Inflammatory bowel diseases (IBDs) including Crohn's disease (CD) and ulcerative colitis (UC) are chronic inflammatory gastrointestinal disorders. The pathogenesis of IBDs is multifactorial and is found to be related to the enteric microbiota and host immune system. Recovery of Escherichia coli pathotypes, especially adherent-invasive E. coli (AIEC), that adheres to the inflamed ileal and colonic mucosa of patients with CD, has attracted considerable interest over the past years. AIEC virulence genes can promote motility, serum resistance, capsule and lipopolysaccharide expression, iron uptake, biofilm formation, and adhesion to and invasion of epithelial cell lines. The AIEC phenotype strongly adheres to and colonizes ileal enterocytes by its type 1 pili to the carcinoembryonic antigen cell adhesion molecule 6 receptors. Colonization of bacteria to host cells activates the mucosal T helper type 17 and increases the level of tumor necrosis factor-alpha (TNF-α), interferon gamma, and interleukin 8. Moreover, the secretion of interferon gamma and TNF-α leads to the elevated expression, enhancing bacterial colonization. In a study by Small et al, persistent infection with AIEC strains in mice was associated with CD-like symptoms.

In recent years, numerous studies focused on the correlation between IBD and cardiovascular disease (CVDs) development. High levels of cytokines in IBD can contribute to endothelial dysfunction and atherosclerosis. Higher levels of coagulation factors which are frequently observed in IBD may predispose individuals to arterial thromboembolic events. The prevalence of venous thromboembolism varies between 1%-7% among patients with IBD. The blockade of TNF-α by monoclonal anti-tumor necrosis factor-alpha antibody (infliximab) was found to improve endothelial dysfunction in CD patients. A meta-analysis study of 9 papers, Singh et al reported that IBD was associated with a modest increase in the risk of cardiovascular disease, especially among women and young patients (<40–50-year old). The increase in risk was observed in patients with CD and UC. IBD was also associated with a 19% increase in the risk of ischemic heart disease both in patients with CD and UC.

The gut itself has an effect on atherogenesis by microbiota during IBD. In the case of AIEC, its products and adhesion led to the release of inflammatory factors from inflamed mucosa into the circulation. The induced rise of proinflammatory cytokines could contribute to endothelial damage, cardiovascular incidence, and atherosclerosis events. According to these data, it is possible that chronic infection with AIEC, which increases the chance of IBD in patients, can lead to CVDs.

Therapeutic strategies which target AIEC (e.g., the use of phage therapy, bacteriocins and antiadhesive molecules, prebiotics/probiotics and postbiotics, fecal transplantation, and combination therapy) may help the prevention and treatment of CD. In addition, restoring autophagy with immunotherapy may relieve active disease or prevent relapse. Antibiotics may influence the course of IBD by decreasing the concentrations of bacteria in the lumen. There is a hypothesis that the administration of antibiotics against AIEC can treat CD. In this way, CD remission in patients treated with broad-spectrum antibiotics such as ciprofloxacin, rifaximin, clarithromycin is shown in a meta-analysis.

Given that the susceptibility or resistance of bacteria to antibiotics is accessible, the theory that CVDs can be relieved by eradicating bacteria (i.e., pathogenic E. coli strains) that cause disease will not be far from expectation.

Conflict of Interest Disclosures
The authors declare that they have no conflict of interests.

Ethical Approval
Not applicable.

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