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Effect of antifungal therapy on salivary β-defensin 1 concentrations in patients with oral candidiasis: A pilot study

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KEYWORDS
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The oral mucosal epithelium acts as a physical barrier against a range of pathogens, including *Candida* species. In addition, the epithelium produces antimicrobial peptides, which serve as chemical barriers to maintain homeostasis within the oral cavity. Human β-defensin 1 (hBD-1) is an antimicrobial peptide that plays a key role in the innate immune defense of the oral mucosa.[1] While most defensins are induced by infection and inflammation, hBD-1 is constitutively expressed. A decrease in the expression of hBD-1 has been linked to several diseases and infections, including oral candidiasis. Previous studies have reported that salivary hBD-1 concentrations are significantly lower in individuals with oral candidiasis compared to non-infected subjects.[2] However, it remains unclear whether antifungal therapy influences salivary hBD-1 concentrations. Therefore, this pilot study aimed to assess changes in salivary hBD-1 concentrations before and after antifungal therapy in patients with oral candidiasis.

Eight patients (5 women and 3 men; median age, 63 years; range, 46–76 years) diagnosed with oral candidiasis were recruited from the Department of Oral Medicine, Hokkaido University Hospital, between January 2020 and December 2021. Diagnosis was made according to previously published criteria.[3] The study protocol was approved by the Institutional Review Board of Hokkaido University Hospital (Approval No. 020–0049). All patients underwent topical antifungal therapy with amphotericin B, administered four times daily for two weeks. Unstimulated whole saliva samples were collected immediately before and after the treatment period. Samples were centrifuged at 4000 rpm for 20 min, and the resulting supernatant was stored at −80 °C until analysis. Total protein concentrations were determined using the Bradford assay, and all samples were standardized with phosphate-buffered saline (PBS). Samples were diluted at a 1:500 ratio and hBD-1 concentrations were measured using a commercially available enzyme-linked immunosorbent assay kit (Cloud-Clone Corp., Houston, TX, USA), following the manufacturer's instructions. Statistical comparisons were conducted using the Wilcoxon signed-rank test, with significance set at $P < 0.05$.

Prior to treatment, the median salivary hBD-1 concentration was 10 ng/mL (range: 0.13–16 ng/mL). After treatment, a significant increase was observed (median: 14 ng/mL; range: 6.9–20 ng/mL; $P < 0.05$) (Fig. 1). Six out of 8 patients tested negative for *Candida* species following therapy. Notably, most patients exhibited an increase in salivary hBD-1 concentrations regardless of complete fungal elimination.

In conclusion, antifungal therapy significantly increased salivary hBD-1 concentrations in patients with oral candidiasis in this study. The number of cases in this study was limited, and it remains unclear whether the reduction in salivary hBD-1 concentrations was due to impaired endogenous production or a consequence of oral candidiasis itself. However, these findings suggest that antifungal therapy may not only eliminate fungal infection but also contribute to the restoration of innate immune function in

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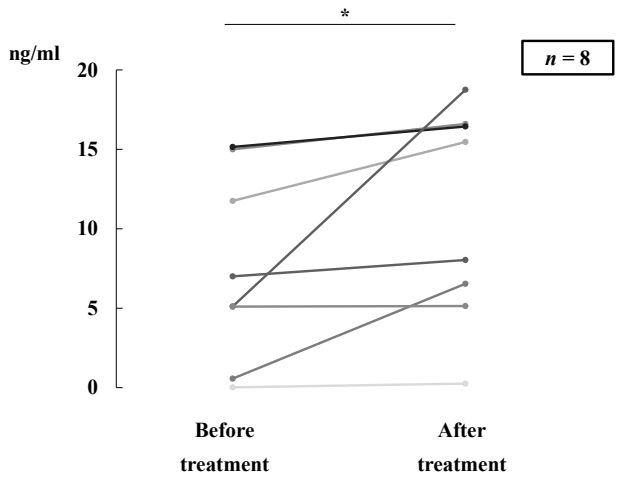

Figure 1 Comparison of t salivary hBD-1 concentrations before and after antifungal therapy in patients with oral candidiasis.

the oral mucosa. Therefore, prompt treatment of oral candidiasis may be essential, as delaying therapy or neglecting symptoms may impair immune defense and exacerbate the infection.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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Not applicable.

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