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Paresthesias "as Clinical Element" and Changes in Cerebrospinal Fluid in Increasing the Level of "Comfort" for Diagnosis

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Abstract

Paresthesias are misperceptions, abnormal, subjective, declared by patients, which occur and are maintained without any stimulants to cause them. There are sensations, usually unpleasant with various manifestations: tingling, numbness, sensations of cold, heat, localized constrictions etc.

We present the case of a 47-year-old patient, known with autoimmune thyroiditis under treatment, whose neurological symptoms started one year before, acutely, with paresthesias in the distal bilateral upper limbs. Subsequently, paresthesias expanded, encompassing the lower limbs distally bilaterally, leading to gait disorders.

The symptoms had a fluctuating evolution (short periods of mild improvement alternating with periods of aggravation, but without complete relapse).

The patient was subject to repeated neurological evaluations, magnetic resonance imaging, brain and cervical spine, native and with contrast medium, and there was a suggestive appearance for a demyelinating condition. Lumbar puncture was performed and the cerebrospinal fluid was analysed, showing a slightly increased immunoglobulin G titer and the presence of oligoclonal bands.

Currently, the determination of oligoclonal bands in the cerebrospinal fluid is the most widely used test to confirm the diagnosis of multiple sclerosis. Their presence at the first attack of multiple sclerosis is predictive of a chronic evolution with relapses.

Although suggestive in the clinical context, changes in cerebrospinal fluid are not pathognomonic for multiple sclerosis, but their presence increases the "comfort level" for diagnosis.

Keywords: *paresthesias, demyelinating lesions, multiple sclerosis, cerebrospinal fluid, immunoglobulins G, oligoclonal bands.*

1. INTRODUCTION

Most neurologists will agree that sensitivity testing is the most difficult part of a neurological examination. On the one hand, the testing procedures are relatively rough and differ from the natural ways of stimulation with which the patient is familiar. It is also fair to say that few diagnoses are strictly based on sensitive examination; most often, this is an exercise that complements the motor examination. [1]

Sensitive symptoms such as paresthesias or dysesthesias may be generated along nerve axons that are not sufficiently affected by the disease to produce or affect sensory function; in the last resort, the loss of function may have been so slight and insidious as to go unnoticed. There is always a difficulty in assessing the response to sensory stimuli, as this depends on the patient's interpretation of the sensory experience. [1]

The presence of persistent paresthesias incriminates an injury that affects the nerve pathways along the nerves, spinal cord or higher structures. More commonly, large fibres in the peripheral nerves or posterior cords are involved. Transient paresthesias are, of course, of no clinical significance. [1]

Demyelinating diseases may be peripheral, such as Guillain-Barre syndrome (GBS), or multifocal motor neuropathy, or they may affect the central nervous system (CNS), such as multiple sclerosis (MS). Demyelinating

disorders involve the destruction of normally formed myelin or oligodendroglia, in contrast to the so-called dysmyelinating diseases (e.g., leukodystrophies) in which myelin is abnormally formed. Many demyelinating disorders, such as MS and acute disseminated encephalomyelitis (ADEM), appear to be immune-mediated, although pathogenesis remains an enigma. [2]

Multiple sclerosis (MS) is considered all over the world a public health problem, although, statistically, incidence and prevalence data would not justify the inclusion in this official concept. [3]

Multiple sclerosis (MS) is an inflammatory demyelinating disorder of the central nervous system (CNS) defined by episodes disseminated in time and neuroanatomical location.[4]

The diagnosis of MS is based on the association of clinical signs (disseminated in time and space) and paraclinical signs (especially magnetic resonance imaging (MRI). [5]

Classically, neurological deficits are multiple, with relapses and exacerbations that progressively cause functional disability. The diagnosis is made based on the relapse history and exacerbation of clinical symptoms, to which is added the objective highlighting of at least 2 distinct neurological abnormalities, through clinical signs, test results, lesions on MRI images or other criteria, depending on the symptoms. [6]

The most common symptoms of onset are paresthesias in one or several limbs, in the trunk or a hemiface, weakness or clumsiness of a hand or foot, and visual disturbances. [6]

If the MRI results and the clinical data obtained are equivocal, it is necessary to perform other tests to objectify distinct neurological abnormalities. Such tests usually begin with the examination of the cerebrospinal fluid (CSF) and, if necessary, brain-evoked potentials. [6]

The IgG in the CSF, expressed as a percentage of CSF proteins (normal <11%), of CSF albumin (normal <27%) or other CSF indices, is usually elevated. IgG levels correlate with disease severity. Oligoclonal bands can usually be detected by agarose gel CSF electrophoresis. [6]

2. Clinical Case

We present the case of a 47-year-old patient known to have autoimmune thyroiditis with hypothyroidism, under treatment, whose neurological symptoms started one year before, acutely, with paresthesias in the distal bilateral upper limbs.

