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# EXPRESSION OF ALPHA-SMOOTH MUSCLE ACTIN IN ODONTOGENIC CYSTS AND TUMORS

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***Background*: Odontogenic cysts and tumors exhibit different degrees of aggressiveness in their biological behavior. There has been evidence that the presence of myofibroblasts (MFs) at the invasion front promotes tumor invasion. Our study is based on the fact that MFs are important in the biological behavior of odontogenic cysts and tumors. *Aim*: To assess immunohistochemically expression of alpha-smooth muscle actin (α-SMA) of MFs in odontogenic cysts and tumors and correlate this expression to their biological behavior. *Materials and Methods*: The archival tissues collected for 1.5 years were obtained from the Department of Oral Pathology & Microbiology, People's Dental Academy, Bhopal (India). A total of 40 cases consisting of 10 cases each of odontogenic keratocysts, radicular cysts, dentigerous cysts and ameloblastomas formed the study group. An immunohistochemical analysis of α-SMA expression and localization was carried out. *Results*: Mean MF counts were the highest in odontogenic keratocysts which was followed by ameloblastomas, entigerous cysts and radicular cysts. Weak α-SMA-expression was found in 50% of cases, moderate in 22.5% of cases, and intense — in 10% cases. MFs were arranged in the spindle, focal, or network patterns in 35; 27.5 and 20% of cases, respectively. *Conclusion*: The analysis revealed that the MFs were distinctly heterogeneous in distribution and pattern of arrangement. This provided persuasive evidence that stroma of these lesions harbor MFs as reflected by α-SMA immunopositive cells.**

***Key Words*: α-SMA, myofibroblasts, odontogenic cysts, odontogenic tumors, immunohistochemistry.**

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Epithelial-mesenchymal interactions are essential in maintaining homeostatic equilibrium in embryonal as well as adult tissues, with the stromal cells maintaining control over cell size, function and response to wounds and other pathologic conditions through modification of the extracellular matrix [1]. Myofibroblasts (MFs) play a role in the synthesis of extracellular matrix (ECM) and in force generation, which results in ECM reorganization and tissue contraction during wound healing. During disease evolution, the stroma composed of inflammatory cells, small vessels, fibroblasts and MFs, and ECM components, reflects disturbed interactions between the pathologic cell population and its surroundings [2]. Various epithelial-associated factors have been indicated and are now considered to play a vital role in promoting relative aggressive biological behavior of the odontogenic epithelium that includes increased proliferative potential [2]. The differentiation of MFs in the stroma has an important function because of their potential to produce collagen and synthesize enzymes, which could be associated with tumor growth and progression [3]. MFs are regarded as cells of uncertain position in the morphologic spectrum between fibroblasts and smooth muscle cells [3]. Over the past 10 years, there has been abundance of evidence that the presence of MFs at the invasion front is not part of the host defense mechanism against tumor invasion, but actually promotes it [4]. MFs are modulated fibroblasts that express alpha smooth muscle actin (α-SMA), which is a key contractile protein and is the actin isoform typically found in vascular smooth muscle cells and coordinately regulated by transforming growth factor beta (TGF-β) found in smooth muscle cells [5]. α-SMA plays an important role in fibrogenesis. Anti-α-SMA antibody labels smooth muscle cells, MFs and myoepithelial cells. It reacts specifically with the α-smooth muscle isoform of actin. Immunohistochemical studies have made it possible to explore the role of stroma in the progression of odontogenic cysts and tumors. There are not much studies on MFs expression in odontogenic keratocysts (OKCs), radicular cysts (RCs) and dentigerous cysts (DCs) and odontogenic tumors (ameloblastoma (AMs) with its variants). Anusai *et al*. [4] and Annegowda *et al*. [5] in their studies on MFs in odontogenic cysts and tumors concluded that OKCs and AMs show higher amounts of α-SMA. Our study is based on the fact that MFs are important in the biological behavior of odontogenic cysts and tumors. We carried out immunohistochemical analysis of MFs by assessing expression of α-SMA and correlated this expression to their biological behavior. We aimed to examine the role played by stroma, with special emphasis on MFs, in the behavior of odontogenic cysts and tumors.

