Colonic wall remodeling in patients with short- and long-lasting ulcerative colitis

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Inflammatory bowel disease (IBD) are chronic and progressive pathologies associated with invalidating abdominal symptoms and increasing incidence in Western countries. Although fibrostenosis is a rare event in ulcerative colitis [UC], it may evolve to fibrosis in the later stages (1, 2). In the present study we examined the histopathological remodeling of the colonic wall from short- and long-lasting (SL and LL) UC patients. Full-thickness left colonic surgical samples were obtained from non-stenotic SL (≤ 3 years) and LL (≥ 10 years) UC patients with a severe exacerbation of colitis and active disease, without clinical signs of fibrostenosis. Collagen and elastic fibres, vascular networks and parameters of fibrosis have been evaluated by histochemistry, immunohistochemistry, confocal immunofluorescence and/or western blot. For comparison, normal colonic control samples from subjects who underwent surgery for uncomplicated colon cancer and without IBD and diverticular disease, were considered. Both SL- and LL-UC showed a thickening in tunica muscularis and activation of transmural neovessels, with proliferating CD105-positive endothelial cells and activated nestin-positive pericytes, as compared with controls. The colonic wall of LL-UC displayed a significant increase in collagen deposition and fibrotic markers (type I and III collagen, fibronectin, vimentin, RhoA), an enhancement of proliferation (PCNA), a decrease in elastic fibres, together with a fibrotic rearrangement of the tunica muscularis smooth muscle cells. In conclusion, a significant full-thickness remodeling of the colonic wall has been documented in the colonic tissues from both SL-UC and LL-UC, as compared to controls: transmural fibrotic thickening and angiogenesis were found in both UC groups, together with a cellular fibrotic/proliferative switch in the tunica muscularis which occurs in the later stages. These histopathological remodeling may contribute to the reduced elastic properties and tissue stretch capability of the wall, thereby impairing the occurrence of a normal motor activity in these subjects.

References

Keywords
Ulcerative colitis; active disease; colonic muscle remodeling; intestinal angiogenesis/fibrosis.