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Historical Perspective of Celiac Disease

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Abstract

Ten thousand years ago, celiac disease (CD) appears on the face of the Earth with the most dramatic change in human history: the agricultural revolution that brought to man's table foods that he had never experienced in his previous 2.5 million years. Among them eventually wheat was domesticated, selected mostly for the appearance of mutants with large grains and with ears resistant to harvesting. Not all humans adapted and, among those who had a genetic predisposition to develop an abnormal immune response rather than tolerance, CD emerged, becoming soon a major killer of infants for many generations. Little less than 2,000 years ago, a clever Greek physician named Aretaeus of Cappadocia wrote a total of 8 books on medicine, providing the first known description of adult patients with CD. He had quite a grasp on this condition, that he named 'koiliakos' after the Greek word 'koelia' (abdomen), to imply that these patients were 'suffering in their abdomen'. Seventeen centuries went by. On October 5, 1887, at Great Ormond Street in London, Dr. Samuel Gee, a Lecturer in Medicine at St. Bartholomew's Hospital as well as Physician in the Hospital for Sick Children at Great Ormond Street, gave to medical students a lecture on the 'celiac affection' that was published the following year: this constitutes the modern 'rediscovery' of CD. Also Dr. Gee had good insights into CD, as he wrote: 'If the patient can be cured at all, it must be by means of diet', adding: 'The allowance of farinaceous food must be small'. In 1924 Sidney Haas described his successful treatment of 8 children whom he had diagnosed as having CD, by using a diet based mostly on bananas. The banana diet, of which he became soon a fervent, strenuous advocate, ruled the world as the only treatment for CD for decades, even years after Dicke, Weijers and van de Kamer had produced their famous series of seminal papers documenting for the first time the role of gluten from wheat and rye in causing the harm of CD. Soon after the role of gluten in causing the flat lesion of CD had been ascertained, theories began to be put forward as to why gluten would be causing that intestinal damage and its subsequent symptoms. The first was an enzymatic one: an enzyme ought to be either missing or malfunctioning, thus leading to an inability to properly digest gluten, generating toxic fragments. This theory persisted for many years without any solid evidence but was put to rest when it became finally clear that CD had an immunological basis, and its autoimmune nature was convincingly demonstrated by the identification, in 1997, by a German group led by Dietrich, of the autoantigen: the ubiquitous enzyme tissue transglutaminase. Along with the progress in the clinical description of CD and in its diagnosis (something that flourished after the availability in the early 60s of the peroral biopsy capsule), efforts were made especially by the European Society for Pediatric Gastroenterology (today the European Society for Pediatric Gastroenterology, Hepatology and Nutrition) to define precise diagnostic criteria. Such criteria were put forward in 1970, revised in 1990 and accepted basically worldwide until now. Currently in fact, the fast advancing knowledge on CD pathophysiology, its many forms and its natural history is knocking at the door for their further revision.