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*Abbreviations used*: α-SMA – alpha smooth muscle actin; AMs – ameloblastoma; DCs – dentigerous cysts; ECM – extracellular matrix; MFs – myofibroblasts; OKCs – odontogenic keratocysts; RCs – radicular cysts; TGF-β – transforming growth factor beta.

## MATERIALS AND METHODS

Formalin-fixed paraffin embedded tissue blocks of biopsy specimens, which were histopathologically diagnosed as OKCs, RCs, DCs and AMs were randomly collected from the archives of Department of Oral Pathology and Microbiology, People's Dental Academy. Clinical information including age, sex, and the location of the lesion was extracted from patients' files. To confirm the diagnosis and inclusion of the samples in the study, first 3–4 micron sections were stained using hematoxylin-eosin staining protocol. Finally, 40 paraffin blocks were included, and 4 groups (OKCs, RCs, DCs and AMs) were made, each consisting of 10 cases. The cases with inflammation, insufficient tissue and incisional biopsy were excluded from the study.

3-μm thick sections were cut and mounted on super frost slides. Sections were dewaxed, washed in alcohol and antigen retrieval was carried out in a pressure cooker with 10 mM citrate buffer solution for 15 min. Endogenous peroxidase was blocked by 0.3% hydrogen peroxide in methanol at RT for 10 min. Slides were washed twice with Tris buffer briefly and incubated with anti-α-SMA antibody (Dako, Denmark) for 60 min. The sections were washed with PBS and incubated with goat-anti-mouse EnVision–HRP–enzyme conjugate. Diaminobenzidine (DAB) was used as the chromogen. The sections were then counterstained with hematoxylin and mounted.

Fields with immunopositive cells were selected in ×10 objective lens and subsequently observed in ×40 objective lens after which the fields were digitally captured using Magnus pro USB camera 2.0. For cystic lesions, the objective lens was placed immediately beneath the cystic epithelial lining and for solid tumors (odontogenic tumors) it was placed adjacent to the periphery of the tumoral islands/nests/cords. α-SMA positive cells within blood vessel walls served as a positive control. The intensity of α-SMA-immunostaining was assessed semiquantitatively as weak, moderate, or intense within the connective tissue. The percentage of α-SMA immunopositive cells were recorded as 0 (nil) = no positive cells, 1 (weak) = 1–25% cells, 2 (moderate) = 26–50% cells and 3 (intense) = 50–100% positive cells. Considering the distribution pattern of MFs, the arrangements of positive-stained cells were classified into 3 groups as follows:

1. Focal: If MFs had a focal arrangement or had no special arrangement in different areas of connective tissue and stroma.

2. Network: MFs arranged in multiple rows with an interwoven network of cytoplasmic extensions forming a network in the stroma of the connective tissue.

3. Spindle: MFs arranged in one to three rows in a regular order in the periphery of the neoplastic islands or in the connective tissue with distinctive cell margins around tumor islands and malignant tissue (Fig. 1).

Differences in the distribution of α-SMA expression among groups and cell population were compared using chi-square test and Kruskal — Wallis test. Significance was set at $p < 0.05$.

## RESULTS AND DISCUSSION

A total of 40 cases comprising OKCs, RCs, DCs and AMs, each with 10 cases formed the study group. Mean age of OKC patients was 36.2 years, RC — 39.2 years, DC — 29.3 years and AM — 35.2 years. There was an almost equal distribution of males and females (21 and 19) respectively. In OKCs, occurrence was slightly more in females (60%) as compared to males (40%). There were equal number of males and females in RCs. Out of 10 DCs cases, 9 cases were males and 1 was female. In AMs there was a preponderance of females (70%).

A total of 33 cases showed variable percentages of positivity for α-SMA. RC showed maximum positivity (n = 9) which included weak, moderate and intense α-SMA expression. This was followed by other groups with (n = 8) each.

50% (n = 20) of cases exhibited weak α-SMA-expression. In the rest of the samples moderate (22.5%) and intense (10%) expression was observed. 17.5% showed a negative expression. The differences were not significant ($p = 0.97$) (Table 1).

