ORIGINAL RESEARCH

# Histochemical characterization of extracellular matrix in desmoplastic ameloblastoma and its comparison with other histological variants

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## Abstract

**Background:** Desmoplastic ameloblastoma (DA) is an uncommon and aggressive odontogenic tumor with different clinical, imaging and histological features. It mainly affects the anterior segments of the jaws and appears like benign fibro-osseous lesions on imaging. The present study assessed and compared the extracellular matrix (ECM) of desmoplastic ameloblastoma, histochemically, with the other variants.

**Material and methods:** In total, 41 cases of different histological types of ameloblastoma and 10 controls were included in the present study after obtaining clearance from the institutional ethical clearance committee, and subjected to Orcein staining for elastic fibres and Van Gieson staining for collagen in the ECM. Data were entered in IBM SPSS software. Any value less than or equal to 0.05 was considered statistically significant.

**Results:** Although not statistically significant, the age of the patients with DA was higher than other variants. Out of 41 cases of ameloblastomas, 13 cases were follicular, 12 cases were plexiform, 9 cases were unicystic ameloblastoma, including intra luminal and mural type, and 7 cases were of desmoplastic ameloblastoma. All 10 controls, which included normal oral mucosa tissue and all seven cases of DA, showed the presence of elastic fibres as analyzed by Orcein staining. The stroma was densely collagenous. In contrast, all but one case of other variants showed the absence of elastic fibres.

**Conclusion:** The presence of an abundance of elastic fibres in desmoplastic ameloblastoma, in contrast to the other variants, suggests a difference in the pathogenesis of the disease and points to alternative mechanisms for its unique imaging features and aggressiveness.


**Keywords:** desmoplasia, fibrosis, histochemistry, neoplastic, odontogenic

## Introduction

Ameloblastoma is a benign yet locally aggressive epithelial odontogenic tumor that primarily affects the mandible and may result in severe morbidity. Excluding odontoma, it has been considered the second most common benign odontogenic tumor of the jaws.[1] Ameloblastoma is classified into four major types: solid or multicystic (conventional), unicystic, metastasizing, and extraosseous or peripheral variants.[2,3] Apart from this clinical classification, ameloblastoma shows variegated histological patterns with a unique arrangement of odontogenic epithelium in a loose, collagenous to extremely desmoplastic stroma. The stroma, in fact, dictates the appearance, histological distinction and even outcome of various histological variants. For example, the stroma is loose in plexiform ameloblastoma, collagenous in follicular or unicystic ameloblastoma and extreme desmoplasia with or without osteoplasia is noted in desmoplastic ameloblastoma.[3-5]

The development and progression of any tumor rely on the interaction of tumor cells with the supporting tissue stroma. The tumor microenvironment (TME) is made up of the extracellular matrix (ECM), which includes stromal cells such as fibroblasts, adipose tissue, immune cells, and bone. Like any neoplasm, ameloblastoma develops inside a complex and rich stroma, where tumor cell infiltration and invasion of the surrounding tissues are evident. The ameloblastoma

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**How to cite the article:** Pandiar D. Dharmaraj K, Krishnan R P. Histochemical characterization of extracellular matrix in desmoplastic ameloblastoma and its comparison with other histological variants. Oral Maxillofac Pathol J 2026; 17(1); 28-34.

**Source of Support:** Nil

**Conflict of Interest:** None

cells have been shown to induce osteoclastogenesis through the RANK/RANKL/osteoprotegerin (OPG) pathway and the release of matrix metalloproteinases (MMPs), which results in the resorption of bone.[6] The stroma chiefly contains collagen (I, III, IV) with varying amounts of oxytalan fibres and laminin V.[7-9] While the tumor stroma plays a significant role by acting as a barrier in preventing the host immune response, providing sufficient nutrients to the tumor and by eliminating waste materials, it also has an essential role in maintaining the structural integrity and in preserving the tissue function.[10]

