Inflammopharmacology. 2020 Apr;28(2):551-561. doi: 10.1007/s10787-019-00670-w.
Epub 2019 Nov 25.

Clindamycin inhibits nociceptive response by reducing tumor necrosis factor-α and CXCL-1 production and activating opioidergic mechanisms

Felipe F Rodrigues [1], Marcela I Morais [1], Ivo S F Melo [1], Paulo S A Augusto [1], Marcela M G B Dutra [1], Sarah O A M Costa [1], Fábio C Costa [1], Franciele A Goulart [1], Alysson V Braga [1], Márcio M Coelho [1], Renes R Machado [2]

Affiliations
PMID: 31768707 DOI: 10.1007/s10787-019-00670-w

Abstract

Clindamycin, a bacteriostatic semisynthetic lincosamide, is useful in the management of infections caused by aerobic and anaerobic Gram-positive cocci, including bacteremic pneumonia, streptococcal toxic shock syndrome and sepsis. It has been recently demonstrated that clindamycin inhibits in vitro and in vivo inflammatory cytokine production. In the present study, we investigated the effects of clindamycin in acute and chronic models of pain and inflammation in mice and the underlying mechanisms. Intraperitoneal (i.p.) administration of clindamycin (400 mg/kg) increased the animal's latency to exhibit the nociceptive behavior induced by noxious heat (hot plate model). Intrathecal injection of clindamycin (2, 10 and 50 µg) also increased the animals' latency to exhibit the nociceptive behavior. Tactile hypersensitivity and paw edema induced by intraplantar (i.pl.) injection of carrageenan were attenuated by previous administration of clindamycin (200 and 400 mg/kg, i.p.). Clindamycin (100, 200 and 400 mg/kg, i.p.) also attenuated ongoing tactile hypersensitivity and paw edema induced by i.pl. injection of complete Freund's adjuvant (CFA). The antinociceptive activity of clindamycin (400 mg/kg, i.p.) in the hot plate model was attenuated by previous administration of naltrexone (5 and 10 mg/kg, i.p.), but not glibenclamide or AM251. CFA-induced production of TNF-α and CXCL-1 was reduced by clindamycin (400 mg/kg, i.p.). Concluding, clindamycin exhibits activities in acute and chronic models of pain and inflammation. These effects are associated with reduced production of TNF-α and CXCL-1 and activation of opioidergic mechanisms. Altogether, these results indicate that the clindamycin's immunomodulatory effects may contribute to a pharmacological potential beyond its antibiotic property.

Keywords: CXCL-1; Clindamycin; Inflammation; Opioids; Pain; Tumor necrosis factor-α.

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