The Role of Mast Cell Degranulation in Ischaemia-reperfusion-induced Mucosal Injury in the Small Intestine

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Abstract

The role of the intestinal mast cell system in the pathophysiology of postischaemic mucosal lesions is not understood. The present goals were to investigate the contributions of mast cells and mast cell-derived vasoactive mediators to mucosal injury caused by arterial occlusion. We evaluated the intestinal ischaemia-reperfusion-induced local morphological changes in mast cell-depleted anesthetized dogs. Animals subjected to complete segmental intestinal ischaemia and reperfusion served as controls. The selective mucosal-type mast cell degranulator Cremophor-El and the nonselective mast cell depleter Compound 48/80 were used to investigate the involvement of mast cells in reperfusion-induced tissue reactions. Ileal biopsies taken at the end of 120 min of ischaemia and after 120 min of reperfusion were evaluated histologically. The number of mast cells was determined and the degree of mucosal damage was evaluated according to the 0 to 5-grade Chiu scale. Mucosal histidine decarboxylase activity was measured in tissue biopsies and the rate of release of histamine was determined from the venous effluent of the segment. In the control group, 120 min reperfusion induced a severe tissue injury. In the Compound 48/80 and Cremophor-El-pretreated groups, the reduction in the baseline number of mast cells was 37% and 53%, respectively, and the basal mucosal histidine decarboxylase activity was significantly increased. In these groups, the ischaemia-reperfusion-induced release of histamine was significantly decreased, and the degree of damage of the intestinal mucosa was significantly reduced. Mucosal mast cell degranulation plays an important role in the initiation of tissue injury after intestinal ischaemia-reperfusion. Depletion of mast cells prior to ischaemia decreases the severity of mucosal damage, probably in consequence of the stimulation of mucosal histidine decarboxylase activity.

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Key words: Arterial occlusion, Histamine, Histidine decarboxylase, Intramucosal pH, Mucosal damage, Vascular resistance