Desmoplastic ameloblastoma (DA) is one of the peculiar and aggressive histologic variants of ameloblastoma, which demonstrates distinctive clinical, radiographic and histopathological features when compared to the other variants.[11,12] It further demonstrates a rare nature to grow to a large size along with invasion into the surrounding structures with wide marrow infiltration beyond the radiographic margins.[10] Previous studies have demonstrated the presence of oxytalan fibres in the stroma of DA as compared to scanty fibres in conventional ameloblastoma.[9] The oxytalan fibres, possibly representing immature or modified elastin, present in the stromal component suggest that the tumor might originate from the epithelial rest of Malassez present in the periodontal ligament (PDL) or its gingival extension.[7,9] In an earlier study, Fisher et al., demonstrated and correlated the presence of oxytalan fibers in ameloblastoma as an indication of an inherent capacity of the cells PDL and gingiva that persists even under neoplastic states.[7] The oxytalan fibres have also been demonstrated in the periapical granuloma and cysts, reparative tissue of periodontal disease, and proliferative periodontal tissues of scleroderma.[13,14] Further, the stroma shows marked desmoplasia similar to other fibrotic states of the body such as oral submucous fibrosis, organ fibrosis and scleroderma, mediated by TGF-β.[15-17] In previous studies on OSMF, we showed the presence of elastic fibers in the stroma, which were noted to become thicker and were seen in superficial and deep connective tissue as the disease progressed, signifying the role of elastic fibers in the stabilization of the disease.[18] We thus, in the purview of other fibrotic conditions, hypothesize that the desmoplastic ameloblastoma contains additional extracellular components which could further be related to the difference in the histopathology. This study aims to evaluate the relative alterations in the stromal components in desmoplastic ameloblastoma as compared to other variants of ameloblastoma using a simple novel orcein staining procedure. To aid in the diagnosis and prognosis of ameloblastoma and its variants, the study intends to analyze the various properties of elastic fibers to assess if there is any significant difference in the ECM of DA as compared to other variants.

## Materials and Methods

The present cross-sectional study was conducted in the Department of Oral Pathology and Microbiology after obtaining clearance from the institutional human ethical committee (IHEC/SDC/OPATH-2204/24/141). Data on histopathologically confirmed cases of ameloblastoma, which underwent excision, were retrieved from the department's archival files and the electronic database of the institution from the years 2021-2024.

