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Involvement of mast cells in a mouse model of postoperative pain.

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Abstract

Recent studies have indicated that nearly half of all surgical patients still have inadequate pain relief; therefore, it is becoming increasingly more important to understand the mechanisms involved in postoperative pain in order to be better treated. Previous studies have shown that incisions can cause mast cell degranulation. Thus, the aim of this study was to investigate the involvement of mast cells in a model of postoperative pain in mice. The depletion of mast cell mediators produced by pre-treatment with compound 48/80 (intraplantar (i.pl.)) widely (98 ± 23% of inhibition) and extensively (up to 96 h) prevented postoperative nociception and reduced histamine and serotonin levels (88 ± 4% and 68 ± 10%, respectively) in operated tissue. Furthermore, plantar surgery produced immense mast cell degranulation, as assessed by histology and confirmed by the increased levels of serotonin (three-fold higher) and histamine (fifteen-fold higher) in the perfused tissue, 1h after surgery. Accordingly, pre-treatment with the mast cell membrane stabilizer cromoglycate (200 μg/paw, i.pl.) prevented mechanical allodynia (inhibition of 96 ± 21%) and an increase in histamine (44 ± 10% of inhibition) and serotonin (73 ± 5% of inhibition) levels induced by plantar surgery. Finally, local treatment with H(1) (promethazine, 100 μg/paw, i.pl.), 5-HT(3) (ondansetron, 10 μg/paw, i.pl.) or 5-HT(2A) (ketanserin, 5 μg/paw, i.pl.) receptor antagonists partially decreased postoperative nociception in mice, but when co-administered together it completely reversed the mechanical allodynia in operated mice. Thus, mast cell activation mechanisms are interesting targets for the development of novel therapies to treat postoperative pain.

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