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LETTERS TO THE EDITOR

Adenoid Ameloblastoma: The Histological Paradox

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Dear Editor,

We read with great interest the article titled 'Diagnostic Enigma of Adenoid Ameloblastoma: Literature Review Based Evidence to Consider It as a New Sub Type of Ameloblastoma' by Jayasooriya PR et al. [1]. We congratulate the authors on a detailed narrative on the debate prevailing on the existence of adenoid ameloblastoma. However, we would like to further contribute to the debate by sharing our thoughts.

Odontogenic tumors (OTs) are a heterogeneous group of lesions that are derived from odontogenic epithelium, ectomesenchyme and/or mesenchymal elements. Over the period of time, there have been innumerable modifications in terminologies and classification which has led only to confusion [1]. The usage of these terms is the result of sporadic case reports where neither a long term follow up was maintained nor the cases were supported by clear photomicrographs. One such terminology is "adenoid ameloblastoma" with sporadic cases described under various terms such as dentinoameloblastoma, adenoid ameloblastoma, adenoid ameloblastoma with dentinoid, plexiform ameloblastoma with dentinoid, atypical plexiform ameloblastoma with dentinoid and dentinoameloblastoma with ghost cells [2–7]. There is no uniform international consensus and neither such terms are included in the WHO classification of odontogenic tumors [8]. All of these aforementioned tumors were similar histologically, causing more confusion. Inductory changes are not commonly seen in ameloblastomas and their clinical relevance is not comprehensively studied yet. Such elements are considered as the products of the supporting stroma rather than the neoplastic elements. Thus, making a diagnosis depending upon such elements is of no practical utility. Practically, an oral surgeon is never interested in the detailed histology of any lesion; all he wants is a straightforward diagnosis on which the entire treatment plan relies upon.

The so-called glandular pattern in various published cases could be due to cystic degeneration of the stroma resulting from self-strangulation rather than true adenoid spaces.

Vilanova et al. have demonstrated that the degree of degenerative change correlates with tumor size and vascular abnormalities in the tumor which led the authors to postulate that an increase in the mass of the tumor would lead to vascular insufficiency which in turn can promote degenerative changes in benign neural tumors [9]. We believe that a similar phenomenon could have led to the formation of pseudoglandular pattern in large plexiform ameloblastomas.

Most of the cases reported in the literature did not show any signs of recurrence which can be attributed to the presence of dentinoid structures. It is commonly believed that the mechanism of dentinoid formation in these cases may be attributed to an inductive effect of the odontogenic epithelial component and thus logically cannot have any correlation with the biological behavior of the tumor [10]. Metaplastic changes in the connective tissue may be considered as an alternative justification for the presence of such dentinoid material [10]. Papagerakis et al. have recently given a reasonable reason for this explanation by demonstrating that, gene products that are commonly expressed by ectomesenchymal cells can be expressed by ameloblastic cells as well in mixed odontogenic tumors and thereby exhibiting co-expression of mesenchymal phenotype [11, 12]. Thus, it is possible that dentinoid which exist in some tumours could have been a product of epithelial cells with ameloblastic differentiation in those tumors. However, these novel findings are divergent from currently popular concept of odontoblasts being the only cells capable of dentin formation as they are of ectomesenchymal origin; future studies are needed to explain this phenomenon in detail.

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In conclusion, we would like to highlight the fact that classical plexiform ameloblastoma may show pseudo-glandular pattern, dentinoid changes, hyalinized vessels and calcifications. Given the significantly enigmatic potential of the odontogenic epithelium, it seems logical to believe that the inductor changes might be of histological interest but least prognostic value.

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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