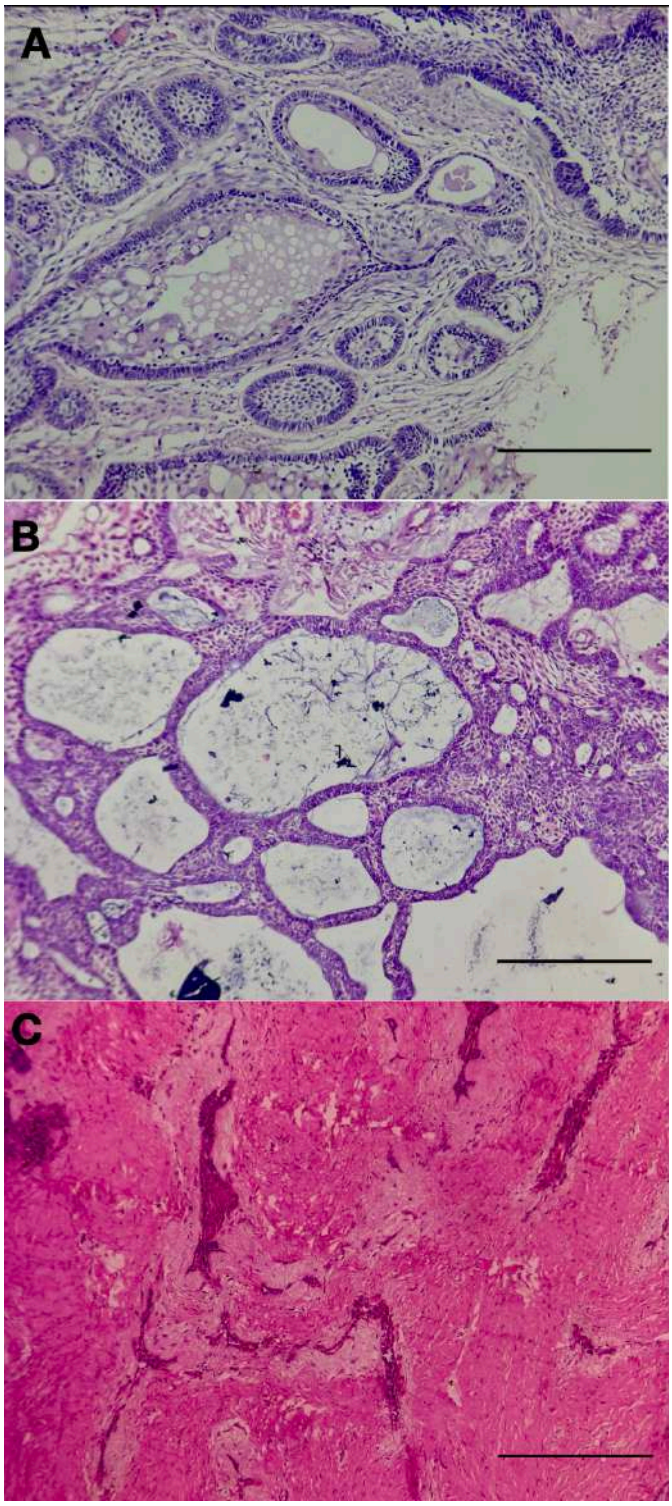


**Figure 1:** Photomicrographs of the H&E stained sections of follicular ameloblastoma demonstrating arrangement of odontogenic epithelium in follicles in a collagenous stroma (a, 10x objective, bar= 50μm), b) plexiform ameloblastoma demonstrating arrangement of odontogenic epithelium in interconnecting plexi proliferating in a loose fibrovascular stroma (10x objective, bar= 50μm) and c) desmoplastic ameloblastoma showing compressed odontogenic epithelium in a densely collagenous stroma, not the myxoid stroma around the odontogenic epithelium (10x objective, bar= 50μm)

Fifty-one cases (41 cases of different histological variants of ameloblastoma and 10 controls) were included in the study. We included seven cases of desmoplastic ameloblastoma, nine cases of unicystic ameloblastoma, including intraluminal and mural variants, and 25 cases of other variants of ameloblastoma. Out of 25 cases, 13 cases were follicular, and the remaining 12 cases were diagnosed as plexiform ameloblastoma histopathologically.

Ten controls from the gingiva of apparently normal individuals were included, who visited for the extraction of the third molars. The cases where the tissue was inadequate for histochemical analysis and cases with incomplete details were not included. In the sections where the staining had faded for re-evaluation, new sections were cut and stained with H&E stain. The sections were examined by two oral pathologists, and the histological diagnosis was made according to the essential diagnostic criteria of the latest World Health Organization classification of head and neck tumors, 2024.[19] All 51 blocks were further prepared for special staining (Van-Gieson and Orcein staining).

### Histochemical procedures

#### Orcein staining

Orcein stain is a dry staining dye that is often used in histopathology to view many histological structures in our study, specifically elastic fibers. The orcein staining was done based on the procedure described previously.[18] Briefly, the working orcein solution comprises 0.5 grams of synthetic orcein dissolved in 50 milliliters of 70% alcohol with the help

