

15 YEARS-OLD GIRL WITH PARESTESIA, HEADACHE AND ABDOMINAL PAIN

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Abstract

Neuronal migration disorders are the conditions for multiple heterogeneous neurological manifestations, difficult to diagnose because of atypical and various clinical expressions. Continuous development of imagistic investigation opened new study possibilities. Paper aim is to present the case of an adolescent, 15 years old girl, admitted in Clinic II Pediatrics for abdominal pain, parestezia/pareses and headaches.

Key words: neuronal migration disorders, children, partial vegetative seizures.

Background

Neuronal migration disorders represent the substratum of numerous neurological manifestations (seizures, psychomotor retardation, dimorphism) that are hard to elucidate in the absence of neuroimaging investigations. The continuous development of new imaging techniques (computed tomography CT scanning, MR imaging, positron emission tomography, the study of cerebral metabolism) opened a new road in the study of abnormal neuronal development, differentiation and migration.

The alteration of the normal process of neuronal migration takes to cerebral malformations that are included under the terminology of ‘neuronal migration disorders’. The causes of inhibition of the neuronal migration process are various: infectious, vascular, (ischemic lesions that produce damage of the radial glioma fibers, of the molecular layer), toxic (exogenous and endogenous) and genetic causes.

For a better understanding diagnosis we will present a ethiopatogenic review. The neuronal migration represents the process in which thousands of nervous cells ‘walk’ from their place of origin (ventricular and sub ventricular areas), to their definitive places in the CNS, where they will stay for the rest of their life.

The neuronal migration disorders represent the alteration of the neuronal migration process; it happens during life in the uterus (the 3rd- 5th month of gestation).

The possibilities of alteration of neuronal migration are various: from very severe, with important alteration of the cerebral cortex, to minimal lesions. Neuronal heterotopias can be:

-Periventricular heterotopias

- Sub cortical heterotopias
- Superficial cortical-leptomeningeal heterotopias
- Schizencephalia
- Localised neuronal migration disorder
- Focal cortical dysplasia
- Microdysgenesis of cerebral cortex

Case presentation

The adolescent girl diagnosed with anxious-depressive disorder, in the context of a particular psychological structure, perfectionist type, with clinical subjective phenomenon, presented with multiples symptoms as: parestesia/pareses of the extremities, episodes of headaches, subjective sensations of warm/ cold, under treatment with sedatives, anti-depressives, with almost 2 months before the moment of this admission in the hospital to the present clinical symptoms are associated also persistent abdominal pain, pyrosis, vomiting, for which the patient is following a symptomatic treatment.

In the context of the digestive symptomatology that is an acute one at the moment, the patient was admitted in our service a month and a half before and was diagnosed with chronic gastritis with Helicobacter Pylori, anxious-depressive disorder, and hypocalcaemia. She was treated for the H.Pylori infection and in this time the sedative medication was totally excluded.

Evolution

After an initial improvement of the clinical status with 14 days before the actual admission she accuses the apparition of parestesia/pareses but this time organised on the left side of the body, on the left side of the skull, these being the reasons of the actual admission.

Ad admittance-subjective: anxiety, headache on left head part, parestezia/pareses on the left side of the body, objective: moderate abdominal pain in the upper abdominal area, bilateral positive Chwostek sign grade 2. The neurological examination was normal.

We were in front of a case with anxious-depressive disorders underlined by a specialist physician but the patient had a real subjective symptomatology, such as headache and parestezia/pareses on the left side of the body. In this context and having in mind the provided data a few questions were raised:

-Is it about an acute episode of the digestive disorder associated with subjective symptoms less representative?

-It represents a particular way of manifestation of hypocalcemia?

-Is the symptomatology strictly related to the anxious-depressive phenomenon?

-Is it a problem of a neurologic organic pathology?

Laboratory investigations revealed hypocalcaemia.

First neuropsychological examination revealed a very high emotional sensibility, immature state of affectivity, anxiety with vegetative hyperexcitability,

suffocating/choking sensation, sleep disorders, bodily discomfort.

An electroencephalography (EEG) was recommended and it was interpreted as normal electrical. The ophthalmologic evaluation and retinal vessels evaluation were in normal ranges. In evolution, the initial subjective symptomatology is maintained with the same characteristics, even with episodes of greater headaches than the initial ones.

A supplementary electrophysiological investigation is made-mapping EEG which revealed a normal route with synchronic peaks of discharging in the right temporal derivations with maximum in T4, P4 (fig.1).

