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Abdominal pain in Irritable Bowel Syndrome: a review of putative psychological, neural and neuro-immune mechanisms.

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Abstract

Chronic abdominal pain is a common symptom of great clinical significance in several areas of medicine. In many cases no organic cause can be established resulting in the classification as functional gastrointestinal disorder. Irritable Bowel Syndrome (IBS) is the most common of these conditions and is considered an important public health problem because it can be disabling and constitutes a major social and economic burden given the lack of effective treatments. IBS aetiology is most likely multi-factorial involving biological, psychological and social factors. Visceral hyperalgesia (or hypersensitivity) and visceral hypervigilance, which could be mediated by peripheral, spinal, and/or central pathways, constitute key concepts in current research on pathophysiological mechanisms of visceral hyperalgesia. The role of central nervous system mechanisms along the "brain-gut axis" is increasingly appreciated, owing to accumulating evidence from brain imaging studies that neural processing of visceral stimuli is altered in IBS together with long-standing knowledge regarding the contribution of stress and negative emotions to symptom frequency and severity. At the same time, there is also growing evidence suggesting that peripheral immune mechanisms and disturbed neuro-immune communication could play a role in the pathophysiology of visceral hyperalgesia. This review presents recent advances in research on the pathophysiology of visceral hyperalgesia in IBS, with a focus on the role of stress and anxiety in central and peripheral response to visceral pain stimuli. Together, these findings support that in addition to lower pain thresholds displayed by a significant proportion of patients, the evaluation of pain appears to be altered in IBS. This may be attributable to affective disturbances, negative emotions in anticipation of or during visceral stimulation, and altered pain-related expectations and learning processes. Disturbed "top-down" emotional and cognitive pain modulation in IBS is reflected by functional and possibly structural brain changes involving prefrontal as well as cingulate regions. At the same time, there is growing evidence linking peripheral and mucosal immune changes and abdominal pain in IBS, supporting disturbed peripheral pain signalling. Findings in post-infectious IBS emphasize the interaction between centrally-mediated psychosocial risk factors and local inflammation in predicting long-term IBS symptoms. Investigating afferent immune-to-brain communication in visceral hyperalgesia as a component of the sickness response constitutes a promising future research goal.

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