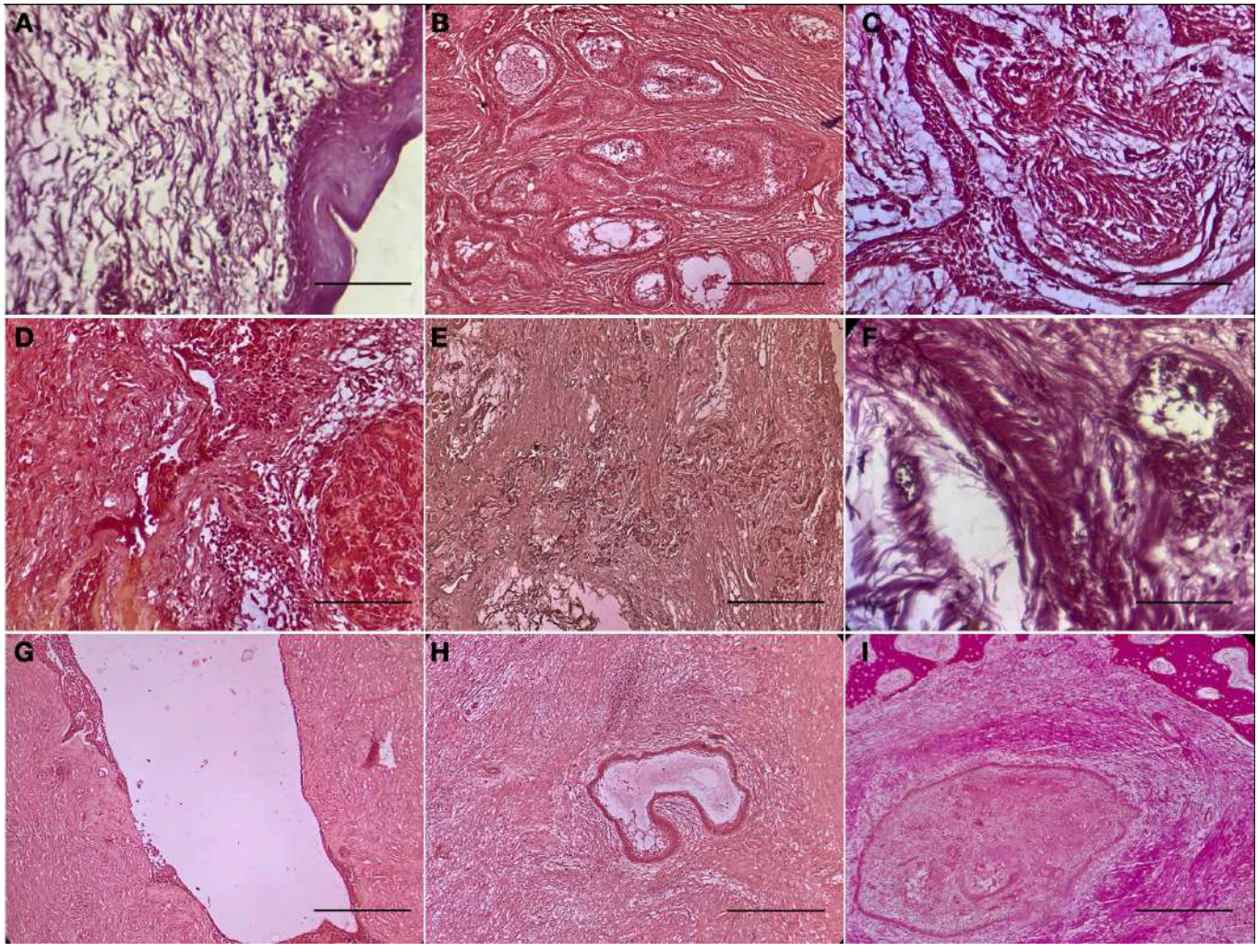


Figure 2: Photomicrographs showing (a) orcein-stained sections of the normal mucosa demonstrating brown-black elastic fibres in the connective tissue (40x); (b) negative staining in follicular ameloblastoma- 4x; (c) only positive case of plexiform ameloblastoma showing fine elastic fibres in the stroma (10x); (d-f) positivity in the stroma of desmoplastic ameloblastoma demonstrating positivity in the connective tissue in haphazard to crisscross patterns with no positivity in perifollicular regions (d-10x, e-4x, f-40x); (g) negative staining of orcein in the luminal (4x) and the mural (h) components of unicystic ameloblastoma-4x; and (i) Van Gieson staining in a case of desmoplastic ameloblastoma showing the distinction between the densities of the tumor-associated fibrotic stroma and less dense stroma unrelated to neoplasm-4x. [4x objective, bar= 2.0 mm; 10x objective, bar= 50μm; 10x objective, bar= 200μm]

of mild heat. 0.5 milliliter of hydrochloric acid is then added and mixed well. For staining, three micrometer thick sections were cut and dewaxed using xylene for 8-10 min. Then, these sections were subjected to various grades of alcohol, submerged in water and stained for 30 minutes at 56–60°C with Orcein solution. The slides were thereafter washed in graded alcohol, counterstained and washed under running water. Finally, the slides were mounted permanently using synthetic resin.

