THE USE OF KETOROLAC TROMETHAMINE ORAL RINSE FOR THE TREATMENT OF PERIODONTITIS IN ADULTS

P.F. CAVANAUGH JR.
Health Care Product Development, The Procter and Gamble Company, PO Box 8006, Mason, OH 45040–8006, USA

ABSTRACT


Periodontitis is a chronic infectious and inflammatory disease which affects approximately three quarters of the world's adult population over the age of thirty-five. Recently published studies have demonstrated that long-term administration (>6 months) of non-steroidal anti-inflammatory drugs (NSAIDs) can halt the progressive loss of alveolar bone associated with this disease. The safe and efficacious twice daily use for six months of 0.1% ketorolac tromethamine oral rinse for the prevention of periodontal disease progression in adults has recently been demonstrated. This presentation summarizes our results to date regarding the pharmacokinetics, pharmacodynamics, safety and efficacy of the topical oral rinse form of this potent NSAID in patients with periodontitis.

Keywords: alveolar bone, ketorolac tromethamine, oral rinse, periodontal disease, periodontitis, prostaglandins

INTRODUCTION

Periodontal disease is an infectious and inflammatory disease that is one of the most common chronic diseases among adults [1]. It is characterized by the presence of Gram-negative subgingival bacteria and an associated inflammatory host response that leads to loss of alveolar bone and connective tissue attachment within the periodontium. Periodontal pathogens can induce a number of inflammatory cytokines such as TNF-α, IL-1α, and IL-1β. Increases in IL-1 production lead to an activation of a number of prostaglandin E2- (PGE2) and cAMP-dependent events, including the induction of matrix metalloproteinase production [2]. This inflammatory cascade leads to a progressive loss of alveolar bone and surrounding connective tissue, both hallmark pathophysiological consequences of the disease. Clinically, this loss of tooth support is manifested by increased tooth mobility, loss of function, and, if left untreated, may lead to eventual tooth loss. In addition, relatively small losses of alveolar bone in multi-rooted teeth may lead to the development of furcation sites which are often difficult for the clinician to manage.

Many pieces of evidence point towards PGE2 as a critical mediator of the bone and connective tissue destruction that is observed in patients with periodontitis. As proposed by Offenbacher et al. [3], to be considered as a critical mediator, a molecule must be present in larger amounts in diseased tissue than healthy tissues; production of