



Tissue remodelling in the colonic wall of patients with ulcerative colitis

<u>Chiara Ippolito</u>¹, Cristina Segnani¹, Sauro Dini¹, Barbara Battolla¹, Maura Castagna², Piero Buccianti², Amelio Dolfi¹ and Nunzia Bernardini¹

Inflammation-driven tissue remodelling may develop to fibrotic rearrangement. With regard to inflammatory bowel diseases, fibrotic remodelling has been evaluated in Crohn's disease, while little attention to such processes has been paid to ulcerative colitis (UC) [1]. The present study evaluated the distribution of connective tissue and angiogenesis in colon of patients with UC, paying particular attention to the tonaca muscularis, which is poorly considered in histopathological studies. Full-thickness left colonic samples were obtained from 10 patients with established, severe and pharmacologically unresponsive UC, who underwent bowel resection. Routine histology, histochemistry and immunohistochemistry were conducted in paraffin cross-sections. The distribution of collagen and elastic fibers was evaluated and quantified by both histochemical (Van Gieson, orcein, Verroheff staining) and immunohistochemical (anti-collagen I and III, anti-elastin) assays. The vascular network pattern was studied by anti-CD31 and nestin immunostaining. For comparison, the same evaluations were performed in healthy colonic control samples obtained from 10 subjects, who underwent surgery for uncomplicated colon cancer. A significant increase in collagen fibers and a decrease in elastin content were detected in the UC inflamed colon, as compared with controls. In particular, enhanced collagen deposition (mainly collagen type III) were found at level of submucosa, and tonaca muscularis within the longitudinal muscle (mainly along the serosal side) and circular muscle layer, and in perivascular connective tissue. By contrast, elastic fibers were significantly reduced throughout the whole muscle compartment, with particular regard for the myenteric ridge. Microvessel density was significantly higher in both submucosa and tonaca muscularis of colonic samples from UC patients compared with those from healthy control individuals. The present findings indicate that a significant tissue remodelling occurs in the inflamed colonic wall in patients with UC, also at the level of muscle layers. This rearrangement of the connective fibers and vascular network, together with the known alterations affecting the myenteric neurons and interstitial cells of Cajal, may contribute to the development of enteric dysmotility, and, accordingly, to the serious digestive symptoms which afflict patients with UC.

References

[1] Fiocchi and Lund. (2011) Themes in fibrosis and gastrointestinal inflammation. Am J Physiol Gastrointest Liver Physiol.

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¹Department of Human Morphology and Applied Biology, Faculty of Medicine, University of Pisa, Pisa, Italy

² Department of Surgery, Faculty of Medicine, University of Pisa, Pisa, Italy