**Van-Gieson Staining**

Similarly, three-micron sections were cut from all 46 blocks on coated slides and incubated overnight at 50 °C. The following steps involved bringing the sections to water and staining in Weigert's hematoxylin for 15 minutes. The sections were then differentiated, washed and stained in Van Gieson stain for 10–12 min (5 ml saturated aqueous picric acid mixed with 1.5 ml of 1% acid fuchsin).[18] Finally, the sections were washed briefly in running water and mounted.

**Scoring and statistical analysis**

Histopathologically, the sections were evaluated semi-qualitatively for the density and arrangement of the fibres, their distribution in the stroma, color and pattern as described previously.[18] The collagen was stained bright pink to red by Van Gieson staining, and the elastic fibres appeared dark brown to black with Orcein staining. The orientation, location and density of the fibres were evaluated by two observers after calibration, and uniform interobserver agreement as assessed by Cohen kappa statistics.[20] A strong inter-observer agreement was noted with a score of 0.833 (p-value- <0.001). The data were tabulated in a Microsoft Excel spreadsheet. The statistical analysis was done on IBM SPSS Statistics, Version 26.0 (Released 2019; IBM Corp., Armonk, New York, United States) software. The Chi-square and ANOVA tests were employed, and any value of ≤0.05 was considered as statistically significant.

## Results

**Clinicodemographic details**

Overall, the mean age of occurrence for all the included ameloblastomas was 38.19±15.96 (in years; Median: 33 years). The number of males observed in the study was twenty-five (61%), and the remaining sixteen were females (39%), with male to female ratio of 1.56:1. Seen individually, desmoplastic ameloblastoma showed the highest mean age of 48.2857 years (±9.32) followed by plexiform (38.25±19.59), follicular (37.69±15.53) and unicystic ameloblastomas (31±14.73). No statistically significant difference was noted between the study groups (p-value: 0.202).

Most cases were seen on the left side (20/41), followed by the right side (12/41) and in the anterior segments (9/41). Out of these nine cases in the anterior segment, 3 cases were histologically diagnosed as follicular ameloblastoma, two as unicystic, one as plexiform and the remaining three were desmoplastic ameloblastoma, with no significant difference between the study groups (p-value: 0.682). Mandible was undisputedly affected more than maxilla for all the cases, but when analyzed individually, no significant difference was noted (p-value: 0.750). The desmoplastic variant showed more of a mixed radio-opaque radiolucent appearance radiographically (4/7), while none of the cases in other variants showed a mixed radiographic appearance. Unicystic ameloblastomas were more

**Table 1:** Comparison of clinicodemographic features between desmoplastic ameloblastoma and other histological variants included in the present study; # not significant, * statistically significant, M/L-Multilocular, U/L- Unilocular

| Parameter | Overall (n-41) | Follicular (n-13) | Plexiform (n-12) | Unicystic (n-9) | Desmoplastic (n-7) | p-value |
|---|---|---|---|---|---|---|
| Age (in years) | 38.19±15.96 | 37.69±15.53 | 38.25±19.59 | 31±14.73 | 48.2857±9.32 | 0.202# |
| Gender | | | | | | |
| Male | 25/41(60.97%) | 6/13 (46.2%) | 9/12 (75%) | 6/9 (66.67%) | 4/7 (57.14%) | 0.502# |
| Female | 16/41 (39.03%) | 7/13 (53.8%) | 3/12 (25%) | 3/9 (33.33%) | 3/7 (42.86%) | |
| Location | | | | | | |
| Mandible | 35/41 (85.36%) | 10/13 (76.9%) | 11/12 (91.67%) | 8/9 (88.89%) | 6/7 (85.72%) | 0.750# |
| Maxilla | 6/41 (14.64%) | 3/13 (23.1%) | 1/12 (8.33%) | 1/9 (11.11%) | 1/7 (14.28%) | |
| Laterality | | | | | | |
| Left | 20/41 (48.78%) | 7/13 (53.8%) | 6/12 (50%) | 4/9 (44.44%) | 3/7 (42.86%) | |
| Right | 12/41 (29.27%) | 3/13 (23.1%) | 5/12 (41.67%) | 3/9 (33.33%) | 1/7 (14.28%) | 0.682# |
| Anterior | 9/41 (91.95%) | 3/13 (23.1%) | 1/12 (8.33%) | 2/9 (22.23%) | 3/7 (42.86%) | |
| Radiological Features | | | | | | |
| M/L | 30/41 (73.17%) | 12/13 (92.3%) | 11/12 (91.67%) | 4/9 (44.44%) | 3/7 (42.86%) | |
| U/L | 7/41 (17.07%) | 1/13 (7.7%) | 1/12 (8.33%) | 5/9 (55.56%) | 0 | 0.000* |
| Mixed | 4/41 (9.76%) | 0 | 0 | 0 | 4/7 (57.14%) | |