Distribution of MFs pattern was mostly spindle form (n = 14) out of which (n = 7) occurred in AM (Fig. 2). The other most prevalent pattern was focal. Focal distribution was equal (n = 5) each in RC and DC. OKC showed more network pattern (n = 5) (Fig. 3) than spindle pattern (n = 2) (Fig. 4). AMs and DCs showed no focal and network distribution, respectively. The difference was significant ($p = 0.019$) (Table 2).

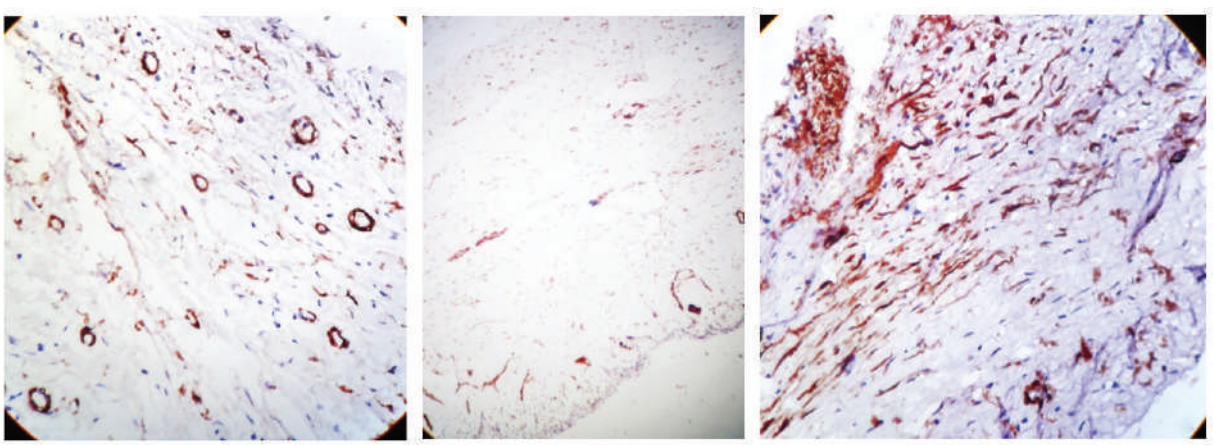

**Fig. 1**. Various patterns of MF arrangements: *a* — focal; *b* — network; *c* — spindle; ×5

Mean value of MF count was (26.15 ± 19.052) in OKC, RC (9.69 ± 8.320), DC (12.58 ± 8.320) and AM (20.73 ± 17.891). There was no significant association observed between the groups and mean MF count ($p$ = 0.162) (Table 3).

OKCs, DCs, RCs and AMs are the most commonly encountered odontogenic lesions. MFs were first discovered by Gabbiani *et al.* in the early 1970's and it was shown to actively promote dermal wound contraction. MFs are differentiated fibroblasts that express α-SMA and have intermediate characteristics between classic fibroblasts and smooth muscle cells [6].

OKCs occur over a wide age range and cases have been recorded with peak incidence in the second and third decade with gradual decline thereafter [7]. The age distribution in our study showed peak incidence in the third and fifth decades followed by the fourth decade. OKCs generally are found more frequently in males than in females. We found female predilection in our study. The mandible was involved more frequently than the maxilla. The percentage of OKCs occurring in the mandible ranges from 65 to 83% and recurrence rate 2.5 to 62% [8]. In our study, the mandible was affected in 70% of OKC, and the most common site was the posterior mandible. This has been observed in various studies.

DCs account for approximately 16.6% of all jaw cysts [9]. They occur over a wide age range with a peak frequency in the 2nd to 4th decades. Present study revealed the age of patients ranging from 9 years to 60 years with peak incidence in the 2nd decade. Maxilla had slightly more predilection in our study and the anterior (30%) and posterior (30%) region was the most frequent site, followed by the posterior mandible (10%).

RCs are the most common of all jaw cysts and comprise about 52 to 68% of all the cysts affecting the human jaws [8]. Anatomically, the apical cysts occur in all tooth-bearing sites of the jaws but are more frequent in maxillary than mandibular teeth. In the present study most RCs occurred in the 4th decade followed by the 3rd decade, whereas male and female were equally (n = 5) involved. In the maxilla, the anterior region appears to be more prone to cyst development whereas in the mandible the RCs occur more frequently in the premolar region. In the present study 40% maxillary anterior and 40% posterior maxilla were involved. This is in accordance with various studies and our results were comparable [10].