Subsequently, paresthesias expanded, encompassing the lower limbs distally bilaterally, leading to gait disorders.

The symptoms had a fluctuating evolution (short periods of mild improvement alternating with periods of aggravation, but without complete relapse).

The patient was subject to outpatient neurological evaluations with native brain MRI, with an appearance suggestive of a demyelinating disease of the central nervous system, then 6 months later, the patient was subject to a native MRI scan and with contrast medium of the brain and cervical spine that showed demyelinating lesions in the supratentorial white matter, some with a long axis perpendicular to the ventricular system, without contrast medium uptake, a demyelinating lesion that affects the white matter on the right side of the brainstem, on a cranio-caudal area of 0.7 cm and several ranges in hypersignal T2 without contrast medium uptake at the level of the cervical spine, some posterior to the C2-C3 vertebral bodies on the left median and paramedian line with axial diameters of 0.8/0.7 cm. and cranio-caudal extension on 1cm, in the posterior-lateral left area near the C4-C5 vertebral bodies with axial diameter of 0.6/0.4 cm. and the right posterior-lateral area of the ventricular bodies C4-C5 with axial diameter 0.5/0.3 cm.

Clinical examination: BP (blood pressure) =120/91 mm Hg, VR (ventricular rate) = 90 beats per minute, conscious, cooperative, no neck stiffness, negative Lhermitte's sign, no damage to cranial nerves, left limbs motor deficit (4/5 MRC), clonus DTR (deep tendon reflexes), SAR (superficial abdominal reflex) absent, hypoaesthesia in the left hemibody, severe urination.

EKG (electrocardiogram): sinus rhythm, no terminal phase changes.

RX (radiography) heart-lung - appearance within normal limits.

Native MRI scan of the brain, cervical and thoracic spine and with contrast medium: focal lesions without restriction of diffusion, with no gadolinium uptake, at the level of the bilateral

infra- and supratentorial cerebral parenchyma - overall unchanged numerically or dimensionally compared to the previous examination. The appearance of the MRI scan argues for chronic, inactive demyelinating lesions - inflammatory substrate (atypical localization for the MS) or ischemic microangiopathic (vasculitis). Grouped pseudofocal lesions, with hyperintense T2 signal, partial cancellation of the signal in the FLAIR sequence, located in the left anterior temporal polar white matter - probably with the same chronic demyelinating substrate (superimposable to the previous examination).

Multiple cervical-thoracic intramedullary demyelinating lesions; only one lesion is active (intramedullary near the vertebral bodies T3 and T4), the rest of the lesions are inactive (chronic). C5-C6 disc protrusion, in the left posterior-lateral area that imprints the left intraforaminal C6 root.

Following the administration of pulse therapy with Methylprednisolone 1g./day, 5 days, the evolution was favourable with an improvement in the gait disorder.

Given the age at the onset and the distribution of the demyelinating lesions mainly in the spinal cord and without severe lesion load in the brain, other diseases that associate demyelinating lesions of the central nervous system with infectious aetiology, systemic autoimmune or systemic vasculitis have been considered.

Lumbar puncture was performed for the CSF analysis which showed a slightly increased immunoglobulin G (IgG) titre and the presence of oligoclonal bands.

The demyelinating lesions clearly meet the Mc Donald criteria for dissemination in space and there are 2 clinical outbreaks for dissemination in time and thus, the diagnosis of recurrent-relapsing multiple sclerosis, DSS score - 2.0 points was formulated and the patient is included in the National Multiple Sclerosis Program for treatment.

3. DISCUSSIONS

Before proceeding with the sensitive test, the physician should ask the patient about his/her symptoms; but this approach also raises certain issues. Patients may experience sensory disturbances, different from anything they have experienced up to that point, and have limited time to describe what they are feeling. They may say that their limbs are "numb" or "dead" when in fact they refer to weakness. [1]

What is paresthesia?

Paresthesia is typically a nonpainful, tingling sensation. [7]

What is myelin?

Myelin is the proteolipid membrane that ensheathes and surrounds nerve axons to improve their ability to conduct electrical action potentials. Oligodendrocytes make central nervous system myelin and wrap the myelin around axons, leaving gaps called nodes of Ranvier, where membrane ionic channels are heavily concentrated and powerful action potentials can thus be generated. [7]

What is multiple sclerosis (MS)?

