PSEDOATETOSIS – AS PRESENTING SYMPTOM OF BIERMER ANEMIA

Adina Roceanu, Monica Rosoiu, Ina Cristian, O. Bajenaru
Neurology Department, Emergency University Hospital, Bucharest, Romania

ABSTRACT

We present the case of 69 years old female with untreatated arterial hypertension, with previous lacunary strokes in the last year, admitted in our department for unsteadiness of the gait and involuntary movement – of the upper limbs (left > right) – started a few days ago, after an emotional stress. We diagnosed a subacute combined degeneration (SCD) of spinal cord due to vitamin B12 deficiency, a Biermer anemia. The involuntary movement was pseudoathetosis, due to deep sensation impairment caused by vitamin B12 deficiency.
Key Words: pseudoatetosis, vitamin B12 deficiency, Biermer anemia

We present the case of a 69 year old female, caucasian, who came to the emergency room for unsteadiness of the gait and involuntary movement of the upper limbs (left > right). These symptoms started few days ago, after an emotional stress.

From the patient's *history* we disclosed the presence of untreated arterial hypertension (TAs max = 190 mm Hg) and of lacunary strokes in the last years.

In the last year she was admitted in several medical services:

– in a cardiology clinic – for angina pectoris and cardiac failure following a myocardial infarction in the inferior territory
– in a psychiatric clinic – for a depression
– in a gastroenterology clinic – for abdominal pain.

There was *no family history* of neurological diseases.

Physical examination revealed that the patient was pale, with an increased blood pressure (190/90 mmHg) and a rhythmic pulse (70 bpm).

Neurological examination disclosed:

– paraparesis
– impaired vibraton and position sense at lower limbs
– involuntary, sudden and abrupt, non-rhytmic jerks with variable and unpredictable timing and topical distribution in the upper limbs (left>right) – more pronounced in the digits and hands.

We diagnosed the involuntary movement as *pseutoatetosis,* differentiating it from the other "classic" involuntary movements such as: tremor, myoclonus, dystonia, hemiballismus, atetosis and chorea.

In order to investigate the cuase of this involuntary movement we performe a complex set of examinations.

- The ECG – showed the previous myocardial infarction (inferior teritorry).
- Fundoscopic examination - hypertensive rethinopaty (stage II).
- EEG – was normal, without any paroxysmal discharges.
- Ultrasonographyc Doppler examination – revealed moderate left internal carotid atheromatosis.
- Native cerebral CT scan – showed the old lacunar strokes in the basal ganglia, but no new lesions
- At lumbar punction – CSF had a gross appearance as clear, colourless, with normal protein and glucose content (protein content = 19 mg/dL – normal value = 12-60 mg/dl; glucose content = 54 mg/dL – normal value = 45-80 mg/dL)
- Laboratory evaluation – disclosed:

– at blood count we found *macrocitar anemia* (Hb = 10,8 mg (dL); HTC=32,4%; MCV=115), with normal white blood cells (WBC=6.5) and PLT=173; and an *inflammation* (ESR = 40 mm/1hour).
– the usual biochemistry was not of cancer, (normal blood glucose, blood ureea, creatinine – normal; normal levels of blood sodium, potassium and normal AST, ALT, CK, CK-MB, LDH). Total cholesterol was

normal, but there was a *mild increase of triglyceridemia (162 mmol/L).*
– the blood clotting tests were also normal.
– special laboratory tests revealed: HIV – negative; HTLV1,2 – negative; T3,T4, TSH – negative; folic acid – normal but a *decreased level of vitamine B12 – < 30 pg/ml (Normal 197-866).*

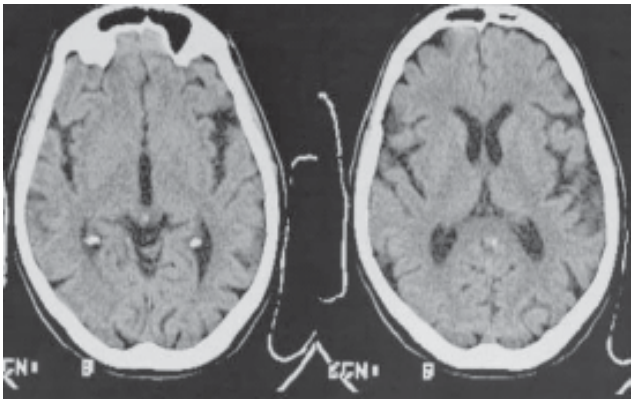

Figure 1
Native cerebral CT scan – showed old lacunar strokes in the basal ganglia, but no recent lesions

Because the patient had no new lesions on cerebral CT scan, we excluded a repeated ischemic stroke, although the patient had risk factors for ischemic stroke, there was the history of previous lacunar strokes, of untreated hypertension, mild hypertriglyceridemia and a moderate left internal carotid atheromatosis.

The lack of cognition impairment and negative familial history of neurological disorders stand against a neurodegenative etiology (Huntington disease), although the involuntary movements were associated with psychiatric pathology.

The epilepsy with focal motor seizures – is also excluded by the lack of myoclonus and the normal EEG recording.

Based on clinical arguments (dorsal colums lesions with impairment of deep sensation and *pseudoatetosis*, paraparesis due to cortico-spinal tracts involvment) and laboratory findings (decreased vitamin B12 levels in blood), our diagnosis was **subacute combined degeneration (SCD) of spinal cord due to vitamin B12 deficiency,** *the vitamin B12 dificiency being a rare cause of pseudoatetosis.*

Other differential diagnosis were:
• Cervical spondilosis
• Multiple sclerosis of cervical cord
• Female carrier of adrenoleuocodystrophy
• Non B12 deficient combined system disease:
– copper deficiency
– chronic decompensated liver disease
– in conjunction with AIDS
– tropical spastic paraplegia (HTLV-I)
– radiation myelopathy
– adhesive spinal arahnoiditis

The patient's outcome was favourable, with a good recovery after B12 replacement therapy.

The case particularity was the presence of megaloblastic anemia complications: myocardical infarction, depression, abdominal pain. Their presence required admission in several speciality clinics and thus the diagnosis was delayed. In this case, the involuntary movements presence was due to deep sensation impairment.

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