AMs of the jaws are relatively less common odontogenic lesions excluding cysts and represent 13–54%

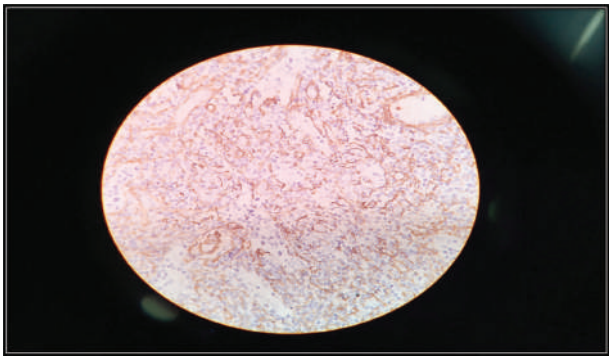

**Fig. 2**. Spindle pattern of α-SMA expression in AM, ×20

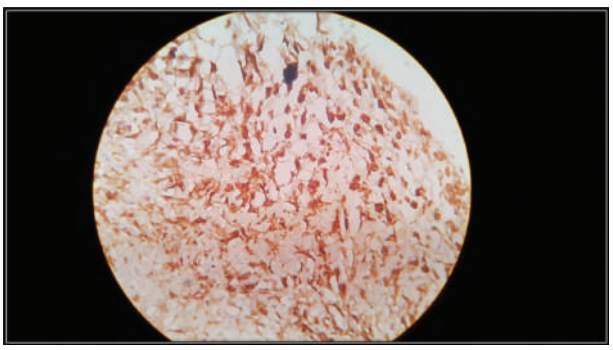

**Fig. 3**. Network pattern of α-SMA expression in OKC, ×40

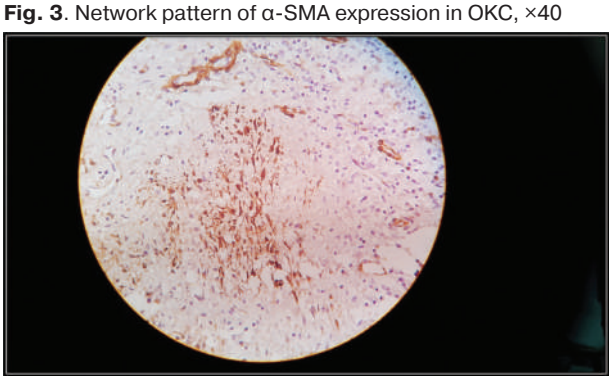

**Fig. 4**. Spindle pattern of α-SMA expression at the peripheral margin of OKC, ×20

**Table 1.** Distribution of α-SMA expression in the groups

| α-SMA expression | OKC, n (%) | RC, n (%) | DC, n (%) | AM, n (%) | Total, N (%) |
|---|---|---|---|---|---|
| Nil (0–0) | 2 (20.0%) | 1 (10.0%) | 2 (20.0%) | 2 (20.0%) | 7 (17.5%) |
| Weak (1–25%) | 4 (40.0%) | 7 (70.0%) | 4 (40.0%) | 5 (50.0%) | 20 (50.0%) |
| Moderate (26–50%) | 3 (30.0%) | 1 (10.0%) | 3 (30.0%) | 2 (20.0%) | 9 (22.5%) |
| Intense (50–100%) | 1 (10.0%) | 1 (10.0%) | 1 (10.0%) | 1 (10.0%) | 4 (10.0%) |
| Total | 10 | 10 | 10 | 10 | 40 |
| Chi-square | | | 2.851 | | |
| *p* | | | 0.970 | | |

