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Format: Abstract

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Irritable bowel syndrome and visceral hypersensitivity: risk factors and pathophysiological mechanisms.

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Abstract

Irritable bowel syndrome (IBS) is a common functional gastro-intestinal disorder, characterized by abdominal pain and altered intestinal motility. Visceral hypersensitivity is an important hallmark feature of IBS and is believed to underlie abdominal pain in patients with IBS. The two main risk factors associated with the development of IBS are gastrointestinal inflammation and psychological distress. On a peripheral level, visceral sensitivity seems to be modulated by several mechanisms. Immune cells in the mucosal wall, such as mast cells, and enterochromaffin cells may sensitize afferent nerves by release of their mediators. Furthermore, increased mucosal permeability, altered intestinal microflora and dietary habits may contribute to this feature. On a central level, an increased prevalence of psychiatric comorbidities is demonstrated in IBS patients, alongside alterations in the hormonal brain-gut axis, increased vigilance towards intestinal stimuli and functional and structural changes in the brain. The pathogenesis of IBS is complicated and multifactorial and the treatment remains clinically challenging. Dietary measures and symptomatic control are the cornerstones for IBS treatment and may be sufficient for patients experiencing mild symptoms, alongside education, reassurance and an effective therapeutic physician-patient relationship. New pharmacological therapies are aimed at interfering with mediator release and/or blockade of the relevant receptors within the gut wall, while modulation of the intestinal flora and diet may also be of therapeutic benefit. Tricyclic anti-depressants and serotonin reuptake inhibitors act both on a central and peripheral level by modulating pain signalling pathways.

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