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Short Communication

Neurologic Diseases and Non-Celiac Gluten Sensitivity

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Celiac Disease (CD) is a systemic, autoimmune, frequent condition of complex nature, and the main lesions are placed at the level of the small intestine [1]. Usually it affects to sensitive individuals with a genetic predisposition, when they are exposed to gluten. It remains throughout life and its treatment is based solely on strict monitoring and continued a Gluten-Free Diet (GFD). It is more common in women, as occurs usually with other autoimmune diseases [2]. Traditionally it was thought that CD was more common in children. However, through clinical studies in recent years have shown, it is much more common in adults, appearing at any age of life, with a ratio of 7 to 10 cases in adults, for every 1 affected children [3].

An emerging problem in clinical practice is how to manage patients who experience wheat or gluten dependent symptoms in the absence of the main stigmata of CD, such as positive results on serum antitransglutaminase or antiendomysial antibody testing and small-bowel villous flattening. This situation is named as Non-Celiac Gluten Sensitivity (NCGS) that is usually characterized by the presence of intestinal symptoms (such as diarrhea, abdominal discomfort or pain, bloating and flatulence) and also by extraintestinal symptoms (such as headache, lethargy, attention-deficit/ hyperactivity disorder, ataxia, or recurrent oral ulceration), which improve or disappear after gluten withdrawal in individuals in whom celiac disease has been ruled out on the basis of negative serologic results or demonstration of normal small-bowel mucosa [4]. The concept of NCGS, is not new. Apart from sporadic case reports in children and adults, a double-blind, crossover trial more than 30 years ago, showed that 6 of 8 adult patients, who had abdominal pain and chronic diarrhea were gluten sensitive in the absence of celiac disease, at the begining of the 1981 [5].

Prevalence of CD is around 1% in the general population worldwide and for NCGS is extremely variable ranging between 10-15%, so is much more frequent tan CD [6-8].

Both CD and NCGS patients have associated with a broad variety of neurological diseases such as migraine. In celiac disease patients, migraines have been described on a series of cases and controls. In one study, migraines were present in 40 out of 188 celiac disease patients (21%), when comparing with 13 out of 178 controls (7%) [9]. In the same manner, the presence of cephalea and brain imaging alterations have been described also on non-celiac gluten sensitive (NCGS) patients [10].

Cerebellar ataxia is the most frequent neurological manifestation and also was the first to be described on the original description made by Cooke and Smith in 1966 [11], several patients had sensory ataxia however three of them, had cerebellar ataxia. After this paper, numerous reports have been made, on CD related ataxia and in recent years also associated with NCGS [12,13].

Celiac disease associated peripheral neuropathy has been reported in many cases and usually they are associated to distal sensitive symptoms, sensitivity loss and imbalance. Peripheral neuropathy can occur even before the development of intestinal symptoms. It is important also to consider clinical neurological manifestations in celiac patients and to research these conditions also in the follow-up, because they may start also 1 year after the start of the Gluten Free Diet (GFD). The diagnosis of NCGS should be considered in patients with persistent intestinal and/or extraintestinal complaints showing a normal result of the CD serological markers on a gluten-containing diet, usually reporting worsening of symptoms after eating gluten-rich food [14-17].

Currently, the commitment of the central nervous system associated to CD is poorly understood.

Although we have greater information on prevalence, pathogenesis, genetic factors and diagnostic tests, in some cases the physiopathological impairments responsible for neurological and psychiatric manifestations are just proposals.

Suspicion of subclinical CD is important; in some cases it is likely responsible for the etiology of multiple central nervous system pathologies. This must be taken into account especially on population groups where the prevalence is considered low.

The fact that patients with NCGS may have the same neurological complications than those who have a CD, it makes the suspected diagnosis must be extended to a larger percentage of neurological patients, since this therapy is free of known side-effects and it consists only in minor dietary changes.

In order to test its clinical efficacy, one strict gluten free diet should be maintained for a minimum of 6 months time, preferably for one year.

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