**Table 2.** Distribution of MF patterns in the groups

| α-SMA patterns | OKC, n (%) | RC, n (%) | DC, n (%) | AM, n (%) | Total, N (%) |
|---|---|---|---|---|---|
| Focal | 1 (10.0%) | 5 (50.0%) | 5 (50.0%) | 0 (0.0%) | 11 (27.5%) |
| Network | 5 (50.0%) | 1 (10.0%) | 0 (0.0%) | 2(20.0%) | 8 (20.0%) |
| Spindle | 2 (20.0%) | 2 (20.0%) | 3 (10.0%) | 7 (70.0%) | 14 (35.0%) |
| Negative | 2 (20.0%) | 1 (10.0%) | 2 (20.0%) | 2 (20.0%) | 7 (17.5%) |
| Total | 10 | 10 | 10 | 10 | 40 |
| Chi-square | | | 19.8 | | |
| *p* | | | 0.019 (significant) | | |

**Table 3.** Mean MF counts in the groups

| Groups | Mean ± SD |
|---|---|
| OKC (n = 10) | 26.15 ± 19.052 |
| RC (n = 10) | 9.69 ± 8.320 |
| DC (n = 10) | 12.58 ± 8.320 |
| AM (n = 10) | 20.73 ± 17.891 |
| Total(n = 10) | 17.29 ± 15.305 |
| Kruscal – Wallis test 5.137 | |
| $p = 0.162$ | |

of all jaw tumors [11]. AMs affect males and females equally, peaking in the third decade, with a wide age range of first-to-ninth decade. In our study, predominantly female predilection (70%) was observed with the peak incidence present in the third decade followed by the fourth decade and least in the fifth decade. In our study, almost all cases of AMs presented in the posterior region of the mandible and our study result showed a significant difference. Similar results were in 1995 observed by Reichart *et al*. [12].

Lombardi *et al.* [13] demonstrated strong expression of MF mainly in subepithelial, midzone and periphery of OKC, DC, RC and epidermoid cysts. We observed two distinct zone i.e. subepithelial and periphery expression in both OKCs (50%), DCs (50%) and RCs (40%). However, the majority (36%) of them exhibited expression in the outer zone (peripheral) next to the host bone.

In agreement with Lombardi *et al.* [13], we support the view that mechanical tension of stroma plays a major role in MFs differentiation. Mechanical tension produced in the stroma directly modulates the bioactivity of TGF-β1, major cytokine inducing MF differentiation. The deeper aspect of the cyst wall is close to the bony interface, which is a rigid structure, and produces mechanical stress within the stroma of the cystic wall that eventually leads to trans-differentiation of more number of MFs. These trans-differentiated MFs because of their contractile property might produce pressure on underlying bone leading to osteoclastic differentiation and bone demineralization. After demineralization of bone by these activated osteoclasts, the organic component is further degraded by the extracellular proteinases secreted by these cells [14].

Adegboyega *et al.* [15] used α-SMA immunohistochemical staining on MFs, positive vimentin for normal colon mucosa, hyperplastic polyps and colorectal adenomatous in their research. The results of this study showed that there seems to be a relationship between cell arrangement pattern and tumor invasive behavior. It can be said that because of the higher number of MFs in network arrangements they show more severe invasive behavior in comparison to spindle arrangement.

The present study provides persuasive evidence that stroma of lesions harbors MFs as reflected by α-SMA immunopositive cells. Our study also revealed that the MFs are distinctly heterogeneous in distribution and pattern of arrangement (focal, network and spindle). The immunohistochemical expression profile of α-SMA has been described in three patterns; namely spindle, network, focal. In the present study, we observed that the majority of the lesions showed the spindle pattern that were interconnected in a network-like manner which is in accordance with the observations reported by Vered *et al.* [16].

In our study of the OKCs, apart from the location in the deeper aspect of connective tissue, MFs also showed subepithelial network (n = 5). Whereas in RCs and DCs MFs showed subepithelial focal type (n = 5 each) and in AMs MFs showed spindle (n = 7) arrangement.

Vered *et al.* [16] found that α-SMA expression in the stroma of solid AMs and OKCs (parakeratinized type) was higher than DCs and unicystic AM and ameloblastic fibro-odontoma. They concluded that α-SMA expression of MFs was an index of invasive behavior of odontogenic lesions and it seems that target therapy can be beneficial as an auxiliary method for treatment of more invasive lesions.

