BULLETIN OF STOMATOLOGY AND MAXILLOFACIAL SURGERY
Volume 21, Issue 8

DOI: 10.58240/1829006X-2025.21.8-454

REVIEW ARTICALE

COMPARATIVE EVALUATION OF ENDOGLIN EXPRESSION IN AMELOBLASTOMA, ODONTOGENIC KERATOCYST, AND DENTIGEROUS CYST: AN INSTITUTIONAL STUDY WITH A COMPREHENSIVE LITERATURE REVIEW

Nausathkhan Ubayathulla[1], M.R. Muthusekar[2], Pratibha Ramani[3*], Abilasha Ramasubramanian[4], Kavya Dharmaraj [4]

[1]Department of Oral Surgery, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Tamil Nadu, India, Department of Oral and Maxillofacial Surgery, EHS, Fujairah Specialized Dental Center and Hospital, Fujairah, UAE Email id : nausathkhan@gmail.com
[2]Department of Oral Surgery, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Tamil Nadu, India Email id : muthusekhar55@gmail.com
[3]Department of Oral and Maxillofacial Pathology, Saveetha Dental College and Research Institute, Saveetha University, SIMATS,Chennai,TamilNadu,India.hod.omfpsaveetha@gmail.com
[4]Department of Oral and Maxillofacial Pathology, Saveetha Dental College and Research Institute, Saveetha University, SIMATS, Chennai, Tamil Nadu, India.

*** Corresponding author: Pratibha Ramani** Department of Oral and Maxillofacial Pathology, Saveetha Dental College and Research Institute, Saveetha University, SIMATS, Chennai, Tamil Nadu, India. hod.omfpsaveetha@gmail.com

Received: *Aug 9. 2025;* ***Accepted:*** *Aug Sep 8, 2025;* ***Published:*** *Sep 20, 2025*

ABSTRACT

Background: Odontogenic cysts and tumors are common areas of interest for Oral and maxillofacial surgeons and pathologists with their unique characteristics and prevalence. Angiogenesis has recently stimulated greater interest in lesion dynamics as vascularity provides nutrient supply responsible for proliferation activities.

Objectives: In order to comprehend its function in the biological behavior of these lesions, the study seeks to evaluate and compare the expression of the putative angiogenic marker CD105 in dentigerous cysts, odontogenic keratocysts, and ameloblastomas.

Methods: A total of N=36 histopathologically diagnosed cases of OKC, DC and Ameloblastoma were selected with control (Angiomatous Granuloma). Immunohistochemistry was performed using a CD105 marker.

Result: Angiogenesis was found to be increased in ameloblastoma followed by OKC and Dentigerous cyst and the results were statistically significant in comparison with that of the control.

Keywords*:* Angiogenesis, Ameloblastoma, CD105, Cyst, Endoglin

INTRODUCTION

Odontogenic cysts and tumors possess unique characteristics and prevalence of which, Dentigerous cyst and Odontogenic keratocyst (OKC) are common with the prevalence of 13.4% and 14.9% respectively [1, 2, 3]. The most common and invasive odontogenic tumor is Ameloblastoma with the annual global incidence determined to be 0.92 per million/person [4, 5].

Though Dentigerous cyst is an innocuous cyst, it behaves like a niche for transformation into squamous cell carcinoma, mucoepidermoid carcinoma and ameloblastoma thus demanding the detection of influencing factors [6]. With variations in incidence, disease progression, and malignancy potential, these three lesions are more prevalent and more aggressive locally than other odontogenic lesions.

Because vascularity provides the nutrients required for proliferation, angiogenesis recently draws increased interest in the study of lesion dynamics [7]. In oral squamous cell carcinoma, angiogenesis (new blood vessel formation) is linked to tumor recurrence [8]. It has also been extensively researched in a number of tumors, such as colorectal, hepatic, and breast cancers, as well as in a number of oral disorders, including odontogenic lesions, oral lichen planus, and oral squamous cell carcinoma, where it occasionally has a prognostic significance. Using immunohistochemistry with antibodies such as CD31, CD34, Factor VIII, VEGF, and CD105, tumor angiogenesis has been evaluated to estimate the tumor progression and survival [9–13]. This demonstrates the importance of angiogenesis in tumor growth, survival, and prognosis. Finding an accurate and trustworthy prognostic marker for angiogenesis aids in the selection of high-risk patients which further allows for more efficient management strategies.

Numerous markers are available for vascularization, in which CD105 (Endoglin) has been utilized for its own potential to detect angiogenesis in a pathological lesion, reflecting the prognosis [14, 15]. CD105 is a transforming growth factor β (TGF-β) transmembrane co-receptor required for angiogenesis [16], and is highly expressed on the surface of actively proliferating microvascular endothelial cells, forming immature, highly permeable tumor neovessels [17]. CD105 is specific for tumor neovascularization and is not expressed on pre-existing/mature vasculature and on large vessels, unlike the other endothelial markers [18]. Thus, CD105 for angiogenesis could be a promising prognostic marker in odontogenic pathology.

