CHAPTER I

INTRODUCTION

1.1 Statement of the problems and objectives

Treating skeletal open configuration cases is a challenge for orthodontists because of their multifactorial etiology. In addition, relapse tendency is high.[1-4] Treatment modalities for skeletal open configuration cases include incisor extrusion, molar intrusion and orthognathic surgery. Incisor extrusion is common for closing an open bite; however, it has limitations. In skeletal open configuration cases, incisors have already been over-erupted to naturally compensate for skeletal discrepancy, so incisor extrusion of overerupted incisors causes relapse.[5-6] During molar intrusion, anchorage control is very important. Unwanted tooth movements still occur during the application of intra-oral anchorage, such as incorporating as many teeth as possible into the anchorage unit. Extra-oral anchorage, such as headgear is sometimes inefficient, because the treatment outcome depends on patient compliance.[7] Recently, miniscrew implants have become widely used in many orthodontic treatments, including molar intrusion, to provide an absolute anchorage in order that less patientcompliance is required. Many investigations have reported successful results of miniscrew implant anchorage for molar intrusion.[7-11] However, these investigations assessed molar intrusion by using only clinical and radiographic parameters.

Orthodontic force causes metabolic changes in periodontal tissue. The mechanism of bone remodeling during orthodontic tooth movement results in alterations in the components of the gingival crevicular fluid, which can be assessed by monitoring inflammatory mediators (interleukin-1ß (IL-1 ß) or prostaglandin E_2 (PGE_2)), enzymes (alkaline phosphatase) and tissue breakdown products (GAGs) in gingival crevicular fluid (GCF).[12] During orthodontic tooth movement, the composition of extracellular matrix breakdown products in gingival crevicular fluid is altered. A previous study pertaining to the biochemical assessment of chondroitin sulphate levels in gingival crevicular fluid (GCF) of orthodontically moved canines showed that the detectable chondroitin sulphate levels were associated with the applied orthodontic forces.[13] Sari and Ucar[14] used the interleukin-1ß level in peri-miniscrew implant crevicular fluid (MICF) as a marker for assessing miniscrew implant stability. Another investigation relating to the monitoring of chondroitin sulphate (WF6 epitope) levels in peri-miniscrew implant crevicular fluid (PMICF) around miniscrew implants during orthodontic force application showed that the chondroitin sulphate in periminiscrew implant crevicular fluid could be precisely detected, and that the chondroitin sulphate (WF6 epitope) levels around failed miniscrew implant was remarkably high about 14 days prior to the miniscrew implant failure.[15] So, the present study was directed to biochemically assess tooth movement and miniscrew implant stability during orthodontic molar intrusion by monitoring chondroitin sulphate (WF 6 epitope) levels in GCF and PMICF.

1.2 Anticipated benefits

The benefit of this study is to justify the role of chondroitin sulphate levels as a biomarker for orthodontic tooth movement and miniscrew implant stability. If the chondroitin sulphate levels are associated with molar intrusion under orthodontic forces, orthodontists can use chondroitin sulphate levels as biomarkers for a non-invasive chair-side assessment for deeper alveolar bone remodeling around the teeth and around miniscrew implants during orthodontic tooth movement. Proper force magnitude for orthodontic molar intrusion in each patient may also be determined. Miniscrew implant failure may, therefore, be reduced or prevented.