



## Author's reply to the letter to the editor of *Journal of the Korean Association of Oral and Maxillofacial Surgeons*

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To the Editor,

We value the considerations of Silveira et al.<sup>1</sup> on our article entitled, "An unusual presentation of peripheral ameloblastoma in the maxilla"<sup>2</sup>. We appreciate the insight into the diagnostic challenges and are well aware of the difficulties of differential diagnoses between pseudocarcinomatous hyperplastic cords and recurrence of solid plexiform ameloblastoma<sup>3</sup>. We welcome the opportunity to address several key aspects of the histopathological diagnosis, as their observations contribute to enriching the discussion of this complex diagnosis.

We agree that the histopathological diagnosis of peripheral ameloblastomas is challenging. The importance of adhering to the established histopathological criteria for ameloblastomas, first proposed in 1970<sup>4</sup> and currently endorsed by the World Health Organization (WHO), cannot be overstated<sup>5</sup>. In our case, multiple tissue sections were analyzed using conventional techniques, including routine hematoxylin and eosin staining, which clearly demonstrated areas of ameloblastomatous differentiation. These findings are critical, as the WHO criteria highlight key features such as basal cell polarization and subnuclear vacuolation in basal cells, which were identified in our case.

However, the presence of an inflammatory component in the connective tissue stroma. Prompted us to supplement the initial analysis with Masson's trichrome staining. This allowed clearer visualization of the extracellular matrix and

fibrosis, which are crucial in differentiating between neoplastic and reactive lesions. Immunohistochemistry and other complementary techniques such as the Masson trichrome method provide highly estimable data on the cellular profile, growth pattern, and basement membrane integrity, helping to differentiate between neoplastic and reactive lesions. The utility of Masson's stain in highlighting connective tissue elements has been well-established, particularly in cases involving potential fibrosis or inflammatory changes<sup>6,7</sup>.

Although an inflammatory component is not typically associated with peripheral ameloblastoma, we acknowledge that it can be observed in focal areas. We believe this feature, along with the core microscopic criteria, does not exclude the diagnosis of ameloblastoma. The Masson's trichrome stain enhanced our ability to examine the stroma, particularly in a lesion with such inflammatory characteristics.

We also want to emphasize that the histopathological significance of this case lies in the demonstration of the continuity between normal epithelial ridges and ameloblastomatous cords. These cords exhibited progressive structural, cellular, and immunohistochemical changes, characteristic of an incipient ameloblastoma. This transition from normal to neoplastic tissue adds an important layer of information to the diagnosis.

Regarding the distinction between ameloblastomatous epithelium and non-neoplastic reactive proliferative epithelium, we agree that this differentiation is crucial. Nevertheless, the indolent behavior of peripheral ameloblastoma and its low recurrence rate justify the clinical and pathological correlation in these cases.

Finally, while we agree with Silveira et al.<sup>1</sup> on the importance of distinguishing between reactive proliferative epithelium and ameloblastomatous epithelium, we believe that our findings, combined with both conventional staining and complementary techniques like immunohistochemistry and Masson's trichrome staining, provide a robust foundation for the diagnosis of peripheral ameloblastoma in this case.

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Once again, we appreciate the comments and thoughtful contributions of Silveira et al.<sup>1</sup> as they enrich the discussion of this rare and intriguing case.

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### Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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