The objectives of the current study were to assess and compare the mean vascular density (MVD) of Ameloblastomas, OKCs, and Dentigerous cysts in a South Indian population using the CD105 marker and to ascertain whether CD105 influences the biological activity of these lesions. To further investigate the function of CD105 in odontogenic diseases, a thorough literature analysis was carried out.

MATERIALS AND METHODS

Ethical approval:

The study was approved by the institutional review board of Saveetha Dental College and Research Institute, Chennai. This was a cross sectional comparative study where patients with surgical resection or biopsy of their primary tumor/cyst at the Saveetha Dental College and Hospitals from 2017 to 2023 were retrospectively reviewed.

Study population:

A total of N = 51 formalin fixed specimens were randomly retrieved, processed and embedded. Pre-diagnosed cases of Dentigerous cyst, Ameloblastoma and OKC were selected and the criteria used are, histopathologically confirmed cases of DC, AM and OKC, primary lesions, and no history of previous

surgery or therapy.

Tissues of peripheral AM, malignant AM, specimens with inadequate tissue, specimens with no clinical details were excluded. As a result, the study included 12 cases of DC (Group 1), 12 cases of OKC (Group 2), 12 cases of AM (Group 3) and 5 cases of PG (control) by random sampling.

Immunohistochemistry analysis:

The cases were then stained with CD105 antibody using the methods of immunohistochemistry. Totally 5 cases of pyogenic granuloma were used as positive control. Formalin-fixed, paraffin-embedded blocks were sectioned into three-millimeter pieces, which were then placed on slides covered in gelatin. The pieces were then rinsed in distilled water after being dehydrated in 100% alcohol for five minutes and deparaffinized in xylene for ten minutes. Using a pH 9.0 Tris-EDTA buffer solution, heat-mediated antigen retrieval was carried out in a pressure cooker for five minutes. Under flowing water, the pressure cooker was depressurized to 37°C. For thirty minutes, endogenous peroxidase activity was inhibited. Primary antibodies against human p53 (Dako, Monoclonal mouse anti-human p53 protein, Denmark) and human cyclin D1 (Dako, Monoclonal mouse anti-human cyclin D1, Denmark) were incubated on the sections for one hour at room temperature. The Polyexcel HRP/DAB detection system (Pathnsitu, conjugated by goat anti-mouse/rabbit IgG) was used for the detection. Then Mayer's hematoxylin counterstaining was done followed by xylene mounting. Every run had both positive and negative controls. Brown cytoplasmic positivity is considered as positive. The presence and frequency of

micro vessels were then assessed by observing the slides under the light microscope.

Mean vascular density (MVD) is a measure of tumor angiogenesis. Various studies have shown that MVD correlates with tumor aggressiveness and poor prognosis [19, 20]. Evaluation of endoglin staining under light microscopy was done using the hot spot method proposed by Weidner et al [21]. MVD was evaluated using an Olympus microscope according to the procedure suggested by Weidner et al. (1993). Sections were screened at a magnification of ×100 (×10 ocular and ×10x objective) and 10 highly vascularized regions (hotspots) were selected. Micro vessels were counted at ×400 magnifications (×10 ocular and ×40x objectives) (field size 0.18 mm^2) in each of the 10 fields. The mean density of the micro vessels was recorded in Microsoft Excel. Slides were simultaneously evaluated by two operators using a double-headed microscope, and both had to agree on each of the individual micro vessels before being included in the count (Figure 1,2,3).

