International Endodontic Journal
doi:10.1111/j.1365-2591.2009.01546.x

Response

Periapical calcifying odontogenic cyst

Dear Editor

We thank Professor Fumio Ide for his letter concerned with our article 'Persistent apical periodontitis associated with a calcifying odontogenic cyst' (Estrela *et al.* 2009). Obviously, this diagnosis was not arrived at without considerable thought. Usually, lesions related to dental development are not classically associated with inflammation. The specimen removed from this case was associated with an intense inflammatory infiltrate adjacent to the covering epithelial tissue. We agree that on the microphotographs presented a squamous stratified epithelium can be observed, which commonly does not characterize an odontogenic cyst. However, we are convinced that the inflammatory environment through cytokines, growth factors and components of the extracellular matrix can induce metaplasia of this epithelium (Kumar *et al.* 2005). It is important to emphasize that other authors also observed a lining epithelium on a lesion with the same diagnosis of calcifying odontogenic cyst (Satoshi *et al.* 2003). Our microscopic diagnosis was established taking into account that in some areas of the lining epithelium characteristics of ameloblastic epithelium were apparent, with loose areas similar to the stellate reticulum, along with cells in the process of dyskeratosis and ghost cell as observed in areas of the classic calcifying odontogenic cyst. The microphotographs that follow may help the visualization of these events (Figs 1 and 2).

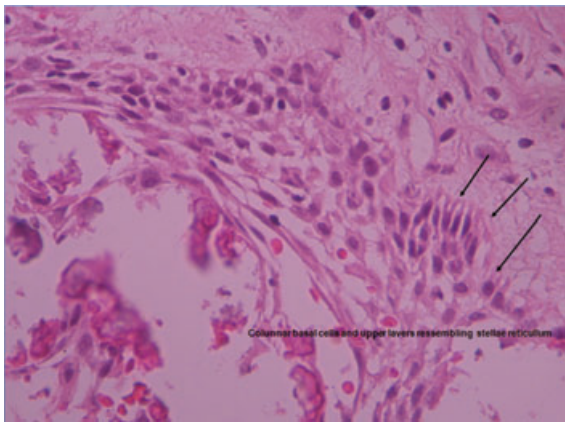

Figure 1 Tall columnar basal cells and upper layers resembling stellae reticulum (HE staining, original magnification ×40).

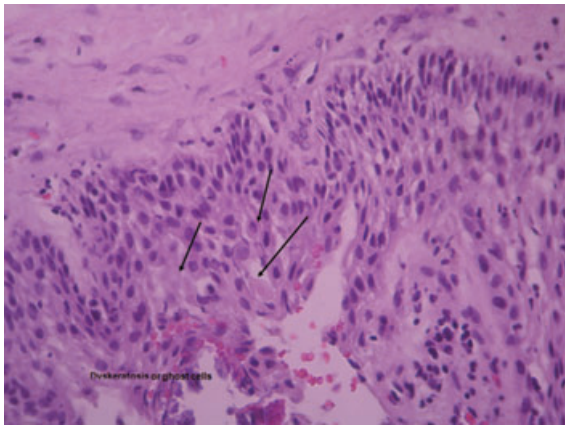

Figure 2 Cells in process of dyskeratosis at periphery of the epithelium (HE, original magnification ×400).

Concerning the presence of a focus of dystrophic calcification in radicular cysts, as referred by Professor Ide (Sciubba *et al.* 2001, Shear & Speight 2007), we agree. Nevertheless, these calcifications are focal and associated with old cysts, generally residual cysts of inflammatory nature and not in all the extension of the area of the covering epithelium (High & Hirschman 1986), as observed in our case report.

As mentioned by Professor Ide in relation to the lack of a discussion on radicular cysts and that the implication of this entity was not considered in the primary diagnosis during the clinical investigation of the patient, we would like to clarify that the asymptomatic nature of apical periodontitis, as described in our discussion, makes the possibility of the lesion being a radicular cyst implicit, as well as the possibility of it being a radicular granuloma or other lesion of nonendodontic origin (Estrela 2004).

In summary, we agree that this case is controversial. However, we consider that the inflammation associated with the main lesion and the procedures prior to the surgical treatment may have contributed to the proliferation of the epithelial lining with metaplasia altering the classic form of the calcifying odontogenic cyst. We recognize, following the letter from Professor Fumio

Ide, that we should have considered the influence of the inflammation on the classic microscopic characteristics of the calcifying odontogenic cyst in our discussion section. However, we remain convinced of the diagnosis based on the evidence and careful review of our histological sections.

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