CASE REPORT

CELIAC DISEASE AND MULTIPLE SCLEROSIS: A POSSIBLE RELATIONSHIP

L. Castorani[1], S. Crudele[1], G. Dipaola[1] ,

INTRODUCTION

Celiac disease (CD) is an autoimmune disease characterized by a permanent intolerance to gluten.

It involves genetically predisposed individuals causing inflammation of the duodenum until the atrophy of the villi. Gluten is the protein complex content in grains such as wheat, barley, rye, spelled, kamut, triticale and spelled wheat.

The CD has a prevalence of 1:100 (1).

It is more frequent among women with a male/female ratio of 1:2.
The risk of CD is higher in type 1 diabetes mellitus (DM type 1), in Down syndrome, in IgA deficiency and in relatives of first degree of celiac patients.

The gluten crosses the barrier duodenal through the opening of tight junctions, inside the duodenum it is deamidated by tissue transglutaminase.

The dimers of gliadin, so obtained, are phagocytosed by antigen-presenting cells (APC) and subsequently expressed on the surface of these cells in association with molecules of the HLA complex.

The activated T lymphocytes, upon binding of APC, promote the formation of Th1 and Th2 lymphocites, with subsequent inflammation of the mucosa and production of autoantibodies.

The clinical spectrum of CD is highly heterogeneous, because today there are intestinal and extra-intestinal symptoms.

The diagnosis of celiac disease is based on a determination of antibodies against transglutaminase IgA (t-TG) and antiendomysium (EMA) which have a specificity around 100%.

The gold standard remains the esophagogastroduodenoscopy (EGDS) with subsequent histological examination with Marsh classification (2).

The current treatment is the gluten-free diet. Multiple sclerosis (MS) is a demyelinating disease that causes inflammation and destruction of myelin in the central and peripheral nervous system.

MS is three times more common in women than in men. The age of onset is between 20 and 40 years.
It is characterized by inflammation, demyelinating and gliosis.

The lesions are disseminated in time and space.

Acute lesions are near the veins and they are characterized by an inflammatory infiltrate resulting in damage to the blood-brain barrier.

The disease can manifest at presentation with optic neuritis, diplopia, fatigue, loss of sensation, numbness and ataxia.

[1] U.O. Medicina e Chirurgia d'accettazione e d'urgenza P.O. "Mons. Dimiccoli" Barletta, ASL BT

Parole chiave:
celiachia, sclerosi multipla

Keywords:
Celiac, autoimmune, demyelinating

This article was published on September 25, 2019, at SIMEDET.EU .

doi.org/10.30459/2019-16

Italian Journal of Prevention,
Diagnostic and Therapeutic Medicine.

We distinguish various forms of MS: relapsing/remitting, secondary progressive, primary progressive, progressive relapsing.

The diagnosis, as well as clinical, is based on: MRI with contrast medium and examination of the cerebrospinal fluid.

The therapy uses drugs, which today are able to slow the progression of the disease, especially in the form RR.

CASE REPORT

We describe the case of a 29 year old woman who at the age of 21 years, after the onset of diplopia and dizziness was admitted to the Clinical of Neurology and discharged with a diagnosis of MS.

In anamnesis she performed therapy with isotretinoin for acne and, an estrogen-progestin therapy for polycystic ovary syndrome.
He had a family history of cancer of breast, uterine and, thyroid, psoriasis, celiac disease and hypertension.

At the time of diagnosis of MS, the patient was subjected to lumbar puncture ("intrathecal synthesis of IgG oligoclonal"), a brain MRI with contrast medium ("periventricular plaques of demyelinating, which exhibit rather sharp contrast enhancement") and visual evoked potentials ("morphology enlarged with increase in the latency time at low and medium spatial frequencies").

She was discharged with an indication to continue therapy with methylprednisolone gradually climbing up the suspension.

The biochemical tests showed hypotriglyceridemia, iron deficiency anemia and positive for thyroid peroxidase antibody (127.1 IU/ml with normal values between 0 and 20) and anti-nuclear (title 1/320 speckled nuclear pattern).

