Etiology, Epidemiology: Pathologic Changes in the Bones Associated with Celiac Disease

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Abstract

Osteopenia, osteoporosis, and low bone mineral density (BMD) are common side effects of celiac disease (CD). Intestinal malabsorption and chronic inflammation are the two primary processes at play in the complex genesis of pathologic bone changes in CD. The only known effective treatment for CD is a strict gluten-free diet (GFD), yet managing CD-related bone problems is still challenging. This review's goals are to clarify the bone issues associated with CD and raise awareness of osteoporosis development, which is seen as a marker of an unusual CD presentation. There is currently discussion on the effi cacy of GFD alone in treating CD patients' altered bone structure. In this review, recent studies on the causes of pathologic bone derangement are presented. Low BMD, osteoporosis, fractures, and treatment of bone issues in CD patients are all epidemiologies. Additionally, transport pathways and the roles of calcium are discussed.

Keywords: Celiac Disease; Gluten; In ammation; Intestinal Absorption; Bone Density; Osteoporosis

Introduction

Accurate diagnosis is made more di cult by the signi cant degree of variability in the clinical presentation of celiac disease (CD). e autoimmune response in CD mostly a ects the intestinal mucosa, but it can also show up as a variety of signs and symptoms that might impact any organ or tissue. Extraintestinal symptoms like low bone mineral density (BMD), decreased bone mass, and increased bone fragility that result in a higher rate of fractures must be taken into account as an indication of an atypical CD presentation [1]. e loss of villous cells in the proximal intestine, where calcium is most actively absorbed, is the primary cause of these bone alternations, which also result in secondary hyperparathyroidism and reduced calcium and vitamin D absorption. Several investigations assessed the condition of the bones in CD patients before and a er gluten-free diets. However, because both ancient and new ndings are wildly discordant, research concentrating on the incidence of bone derangement in patients with CD is still unclear. A strict, lifelong GFD is currently the only e ective treatment for CD [2].

Material and Methods

It is not yet known, however, if GFD alone is su cient to reverse the changes to the bones or whether certain metabolic bone illnesses are curable. Research on the impact of GFD on bone change in CD has produced con icting ndings. According to several researches, people with CD who follow a GFD had a much lower likelihood of having low BMD. In contrast, the outcomes of additional research revealed that patients with persistent small-intestinal mucosal villous atrophy, despite adherence to a rigid GFD and the absence of had a signi cant risk of osteoporosis because of symptoms. Patients with CD who also have other bone metabolic issues and bone mineral loss unquestionably need to be managed properly. Early CD treatment may help to reduce the risk of developing cancer, osteoporosis, and other autoimmune illnesses [3]. Given that the majority of bone mass is acquired during the rst two decades of life, early CD diagnosis and adherence to a GFD are crucial to ensuring optimal bone metabolism in such situations. Recent clinical trials for several innovative CD therapy modalities are still continuing, however these therapies attempt to reduce the need for a rigid GFD by altering dietary food products, minimise gluten

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