MS is the most common condition that destroys myelin in the central nervous system. It affects approximately 250.000 Americans, mostly between the ages of 20 and 40, making in the leading disabling neurologic disease of young people. [7]

Neurological examination of patients with MS may be normal, minimally abnormal, or marked abnormally. Focused testing of cranial nerve functions, muscle strength, coordination, gait and sensitivity and assessment of pathological reflexes is important. [8]

In the emergency care unit, the de novo onset of MS is most commonly suspected in young patients (second or third decade of life) with acute or subacute neurological dysfunction. [8]

When dealing with a patient whose symptoms and clinical examination raise the suspicion of a diagnosis of multiple sclerosis, the neurologist is often forced to perform a series of laboratory and imaging investigations, which can be exhausting for the patient and can create a false impression of insecurity or ignorance on the part of the doctor. It must be said from the beginning that there is no specific test to diagnose multiple sclerosis, as there is for diagnosing a metabolic or infectious disease. Communication with the patient is essential for the patient to understand the need to perform each of the recommended scans and that each of these tests contributes to the elimination of other possible diagnoses (or leads the diagnosis to a different path, if the test results are modified. [3]

What are the most common symptoms of MS?

- Pyramidal weakness – 45%

- Visual loss – 40%
- Sensory loss – 35%
- Brain stem dysfunction – 30%
- Cerebellar ataxia and tremor – 25%
- Sphincter disturbances – 20% [7]

The way the CSF is formed and its particular relationship with the cerebral parenchyma determines the appearance of its biological changes, important in the diagnosis of MS. [9]

In current practice, the analysis of the IgG content in the CSF as a global marker of a local immune activity in the CNS and the qualitative identification of abnormal Ig synthesized intrathecally in the form of oligoclonal electrophoretic lesions are important. [9]

These bands are detected by high-resolution membrane electrophoresis, the method having an increased sensitivity and specificity, but not 100%, being also operator dependent, with quite large variations between laboratories. [9]

The presence of oligoclonal bands is not pathognomonic for MS, and however they can be found in some CNS infections and inflammations (neurolues, neuroboreliosis, cysticercosis, subacute sclerosing panencephalitis), and complete criteria of positive and differential diagnosis must be met for MS. [9]

How can the cerebrospinal fluid be used to diagnose MS?

Immunoglobulins are increased in the central nervous system in patients with MS. When the immunoglobulin G (IgG) is examined by electrophoresis, it may concentrate into specific bands. The finding of multiple bands in the IgG region, called oligoclonal bands, is reasonably sensitive and specific for MS. However, it remains unclear how or why oligoclonal bands are produced or exactly what they represent. [7]

Do other diseases have oligoclonal bands?

Yes, especially inflammatory and infectious conditions such as syphilis, meningoencephalitis, subacute sclerosing panencephalitis (a latent measles infection), and Guillain-Barre syndrome. However, these are unlikely to be confused with MS. [7]

Relapsing-remitting multiple sclerosis (RRMS). This subtype is the most common (85% of all patients fit into this disease category at diagnosis). It is characterized by relapses and remissions of neurologic disability over years to decades. Incomplete recovery from relapses often leads to disability accumulation. [10]

The evaluation and grading of neurological deficit is important for assessing the evolution of the disease, but also for establishing the degree of effectiveness of therapy schemes either individually or in the case of groups of patients in trials exploring new therapies or new valences of drugs already in use. [9]

The evolution of the disease is quantified using the EDSS (Expanded Disability Status Scale) developed by Kurtzke (Neurology, 1983). [11]

What is the Expanded Disability Status Score (EDSS)?

The EDSS is a number that rates a patient's degree of disability from MS on a scale of 0 to 10. Deficits are determined in various functional systems (motor, sensory, cerebellar, etc.). A patient with a score of 6 requires a cane to walk and with a score of 8 is confined to a wheelchair. The EDSS is widely used as a standard method of evaluating MS patients. [7]

Therefore, although all neurologists need to know the particularities of this disease, it is necessary that, for care through immunomodulatory treatment, these patients be sent to centres of expertise. And this is because the diagnosis of MS is not easy to establish and requires the correlation of much more data, as well as the realization of a differential, clinical and imaging diagnosis, which is also not easy to achieve. [3].

4. Conclusions

Although suggestive in a clinical context, the CSF changes are not pathognomonic for MS, but their presence increases the level of "comfort" for diagnosis in a patient with progressive disease from the onset.

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