replacement treatments in their practice.

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9. Figure 1, Timeline of symptomatology development and diagnostic tests.
10. Figure 2, RMN T2 weighted sequence that showed myelin degeneration on a dysmetabolic-deficient basis.
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