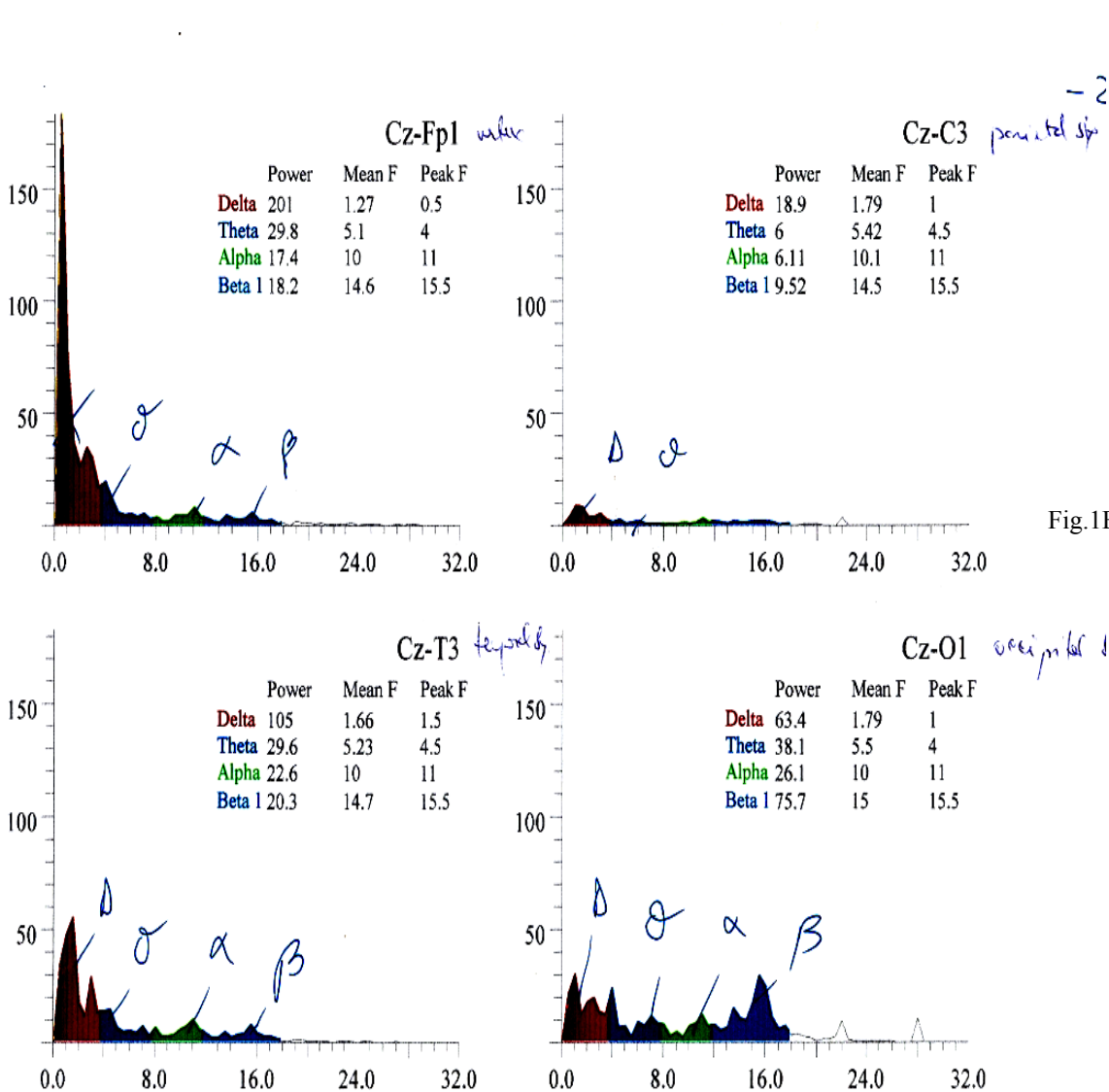


Fig.1 EEG mapping.

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Another neuropsychiatric children examination recommended a magnetic resonance procedure (MRI) which revealed an image in T1T2 weighed with grey substance in

the parietal-frontal area, anterior F at the level of the left semi-oval center-grey substance fig.2+ fig.3.