At a distance of 4 to 10 months she performed two new brain MRI that didn't show new lesions.

One year after the diagnosis of MS, iron deficiency anemia, hypocholesterolemia and hypotriglyceridemia were investigated.

The patient dosed autoantibodies for celiac disease. The t-TG (18.33 U/ml with normal values between 0 and 7) and EMA were positive.

She performed a EGDS which showed: "duodenal mucosa with mosaic appearance, reduction and scalloped of Kerkring folds".

Histology confirmed the diagnosis of celiac disease: "frustules of mucosa of the small intestine with a marked lymphoplasmacytic and eosinophilic inflammatory infiltration, increased intraepithelial lymphocytes and subtotal villous atrophy (celiac disease Marsh 3b)".

She subsequently performed the study for the HLA class II, which showed the presence of DQ8. After the diagnosis of CD she started the gluten-free diet.

At 6 months of diet ANA, EMA and t-TG were negative. The patient didn't show iron deficiency, hypotriglyceridemia and hypocholesterolemia.

She continued to perform annual radiological controls.

At 5 years from diagnosis of MS, MRI showed an increase in the number of lesions without contrast enhancement (ce).

It was decided to have a wait and see approach with a short interval control.

6 months later, the patient repeated MRI that showed two new lesions in the upper mid-portion of the ascending frontal gyrus and left around the ascending parietal ipsilateral substantially at the same level, the first of which exhibited net contrast enhancement, and the second, late, a tenuous, almost null, degree of ce.

The patient began an immunomodulatory therapy with glatiramer acetate (1 fl / day sc).
Five years starting therapy with glatiramer acetate, the patient didn't show new lesions.

In these five years the patient performed two brain and spinal cord resonances every year without showing new lesions.

With the gluten-free diet (persistence of negativity of anti transglutaminase and endomysium) other biochemical changes pre-existing were resolved.

CASE REPORT

The MS and CD are two autoimmune diseases, but today only for CD exists a therapy: the gluten-free diet.

Many studies report the various neurological disorders that may occur in the course of celiac disease, even in the absence of gastrointestinal disorders [(3)].

Furthermore, often in the course of CD we can find white matter lesions similar to those of the MS [(4)].

There are some reported cases of patients diagnosed with MS and CD. Often the CD is diagnosed after another autoimmune disease and it is believed that the more time the patient is in contact with the gluten more there is the possibility of developing a complication.

Often, when we are faced with a patient with a neurological disease, such as MS, CD should be suspected, especially if there are signs of malabsorption or if there is another autoimmune disease such as Hashimoto's thyroiditis.

Recent studies indicate an increased intestinal permeability in the course of MS and so it is always appropriate to assess in patients with MS a possible duodenal involvement [(5)].

In the case described above, an unrecognized and undiagnosed CD in time may have favored the onset of MS.

At present, the patient seems to respond well to treatment with glatiramer acetate, although we believe that the correct adherence to the gluten-free diet may have helped and continue to help to not cause a severe and progressive neurological involvement.

We believe that further studies should be done to better understand the pathophysiological mechanisms underlying these patients with multiple autoimmune diseases, and especially in the study of intestinal permeability which, if altered, would favor the passage of various antigens responsible for damage in various organs.

BIBLIOGRAFIA

1. *Coeliac disease. A. Di Sabatino, G.R. Corazza. Lancet 2009; 373: 1480-93.*

2. *Grains of truth: evolutionary changes in small intestinal mucosa in response to environmental antigen challenge. Marsh MN. Gut. 1990 Jan; 31 (1): 111-4. Review.*

3. *Neuropathy associated with gluten sensitivity. M Hadjivassiliou et al. J Neurol Neurosurg Psychiatry 2006;77:1262-1266.*

4. *Brain White-Matter Lesions in Celiac Disease: A Prospective Study of 75 Diet-Treated Patients. Matthias Kieslich et al. Pediatrics 2001;108;e21*

5. *Leaky Gut and Autoimmune Diseases. Alessio Fasano. Clinic Rev Allerg Immunol (2012) 42:71-78*