

Case study

Digestive disease in chronic inflammatory diseases in children (about 10 cases)

Abstracts : Restructure and beef up the abstract in a scientific fashion consistent with : brief background ; aim of study ; material and methods ; result ; conclusion/recommendation !!!

This work consists in the retrospective analysis of 10 children with chronic inflammatory diseases, with digestive involvement at the Children's Hospital of Rabat during a period from 2001 to 2018.

The types of digestive involvement found were diverse: Crohn's disease, ulcerative colitis and celiac disease. The frequency of digestive involvement in chronic inflammatory diseases in our series is 1, 58%. The incidence of association of juvenile idiopathic arthritis with IBD is 1.08% and with celiac disease is 0.4%, while the frequency of association of systemic lupus with celiac disease is 2.8%.

Our series includes 7 female and 3 male children.

The average age of our patients at the time of diagnosis of JIA or SLE is 10 years and the average age of the digestive involvement is 07 years.

The common etiopathogenic mechanisms that explain the link between these diseases are not yet well defined, but genetic predisposition seems to play an important role in these associations, as well as environmental factors such as long exposure to gluten, for celiac disease.

Keywords : chronic inflammatory diseases, JIA , SLE , crohn disease, ulcerative colitis, celiac disease.

Introduction: Too scanty with no introduction of the study matters and references to already known facts, beef up !!!

Juvenile idiopathic arthritis (JIA) and systemic lupus erythematosus (SLE) are the most frequent pediatric chronic inflammatory diseases associated with digestive involvement. IBD (Crohn's disease and ulcerative colitis), as well as celiac disease are the most frequent associated .

Objective:

To describe the epidemiological and etiopathogenic aspects of digestive involvement in chronic inflammatory diseases of children .

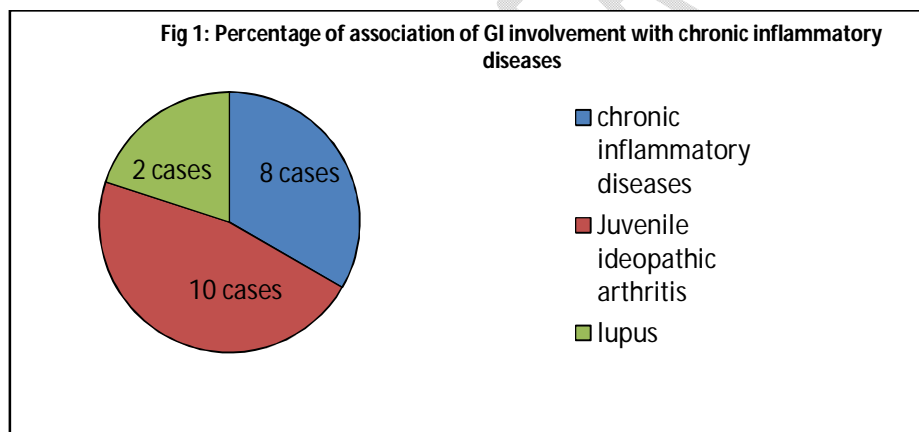
Material and methods: Not written properly to depict the materials, data collected and parameters used for the study. Pls beef up

This is a retrospective analysis of 10 children with chronic inflammatory diseases with digestive involvement collected at the pediatric department IV and at the pediatric rheumatology consultation of the children's hospital of Rabat over a period of 17 years (2001-2018). digestive disorders.

- Average age of association of chronic inflammatory disease and digestive involvement: 10 years.
- Average age at diagnosis of chronic inflammatory disease in our series: 10 years.
- Average age of digestive involvement: 07 years.
- Female predominance (70%).

Results : NO tables or statistical analysis of data

Percentage of association of GI involvement with chronic inflammatory diseases :



Discussion: This is not well discussed, the present study results/outcome should be assessed, compared and corroborate with the existing body of knowledge. Implication and inferences of the present study are drawn. !!!!

1. Association of juvenile idiopathic arthritis and inflammatory bowel disease diseases:

Juvenile idiopathic arthritis (JIA) is the most common chronic rheumatic disease of childhood and represents a heterogeneous group of conditions.

common in childhood, it represents a heterogeneous group of conditions including enthesitis-related arthritis, also known as juvenile spondyloarthritis, which represents the juvenile spondyloarthritis which represents the pediatric form of spondyloarthropathies.

Classically, five entities define spondyloarthropathies: Ankylosing Spondylitis ankylosing spondylitis, psoriatic arthritis, reactive arthritis, arthritis associated with Inflammatory bowel diseases (IBD) and Undifferentiated spondyloarthritis.

In addition to skeletal disease, extra-articular disorders such as uveitis, and inflammatory bowel disease (IBD) often occur in the course of spondyloarthropathies , offering a significant aid in the diagnosis of these diseases .

It should be noted that IBD contributes to the diagnosis of axial SpA in patients with chronic low back pain .

