ABSTRACT

There is considerable confusion regarding the origin and classification of the AOT with the most recent WHO classification including the AOT as a non-inductive tumour or hamartoma showing metaplastic mineralisation. This study reviewed the clinical and epidemiological features of 51 AOTs retrieved from the archives of the Division of Oral Pathology, University of the Witwatersrand. In addition a detailed histological analysis, including histochemical and immunohistochemical investigations, was undertaken with a view to provide evidence for induction in AOTs.

4μ haematoxylin and eosin sections were examined. Selected cases were stained with PAS, alcian blue at pH 2.5, Congo red, reticulin, mucicarmine, von Gieson, Masson's trichrome and Prussian blue. Melanin bleach was performed on certain sections. Immunohistochemistry was performed in the presence of adequate preparations and controls with MNF 116 and Vimentin antisera.

Analysis of the clinical and epidemiologic data revealed that the AOT in our series had the same clinicopathological features as those reported from other parts of the world. This data will be included in a review article being prepared to commemorate the 100th anniversary of the description of this lesion.

AOTs occur in both follicular (64%) and extrafollicular forms (21%) most commonly in the anterior maxilla (62.7%) in females (63.6%) in the second decade (66.6%) where they are frequently associated with unerupted canines (42%). There is some evidence

suggesting that extrafollicular AOTs may originate in other odontogenic cysts and that this might explain why some AOTs grow to a large size and behave aggressively causing root resorption and expansion.

Histologically the unique and important presence of tall columnar cells resembling ameloblasts or odontoblasts were identified in 5 cases of AOT. These cells were arranged in a circular configuration and were actively secreting PAS positive material, which we have interpreted as dental matrix material. We have called these *'circular secretory units'*. The tall columnar cells did not always surround the entire secretory unit suggesting either that there was a variable rate of differentiation or that having completed their function these cells change shape and become unrecognisable. We regard these *circular secretory units* as providing definite evidence of induction. Further evidence of induction is provided by the presence of clusters or strands of odontogenic epithelium intimately associated with a lace-like pattern of dental matrix material. No evidence of residual ectomesenchyme was found, but this does not rule out the possibility that induction has indeed occurred.

We can also find no evidence linking the *circular secretory units* with the pseudo-ductular spaces, which characterise the AOT.

In conclusion, based on our observations, we recommend that the AOT be classified as a benign tumour with inductive capacity.