Gabhane *et al.* [17] evaluated quantitatively stromal MFs in odontogenic cysts and found that OKCs had the highest mean number of α-SMA positive cells per field followed by DCs, while RCs had the lowest number of such cells. In our study, we found that the mean number of MFs count per field was the highest in OKCs, followed by AMs, DCs and RCs being the lowest (Table 3). Although results were non-significant in our study, we observed more positivity in OKCs than others. This showed that there could be a certain role of MFs responsible for the aggressiveness. Similar results were also observed by Anusai *et al.* [4] and Annegowda *et al.* [5].

Shruthi *et al.* [18] found that the mean number of MFs in AM was higher than OKC. Our results showed that the mean number of MF counts was higher in OKC than AM. Thus, a positive link can be suggested, when more MFs are present in the stroma, a more aggressive behavior of the odontogenic cyst/tumor can be anticipated.

In a study of 24 cases of AMs by Sherlin *et al.* [19], α-SMA positivity was noted at the tumor front of the lesion, which clearly indicates the pivotal role of SMA positive MFs in tumor progression.

We observed weak α-SMA positivity (50%) in AM cases suggesting that the behavior of the tumor may not be dependent entirely on stromal elements but also on other factors. Our results are in contrast to the results observed by Vered *et al.* [16] and Mashhadiabbas *et al.* [20].

Recent studies have shown that smooth muscle fibroblasts may originate from fibroblasts within the tumor stroma or even from carcinoma cells by the process of epithelial-mesenchymal transition. Several cytokines, including TGF-β, platelet-derived growth factor, interleukin-4 and insulin-like growth factor II have been reported to induce myofibroblastic differentiation. Cancer cell derived TGF-β1 stimulation is the key cytokine. In recent years, the tumor microenvironment has become the focus of intense research, with the understanding that the alterations that occur in the tumor stroma might prove useful in prognosis and generate new therapeutic targets.

In conclusion, the present study semi-quantitatively evaluated the presence of MFs in stroma of OKCs, RCs, DCs and AMs. We revealed that the MFs were distinctly heterogenous in distribution and pattern of arrangement. This provided persuasive evidence that stroma of these lesions harbor MFs as reflected by α-SMA immunopositive cells.

The studies correlating the expression of MFs and biological behavior of these odontogenic lesions with special emphasis on recurrence and local invasion are essential in understanding the true nature of these lesions.

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## ЕКСПРЕСІЯ АЛЬФА-АКТИНУ ГЛАДЕНЬКИХ М'ЯЗІВ В ОДОНТОГЕННИХ КІСТАХ ТА ПУХЛИНАХ


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***Стан питання***: Одонтогенні кісти та пухлини розрізняються за ступенем їх агресивності та біологічними властивостями. Наявність міофібробластів (МФ) у фронті інвазії спричиняє інвазію пухлини. Таким чином, МФ є важливими щодо біологічної характеристики одонтогенних кіст і пухлин. ***Мета***: Визначити імуногістохімічно експресію альфа-актину гладеньких м'язів (α-АГМ) в одонтогенних кістах і пухлинах та пов'язати характер такої експресії з біологічними властивостями пухлин. ***Матеріали і методи***: Архівні зразки тканин, зібрані впродовж півтора року, отримані у відділі патології ротової порожнини та мікробіології Народної стоматологічної академії м. Бхопал, Індія. Усього проаналізовано 40 зразків одонтогенних кератокіст, радикулярних кіст, дентигеричних кіст та амелобластом (по 10 зразків кожної патології). Проведено імуногістохімічне дослідження експресії та локалізації α-АГМ. ***Результати***: Найбільший вміст МФ було виявлено у зразках одонтогенних кератокіст. Слабка експресія α-АГМ виявлена в 50% випадків, помірна — у 22,5% та інтенсивна — в 10%. За характером розташування МФ розрізняли веретеноподібний, фокальний або сітчастий типи (35; 27,5 та 20% відповідно). ***Висновки***: МФ в одонтогенних кістах та пухлинах є досить гетерогенними за вмістом та характером розташування. Таким чином, строма цих утворень містить МФ, що доведено наявністю клітин, позитивних за α-АГМ.



***Ключові слова***: альфа-актин гладеньких м'язів, одонтогенні кісти та пухлини, імуногістохімія.