Chronic inflammatory bowel disease (IBD) occur much more frequently in spondyloarthritis than in the general than in the general population and are associated with disease activity .

Similarly, in a large population-based study, it was shown that the incidence rate of IBD was shown to be 5.3 times higher in patients with SpA compared to healthy controls .

Axial and peripheral SpA is more common in Crohn's disease than in ulcerative colitis . This is consistent with the results of our study.

It is well known that there is a close association between IBD and SpA .Several etiopathogenic mechanisms have been suggested to link these two entities, including genetic predisposition.

1.1. Genetic susceptibility :

Genetic factors play an important role in the link between SpA and IBD, by modifying the adaptive and innate immune pathways .

1.1.1. HLA genes :

Certain human leukocyte antigen (HLA) alleles have been identified in IBD patients who are patients with IBD who are at greater risk of developing SpA, in fact HLA-B27 confers an additional risk of an additional risk of inflammatory low back pain in IBD patients. ThusHLA-27 typing may be a potential factor, as the frequency of HLA B27 in IBD patients is generally no higher than in the general population.

Other alleles have been incriminated in the JIA-ICHHD association; the MHC class II allele DRB1 0103 as well as HLA-B35 are frequently associated with peripheral SpA .

MHC class II allele and HLA-B35 are frequently associated with peripheral SpA , while approximately 38% of patients with UC or active Crohn's disease have been identified as carriers of the identified as carriers of the DRB1 0103 allele .

1.1.2. Non-MHC (non-HLA) genes :

Genetic factors outside the HLA system have also been described. The variations in the CARD15 gene (which codes for the NOD2 protein product) increase the risk of Crohn's disease approximately 4 to 40 times and have been linked to the development of sacroiliitis in IBD patients .

NOD2 is an intracellular receptor for bacterial molecules that is expressed on the surface of macrophages, lymphocytes, and expressed on the surface of macrophages, lymphocytes, Paneth cells and intestinal epithelial and intestinal epithelial cells.

This receptor plays a role in the innate immune response by activating the nuclear factor κ B (NF κ B) which is a transcriptional regulator of a wide variety of genes encoding pro-inflammatory cytokines, adhesion molecules, cytokines, growth factors and growth factors and enzymes) .

Accordingly, NOD2 is responsible for the up-regulation of the immune immune defense in the gut and the induction of a pro-inflammatory state .

In our series, the genetic study could not be performed due to lack of means. only the search for HLA B27 was carried out in the 5 cases presenting the association HLA B27 was positive in 3 cases.

1.2. The intestinal synovial axis :

Several other results also highlight the underlying etiopathogenic mechanisms underlying etiopathogenic mechanisms common to IBD and SpA.

Notably, the integrin α 5 β 7 that is expressed by intraepithelial T cells in the intestinal mucosa and which binds to the glycoprotein E-cadherin expressed by intestinal epithelial cells, and has been shown to be elevated in patients with SpA .

2. Association of celiac disease and autoimmune diseases :

The causal relationship between celiac disease and other autoimmune diseases such as juvenile idiopathic arthritis and systemic lupus erythematosus is still a controversial. The two most accredited theories propose: this association is secondary to a common genetic background predisposing to celiac disease and the associated autoimmune disease) or untreated celiac disease leads to the onset of other autoimmune diseases in genetically predisposed individuals .

Genetic susceptibility :

The genetics of celiac disease are complex because multiple genes contribute. Some linkage and association studies have identified several genomic regions that likely contain susceptibility genes for celiac disease. The most important genetic factor identified is the MHC region that contains the haplotypes coding for the celiac molecules HLA-DQ2 and HLA-DQ8. These two

haplotypes are necessary but not sufficient for the development of the disease. This suggests that other non-HLA genes contribute to pathogenesis of the disease .

In our study: the association of celiac disease and lupus erythematosus and systemic lupus erythematosus in brother and sister points to the almost certain existence of a genetic component.

Unfortunately the genetic study could not be done (lack of means). Similarly for the association of juvenile idiopathic arthritis and celiac disease disease, the genetic study could not be carried out .

In total:

The pathogenesis pathways involving the association of celiac disease and juvenile idiopathic arthritis juvenile idiopathic arthritis and systemic lupus erythematosus are not yet well elucidated.

The development of systemic lupus or juvenile idiopathic arthritis in a celiac patient is thought to be due to the celiac patient would be due to the presence of a common genetic background HLA B8 DR3 and to the duration exposure to gluten, and the contribution of IL-21 in the etiopathogenesis is considered to be today among the theories that can explain the association between celiac disease and autoimmune diseases.

Because of the existence of a common immunogenetic background, the association between celiac disease and autoimmune diseases or even JIA and SLE deserves the attention of attention of researchers. **No references in the discussion**

Conclusion: Is it a combination of two different autoimmune diseases or clinical-paraclinical manifestations within a single disease? **NOT RELATED TO THE TITLE OF STUDY**

Références :TOO SCANTY

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