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"Gluten Ataxia and Its Connections to Non-Celiac Gluten Sensitivity and Celiac Disease"

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Definition:

According to its etymology, "ataxia" signifies clumsiness or incoordination. It is a symptom, not a clinical entity or a diagnostic in and of itself. The diseases that cause it have one thing in common: a persistent, progressive change in balance, which is more noticeable when standing and walking, as well as a lack of coordination between the extremities and movement. These conditions are all accompanied by language disorders, which include trouble pronouncing words correctly and needing to put in extra effort to understand them, particularly when they are difficult or during lengthy conversations. The underlying lesion is primarily located at the level of the cerebellum, which is the part of the brain responsible for language, postural balance, and movement coordination. His clinic is very distinctive and is identified by the presence of multiple language disorders, including difficulty pronouncing certain words and phrases, known as "dysarthria," and frequent disturbances of balance movements, which are very evident during walking, which is very unstable (it is graphically compared to the way used by very drunk or simply "drunked"). Additionally, a disturbance of hand mobility may be present, characterized by difficulty performing fine movements, a generalized loss in strength, and increased physical weariness following little exertions.

GA, or gluten ataxia:

The term "sporadic idiopathic ataxia" was initially used to describe this condition, which was defined by the presence of positive gluten sensitization indicators in serum, primarily anti-gliadin antibodies (AGA) [1]. It is an autoimmune condition, similar to celiac disease (CD), which develops as a result of cerebellar lesions and causes ataxia.

The study of epidemiology

Over the course of 15 years, Dr. Hadjivassiliou et al. at the University of Sheffield in England examined 800 patients with progressive ataxia. They discovered that, of the 635 cases they examined, 148, or 23% of the population, had positive blood anti-gliadin antibodies, which are linked to gluten intolerance and are shortened to gluten ataxia (GA). Similar occurrences have since been discovered in a number of series of research conducted on ataxia patients. All of these studies had one thing in common: ataxic patients consistently had a much greater prevalence of AGA than the general population of healthy controls [2–5].

Pathogenesis

Some evidence points to the possibility of cross-reactivity between different gluten proteins and antigens found at the level of the cerebellum's Purkinje cells. [6–8]. It has been demonstrated that people with gluten ataxia have anti-transglutaminase antibodies, or TGT, which are found around the brain's blood vessels and are linked to gluten intolerance just like AGA. Its distribution at the level of the spinal cord, protuberance, and cerebellum is more significant and obvious. A subtype of them, known as TGt-6, has been identified recently; this is the one that is most frequently found in patients with gluten ataxia [9–11].

GA in conjunction with NCGS (non-celiac gluten sensitivity): More examples of gluten ataxia in individuals who do not technically meet the criteria for celiac disease and are more appropriately categorized as non-celiac gluten sensitivity (NCGS) have been reported in recent years. This clinical entity was originally identified in 1980, but it wasn't until 2010 that it was acknowledged as a distinct disease. It was subsequently categorized as part of the spectrum of gluten-related disorders, which also includes wheat allergy (WA) and celiac disease (CD). The most common of these is the NCGS. Up to 13% of the general population is thought to be affected, indicating a significant incidence [12–14].
The NCGS presents with a wide range of symptoms that are nearly the same as those associated with celiac disease (CD). Since the histological and serological markers against gluten are typically negative and show a positive response, similar to celiac patients, to the removal of gluten from the diet (GFD), it is diagnosed by first ruling out a CD. NCGS typically manifests only as extra-intestinal symptoms, with the skin, musculoskeletal system, and nervous system typically being the most affected. Similar to what happens to celiac patients, all symptoms get better with a GFD [17].
The frequency of celiac serological markers (AGA and transglutaminase antibodies) was compared between 31 patients with gluten ataxia, 48 celiac patients, and 37 SGNC patients in a comparative clinical investigation that our group did.

Duodenal biopsies and genetic susceptibility markers (HLA-DQ2 and DQ8) revealed that patients with GA were more similar to those with CD [18]. There have also been reports of GA cases involving multiple siblings in the same family, whose inheritance is most likely dictated by the existence of one related CD [19]. Early detection of a CD or NCGS in GA patients allows for a gluten-free diet to begin more quickly, which in turn causes the neurological symptoms to gradually improve until, in many cases, they completely disappear.
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