Colonic diverticular disease: neuromuscular function abnormalities

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Colonic diverticular disease is an age-related disorder of the large bowel, characterized by outpouching of the colonic wall, that is relatively frequent in the general population, up to 50% in subjects over 75 years old [1]. It is commonly thought that abnormal colonic motility plays an important pathophysiologic role in this condition, even though other factors such as genetic predisposition, intrinsic anatomic features of the large bowel, colonic wall modifications with aging, dietary fiber, and abnormal intraluminal pressures are likely to interact each other to cause the onset of colonic outpouchings [2].

The abnormalities of contractile activity and perceptive capability of the large bowel observed in patients with colonic diverticulosis are the final events originating from more subtle alterations of both anatomical structures and of the physiological properties of the viscus. Thus, the researchers’ interest had initially focused on the muscular thickening often found in this disease [3], attributing it to increased intraluminal pressures [4]. However, this thickening is also related to colonic wall elastosis [4], and recent studies have shown that these patients display specific abnormalities in longitudinal muscle relaxation in addition to an increased elastin content of the colonic wall [5]. Also, there is evidence for an abnormal myogenic activity _in vitro_, in both basal conditions and in response to cholinergic stimulation, as well as a marked reduction of muscular contractile responses to tachykinins [6].

The decreased action of non-adrenergic non-cholinergic inhibitory nerves and other agents such as nitric oxide might determine high intraluminal pressure caused by colonic hypersegmentation [7], whereas the increase of vasoactive intestinal polypeptide (that
inhibits peristalsis) might initiate or contribute to the motility changes [8]. In addition, mucosal neurotransmitters may play some important role in the motility disturbance of these patients, as shown by the higher number of serotonin-containing cells [9], the significant attenuation of serotonin transporter expression and function in patients with recent history of acute diverticulitis, and the increase in colonic mucosal neuropeptides in symptomatic patients, which may reflect resolved previous inflammation [10]. Immunohistochemical studies have revealed that patients with colonic diverticular disease have significant reduction of interstitial cells of Cajal and enteric glial cells [11-13], two cell populations extremely important for the control of gut motor activity in health and disease [14]. Although the pathogenesis of this disorder relies on more than one abnormality, there is presently enough evidence to suggest that neuromuscular dysfunction may play a paramount role. However, due to the relative paucity of studies, more investigations are needed in this area, to hopefully help to establish in a definitive way the true role of this dysfunction.

References


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