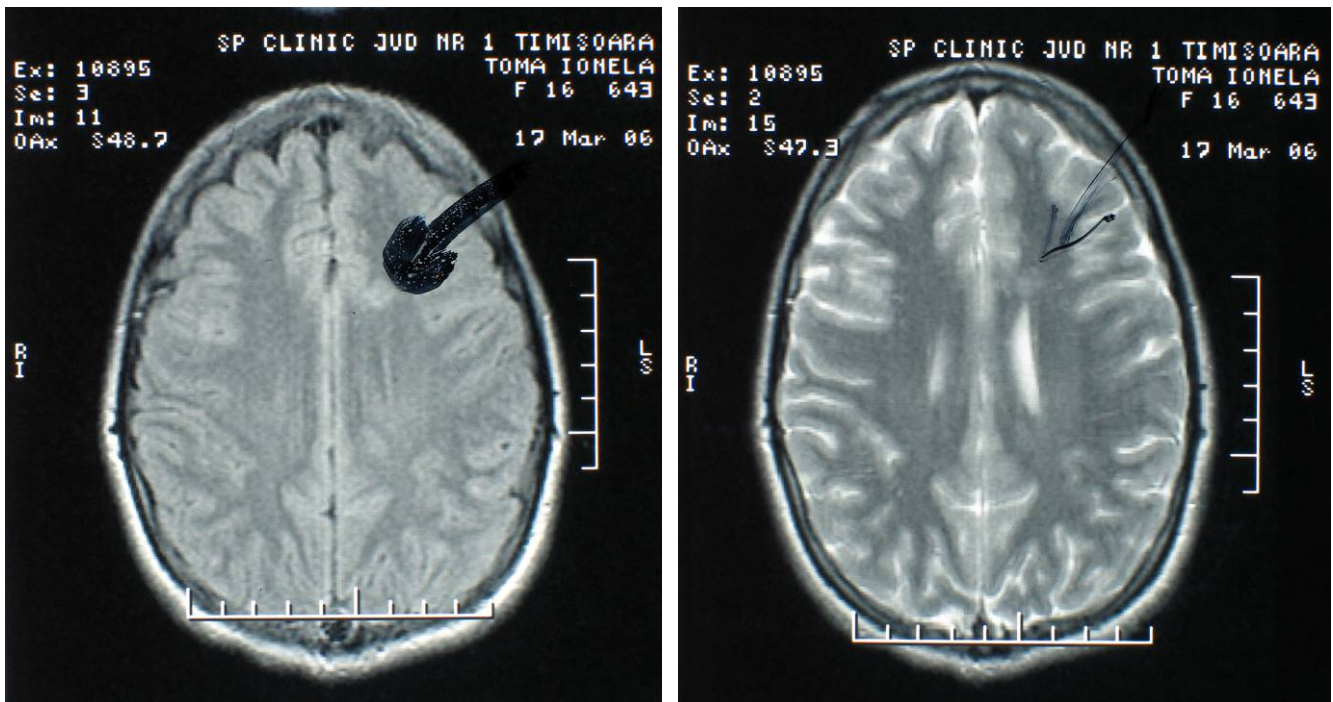


Fig 2 and 3. Magnetic resonance procedure (MRI).

The established diagnosis were:

-Partial comital crises through congenital neuronal migration disorders

-Hypocalcaemia

-Anxious-depressive disorders

The treatment applied was: Valproic acid-50mg/kg, 2x1tb/day of 300mg/day and calcium therapy.

After the clinical examination and the modification on the MRI very well defined and limited as extension we included this case in cortical focal dysplasia type I, in which there is evidence of a thickening of the brain in the affected area. The anatomic-pathologic examination is the one that confirm the exact diagnosis. The differential diagnosis of the partial seizures trough neuronal migration disorders could be made with:

1. First of all with type II of cortical focal dysplasia

2. Secondly with heterotopias with minor lesions, and from these with the neuronal migration disorders that are localised.

3. Other causes of partial convulsions have in their clinical aspect sings and symptoms that are present also at our patient:

-Partial convulsions with sensorial symptoms: transitory sensation of stinging, itchiness, numbness of the left side of the body. It reflects an epileptic discharge in the parietal cortex.

-Smell related convulsions- they can begin as an unpleasant odour felt by the patient, then they become generalised seizure, being an early manifestation of a temporal lobe tumour.

Partial convulsions with vegetative symptoms:-they include paleness, redness of the skin, headaches, tachycardia, pupil dilatation, abdominal pain, loss of urinal bladder control.

-Complex partial convulsions that appear because of the convulsive discharges in the temporal lobes ,the patient accusing a state of dreaming, illusions, complex visual hallucinations, impossible or fractionated talking, affective emotional disorders, anxiety. .

4. We also considered the intracerebral expansive processes (astrocytoma, ependimoma, ganglioma, meningioma), because of the various symptomatology that is present with simple partial epileptic crises or complex ones, progressive hemiplegia, intracranial hypertension. But in these cases, the neurological examination reveals sings of focus correlated with the tumors localization. The CT scanning reveals the location and aspect (calcification, chystic cavities) of the modifications. Neuronal ectopies can also bring up in discussion, like ectopic pinealoma.

Regarding hypocalcaemia could be secondary to decrease of calcium level or hypomagnesaemia.

The differential diagnosis of the anxious-depressive disorders raises the problem if they are primary in the context of a particular psychological structure of perfectionist type of the patient, being stressed out by the disease or they are secondary to the disease. Time will help to tell the delimitation of the exact ethiology.

The case evolution was favourable, with the disappearance of seizures. It still remains to discuss the

potential complications, in the absence of treatment and the secondary effects of medication, being well known that the valproic acid can accentuate the hypocalcaemia.

Conclusions

Extremely common symptoms like: abdominal pain, headaches could “hide” less frequent diagnosis, but with important outcome.

Even if there is an explanation for a clinical sign, like parestesia, differential diagnosis should be performed and the follow up of the patient is necessary.

We must consider every symptom and sign, especially if is sustained by paraclinical investigation. Interdisciplinary team approach is the most correct manner of management for complex cases.

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