unilocular, while the follicular and plexiform variants were almost exclusively multilocular. The difference in radiographic appearance between the groups was statistically significant (p-value: 0.000). The detailed values are shown in Table 1. The current study included 25 cases of ameloblastoma consisting of follicular variant and plexiform variant, 9 cases of Unicystic ameloblastoma including intra luminal and mural type, 7 cases of desmoplastic ameloblastoma and 10 controls which included normal oral mucosa tissue (Figure 1).

**Special staining**

The control group, normal mucosa, showed prominent elastic fibers on orcein staining, which appeared dark brown and loosely arranged.

After orcein staining, the elastic fibers in the stromal component of the desmoplastic variant of ameloblastoma appeared more prominent and intensely stained. These fibres appeared dark brownish, were dense and short fragmented with the haphazard arrangement, and were located throughout the tumor. These fibres were prominently seen around the compressed odontogenic epithelium. Out of the seven desmoplastic cases retrieved, 3 cases showed moderately dense fibers arranged sparsely in the stroma. The other four cases showed a cris-cross arrangement of elastic fibers haphazardly dispersed dense within the stroma. The ECM juxtaposed to the odontogenic epithelium lacked any evidence of elastic fibres. However, the other variants of ameloblastoma showed no staining by orcein dye except for one case of plexiform ameloblastoma (Figure 2). There was a statistically significant difference in the presence of elastic fibres in DA with the other study groups (p-value: 0.000).

Qualitatively, the stroma of desmoplastic ameloblastoma was more collagenous than the other variants, as evidenced by Van Gieson staining (Figure 3).

## Discussion

Ameloblastoma is a benign, locally aggressive tumor most prevalent in the fourth and fifth decades of life, and there is no predilection for gender.[2,4,8,21,22] 18% of the ameloblastomas show an association with impacted teeth. As explained in the classical description by Robinson, ameloblastoma bears a limitless growth potential and may show distant metastasis in rare instances.[2,23] Other rare but worrisome complications include malignant transformation. 1% of the ameloblastomas may show malignisation, and the secondary type of ameloblastic carcinoma is rarer than the de novo variants.[24] Nonetheless, ameloblastomas need management by wide surgical excision, particularly the desmoplastic variant, which can show infiltration beyond the radiographic margins. As aforementioned, the desmoplastic ameloblastoma shows unique histopathological features with intense desmoplasia resulting in squeezing of the odontogenic epithelium in an 'animal-like' configuration. The present study was conducted to analyze the stroma of DA with histochemical comparison with other variants to shed light on the pathogenesis.

According to the literature, DA accounts for between 0.9-12.1% of all ameloblastomas worldwide.[12] The histopathological differential diagnoses included squamous cell carcinoma, odontogenic fibroma, metastatic tumors and squamous odontogenic tumor.[19] When compared to other forms of ameloblastoma, desmoplastic ameloblastoma has more aggressive behavior, which may be attributed to its anatomic location, causing an early invasion of nearby structures and bone. In line with the literature, all but one case of desmoplastic ameloblastoma affected the mandible, and the majority of them were mixed radioopaque-radiolucent on imaging.[25,26] This unique radiographic appearance is the result of bone formation in the sclerotic stroma, a phenomenon referred to as 'osteoplasia'. This appearance usually resembles the radiographic findings of craniofacial benign fibro-osseous lesions. Although not statistically significant, the age of patients with DA was higher than other variants. The cases with unicystic ameloblastoma were the youngest. The insignificant values could be due to the lesser number of cases of DA included in the present study.

