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Etiology and Risk Factors Associated with Inflammatory Bowel Diseases

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Introduction

Inflammatory Bowel Disease (IBD) is a chronic, recurring, inflammatory disorder of the gastrointestinal tract. There are two type of IBD one is Ulcerative Colitis (UC) and another one is crohn's illness (Disc), which shows differences in term of the etiology, pathogenesis and clinical presentation. As per the latest knowledge, the etiology and pathogenesis of IBD are still inadequately explained. It is broadly acknowledged that the pathogenesis of IBD has association with hereditary factors and environmental aspects [1]. More than 100 genes have been distinguished based on genome wide association that ate associated with susceptibility of the individual to IBD [2]. Nonetheless, genetic susceptibility alone cannot totally account for high prevalence of IBD. IBD was first recognition of in European nations. Incidence and prevalence of IBD have increased dramatically during the past century. One of the recent studies analyzed the epidemiology of IBD and the association between environmental exposures and IBD.

This short communication provides an overview of the incidence, prevalence, and risk factors for inflammatory bowel disease. IBD, which encompasses Crohn's Disease (CD) and ulcerative colitis, is a long term, recurrent, inflammatory condition of the gastrointestinal tract. IBD prevalence is expanding around the world with rising incidence and prevalence in most nations. The pathogenesis of IBD has been discovered to be influenced by environmental factors, lifestyles, and other genetic factors. Smoking, the hygiene hypothesis, microbes, appendectomy, medicine, nutrition, and stress have all been linked to the IBD etiology, but the outcomes of several studies that are now accessible are conflicting on this subject. Therefore, additional research is needed to accurately identify the cause and comprehend the environmental factors that influence IBD.

IBD is a chronic, relapsing, and remitting illness with a still unclear origin. IBD is more common in some places than others and at particular seasons of the year. Since the 19th century, the prevalence of IBD has increased in both industrialized and developing nations, but it has started to drop in some regions. Western lifestyles have been linked to the high incidence and prevalence of IBD. Studies on the migrant population, meanwhile, show that immigrants who move from areas with low incidence to those with high frequency are more likely to develop IBD. Environmental exposures are therefore thought to play a key role in the etiology of IBD.

Smoking, the hygiene aspects, bacterial infections, appendectomy, medication, nutrition, and stress are a few environmental risk factors that are linked to IBD incidence. Studies have found rising risk variables, but the precise mechanism behind the link between environmental factors and IBD is still poorly understood. Clinicians are motivated to investigate environmental risk factors for IBD because of the genetic predisposition and symptoms of IBD.

Description

The chronic intestinal condition of "Inflammatory Bowel Disease" (IBD) frequently develops in young adults and progresses in a relapsing remitting pattern. Crohn's Disease (CD) and ulcerative colitis are both included under IBD. Immunosuppressive therapy, hospitalization, or surgery is frequently needed for the treatment of refractory disease or disease related consequences. A deregulated immune response to commensal flora in a genetically vulnerable host is the main mechanism leading to CD and UC.

Understanding the involvement of genes, the intestinal immune system, and the gut microbiota in the aetiology of CD and UC has significantly advanced in recent years. Nearly 163 unique genetic loci that affect the risk of either condition have been identified. More than two thirds of these loci were shared by the two disease conditions wherein 30 loci were only related with CD, and 23 alleles were associated with increased risk of UC. The susceptibility to infections and the host microbial response are affected by a number of these identified risk loci, supporting the interaction between the intestinal immune system and gut microbiota in the development of IBD.

However, less than one third of the heritability of either disease is explained by the extended panel of genes. Epidemiologic statistics indicate that a sizable portion of this disparity might be explained by external environmental factors. The rapid rise in IBD cases in nations where the condition was previously thought to be rare, which in many cases has paralleled industrial development indicates a potential contribution from environmental elements that are linked to the westernization of lifestyle. Immigrants from low-risk to high-risk nations tend to

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pick the disease risks of their new home nations over their countries of origin within two generations.

The major risk factor that is most frequently and continuously mentioned is smoking. However, it offers protection against UC while also raising the risk of CD. Numerous external variables may have an impact by altering the gut microbiome's composition or compromising the intestinal barrier. The use of antibiotics or nonsteroidal anti-inflammatory medicines, as well as the presence of enteric diseases, are some of the examples of these exogenous impacts. Dietary information has been patchy, although increased fibre intake especially soluble fiber seems to reduce the risk of CD, whereas protein intake might do the opposite. In people with CD, vitamin D may also play a significant preventive function. Stress and depression are neurobehavioral variables that influence the risk of IBD.

Newer risk variables, including as nutrition, hormone use, stress, and vitamin D status, have also been identified. Recent research studies have validated some of the previously known connections, such as smoking, and have revealed these newer risk factors. Systematic and rigorous studies of environmental exposures in disease management are required, especially if modification of environmental factors can be demonstrated to lower the risk of relapse, given the suboptimal rates of response to current immunosuppressive therapy and persistent concerns about long term safety and cost. The understanding of the pathophysiology of IBD has considerably improved thanks to developments in genetics and immunology.

It should also be noted that the evolving epidemiology of IBD is unlikely to be adequately explained by variations in specific environmental factors acting alone (either spatially or over time). In addition to underlying host genetics and microbial makeup, significant variation in sensitivity to each environmental effect undoubtedly exists among people, both in isolation and in codependence with other exposures [2].

Conclusion

The rapid rise in the incidence of Inflammatory Bowel Disease (IBD) supports the idea that the environment plays a significant role in its causation. In Canterbury, New Zealand, a population based case control study was conducted. Participants included 600 randomly chosen sexes and age matched controls as well as 638 prevalent Crohn's Disease (CD) cases, 653 prevalent Ulcerative Colitis (UC) cases. Comparing exposure rates to environmental risk variables. IBD was significantly associated with having a family history of the condition, smoking at diagnosis, being from a wealthy family at birth, being of Caucasian ancestry OR Living in a city linked to CD. Migrant status was linked to UC. Childhood vegetable gardening and breastfeeding both had duration response effects and were protective against IBD. Patients with CD had higher rates of tonsillectomy, appendectomies, infectious mononucleosis, and asthma. It is known that childhood environmental factors play a significant role in the emergence of IBD. Further research is needed to determine the duration-response protective relationship between breastfeeding and the emergence of IBD, as well as the preventative impact of growing vegetables as a child [3].

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