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Cardiac Ischemia in Patients with Inflammatory Bowel Disease

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Introduction

Idiopathic, persistent, and recurrent gastrointestinal inflammation is known as "inflammatory bowel disease" (IBD). Its pathogenesis is influenced by a number of variables, including the host's genetic predisposition, and it is accelerated by environmental and microbial causes. The two main subtypes of IBD are Crohn's disease (CD) and ulcerative colitis (UC), both of which are characterised by chronic intestinal inflammation. The most prevalent symptoms of these conditions are frequent diarrhoea, frequently accompanied by blood and pus in the stools, abdominal pain and cramping, fever, and weight loss [1]. IBD is still becoming more common and more prevalent all around the world. A wide range of extra-intestinal symptoms and IBD consequences have been described in addition to the core gastrointestinal issues of IBD because of chronic, long-lasting systemic inflammation. Due to the fact that ischemic heart disease (IHD) continues to be the main cause of mortality worldwide, it is important to identify individuals who have cardiovascular risk factors and take preventive actions.

About the Study

The presence of chronic inflammation in IBD increases the risk of atherosclerosis and coronary artery disease, which in turn increases the risk of acute coronary events. It can also cause endothelial dysfunction and platelet aggregation. Increased circulating inflammatory cytokines have been linked to increased carotid intimal thickness, endothelial dysfunction, and wall stiffness in IBD patients. As a result, both the pathogenesis of IBD and atherosclerosis involve a number of inflammatory mediators, such as elevated C-reactive protein (CRP) and circulating pro-inflammatory markers like tumour necrosis factor- (TNF-) and interleukins. IBD patients are more likely to develop IHD due to elevated levels of the aforementioned inflammatory mediators and elevated load of classic cardiovascular disease risk factors in the general population [2,3].

IBD and IHD have been linked in a number of large population studies, particularly in women and young patients, but the results are still debatable. This phenomenon's pathophysiological processes are not fully understood. We hypothesise that the higher frequency of conventional cardiovascular risk factors in males compared to women is what causes the difference in IHD risk between IBD and non-IBD men to become more pronounced. Additionally, younger IBD patients may have a higher risk of acute arterial events due to the varied effects of inflammation on different age groups. Higher CRP levels in women and the usage of birth control tablets could both play a role [4]. Obesity, type 2 diabetes mellitus (DM), hypertension, hyperlipidemia, smoking, and stress are traditional cardiovascular risk factors linked to IHD. Some of them Western lifestyles, ongoing stress, and tobacco use in CD are linked to both diseases.

IBD patients are frequently underweight because of malnutrition. However, with the rise of novel medicines that manage and sustain remission in IBD and the rising incidence of obesity in the general population, the prevalence of obesity can reach 40% of IBD patients. Obesity increases the risk of thromboembolism, surgical risk in UC, perianal injury, and the requirement for hospitalisation in CD. Hu's meta-analysis, however, shows that obese IBD patients have a better prognosis than non-obese individuals and are less likely to require hospitalisation, surgery, or corticosteroid medication [5].

Conclusion

IBD patients are more likely to develop IHD, especially women and young patients who are experiencing an IBD flare. Therefore, gastroenterologists and cardiologists should collaborate to screen for cardiovascular risk factors and optimise anti-inflammatory treatment in IBD patients. The management of IBD patients should focus on a multidisciplinary, team-based approach to preventive care, remission of IBD disease activity, and aggressive reduction of cardiovascular risk factors. Future prospective studies are required to identify biomarkers and scores for patient stratification, to comprehend shared etiopathogenic pathways, and to determine the best course of treatment.

Conflicts of Interest

The authors declare no conflict of interest.

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