Previous studies have analyzed the stroma in ameloblastoma for the type of collagen, components of the basal lamina, and, less commonly the presence of oxytalan fibres. However, the data regarding the presence and role of elastic fibres is meagre. The histochemical analysis of the stroma of different types of ameloblastoma employing a novel and simple method (Orcein staining) in conjunction with Van Gieson staining yielded interesting results. Regarding collagen fibres, the quality and orientation between various subtypes showed similar features, except for the density of collagen fibres. The desmoplastic ameloblastoma showed intense fibrosis. This is in accordance with the previous studies.[8,9] In DA, type VI collagen staining next to tumor islands shows that extracellular matrix proteins are being synthesized, resulting in newly formed connective tissue rather than scar tissue. The odontogenic epithelium in DA can also produce transforming growth factor (TGF)-β, which is a strong local factor for regulating extracellular matrix production, analogous to fibrosis in other sites.[15,16,18,27,28] This shows that TGF-β contributes to the formation of a desmoplastic matrix. Collagen I remains the main component of the fibrous stroma, which is synthesized either by the fibroblasts or the myofibroblasts and contains two COL1A1 and one COL1A2 chain. Interestingly, TGF-β1 has been shown to stimulate the transcription of both these proteins.[29]

So, if all the variants show similar types of collagen, what makes DA more desmoplastic than the other variants? The answer may lie in provenance. The previous demonstration of oxytalan fibres, which are considered to be an immature form of elastic fibres, and the almost exclusive presence of elastic fibres in desmoplastic variants supports an origin from the PDL or its gingival extensions.[30] The presence of elastic fibres randomly, haphazardly and in a crisscross manner in DA could be responsible for the resistance of collagen by matrix-metalloproteinases, alongside the stabilization of fibrosis. The presence, orientation and distribution of elastic fibres in DA and absence in the other subtypes were found to be similar to the characterization of oxytalan fibres as described by Inoue et al.[9] A similar mechanism has been described in oral submucous fibrosis, another progressive fibrotic state of the oral cavity.18,31 The authors found that the presence of elastic fibres positively correlated with the histological stage of the disease. It was further opined that these fibres play a major role in cross-

linking and stabilization of OSMF as the stage advances. Similar results were found in other studies.[18] The presence of elastic fibres has also been negatively correlated with prognosis and severity of disease in pulmonary fibrosis and liver cirrhosis.[32,33] A definitive role of elastic fibres in the pathogenesis of another fibrotic condition, fibroelastosis, is established.[34] Fibroelastosis is a rare idiopathic fibrotic disease that has been demonstrated in association with immune-mediated or infectious diseases or even after chemotherapy. We thus opine here that the presence of elastic fibres in DA is directly correlated with the stabilization of the neoplastic state alongside the presence of mature, dense collagen in DA, analogous to other fibrotic conditions leading to resistance to the degradation of the collagen. Reichart and Philipsen further showed a unique histopathological feature in DA, i.e., the presence of myxoid stroma around the odontogenic epithelium, which might correspond to the perifollicular areas that showed an absence of elastic fibres in the present study.[35] The other variants of ameloblastoma showed a difference in density of collagen and lacked elastic fibres in the extracellular matrix, except for a single case of plexiform ameloblastoma. This difference could be due to a) the complete absence of elastic fibres or the very low density of these fibres in conventional ameloblastoma or b) a difference in the pore size between mature and immature fibers causing varied differential staining.

The study, however, had a limitation of a lesser and unequal number of cases, owing to the rarity of desmoplastic ameloblastoma with only seven cases reported in the past four years. Multi-institutional studies may be performed for further evaluation of the results obtained from the present study.

## Conclusion

The presence of an abundance of elastic fibres in desmoplastic ameloblastoma, in contrast to the other variants, suggests a difference in the pathogenesis of the disease and points to alternative mechanisms for its unique imaging features and aggressiveness. Thus, desmoplastic ameloblastoma appears to be a distinct variant of ameloblastoma; the elastic fibers in the desmoplastic variant could be held responsible for the stabilization of desmoplasia. Studies with larger sample sizes are warranted for confirmation.

**Competing Interests:** The author(s) declared no potential conflicts of interest concerning the research, authorship, and/or publication of this article.

**Author Contributions:** All authors contributed to the literature review and were responsible for drafting and/or critically editing the manuscript.

**Funding:** No funding was received for the completion of this project.

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