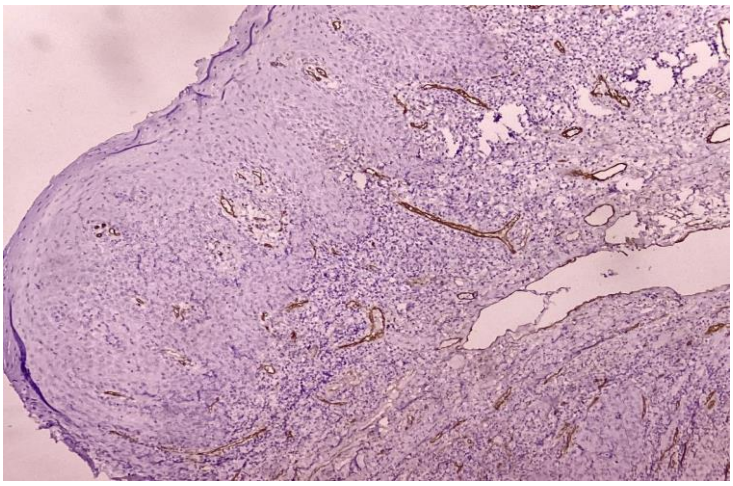

Figure 1. Microvessels in the connective tissue wall of dentigerous cyst

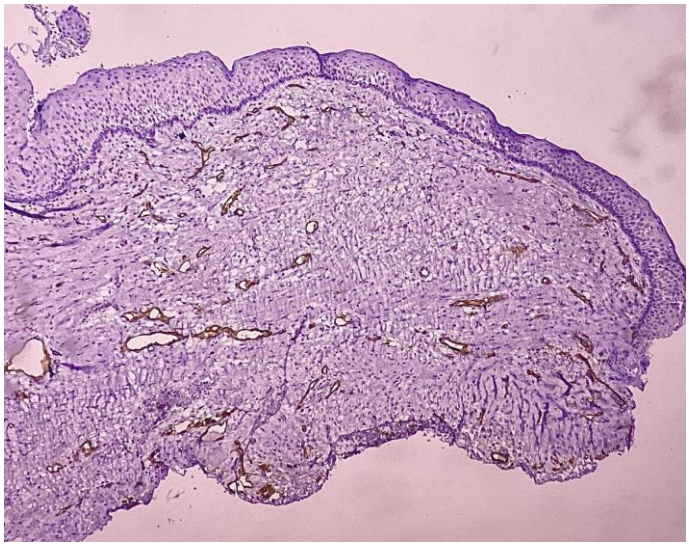

Figure 2. Microvessels beneath the epithelium in odontogenic keratocyst expressed by CD105 antibody

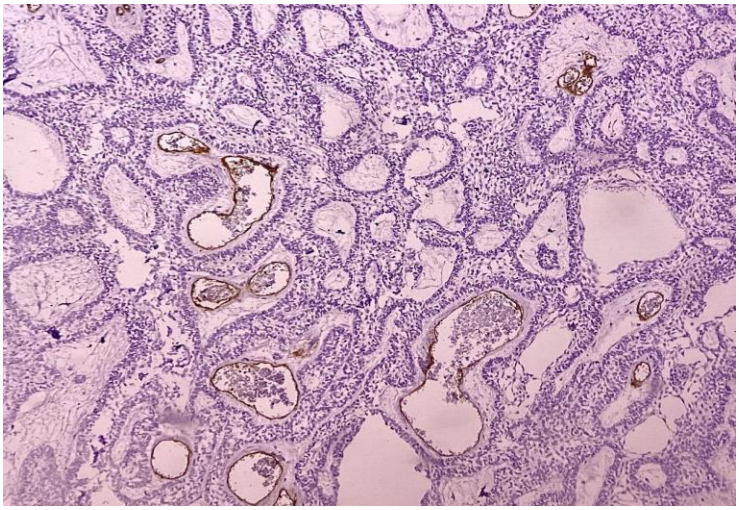

Figure 3. CD105 positive microvessels adjacent to the epithelial islands in ameloblastoma

STATISTICAL ANALYSIS

Statistical analysis was performed using ANOVA to compare the outcomes among the groups. Post Hoc multiple comparison test was employed to compare between individual groups. $p < 0.05$ was considered as statistically significant. The statistical package for social sciences (SPSS 14) software was used for computations.

RESULTS

In the present study, the mean MVD of AM was high (14.17 +- 2.552) when compared with that of OKC (13.00+-3.593) and DC (11.50 +- 5.419). However statistically significant results were obtained with all the three lesions in comparison with that of the control (Table 1) (Figure 4, 5).

Table 1 Statistical Comparison of the Mean Vascular Density (MVD) between the 4 groups (AM, OKC, DC and PG).

Multiple Comparisons					
Dependent Variable: Mean Vascular Density					
	(I) Groups	(J) Groups	Mean Difference (I-J)	Std. Error	Sig.
LSD	Dentigerous Cyst	Ameloblastoma	-2.667	4.538	0.56
		Odontogenic Keratocyst	-1.5	4.538	0.743
		Pyogenic Granuloma	-23.083*	4.538	0

	Ameloblastoma	Dentigerous Cyst	2.667	4.538	0.56
		Odontogenic Keratocyst	1.167	4.538	0.798
		Pyogenic Granuloma	-20.417*	4.538	0
	Odontogenic Keratocyst	Dentigerous Cyst	1.5	4.538	0.743
		Ameloblastoma	-1.167	4.538	0.798
		Pyogenic Granuloma	-21.583*	4.538	0
	Pyogenic Granuloma	Dentigerous Cyst	23.083*	4.538	0
		Ameloblastoma	20.417*	4.538	0
		Odontogenic Keratocyst	21.583*	4.538	0
Bonferroni	Dentigerous Cyst	Ameloblastoma	-2.667	4.538	1
		Odontogenic Keratocyst	-1.5	4.538	1
		Pyogenic Granuloma	-23.083*	4.538	0
	Ameloblastoma	Dentigerous Cyst	2.667	4.538	1
		Odontogenic Keratocyst	1.167	4.538	1
		Pyogenic Granuloma	-20.417*	4.538	0
	Odontogenic Keratocyst	Dentigerous Cyst	1.5	4.538	1
		Ameloblastoma	-1.167	4.538	1
		Pyogenic Granuloma	-21.583*	4.538	0
	Pyogenic Granuloma	Dentigerous Cyst	23.083*	4.538	0
		Ameloblastoma	20.417*	4.538	0
		Odontogenic Keratocyst	21.583*	4.538	0

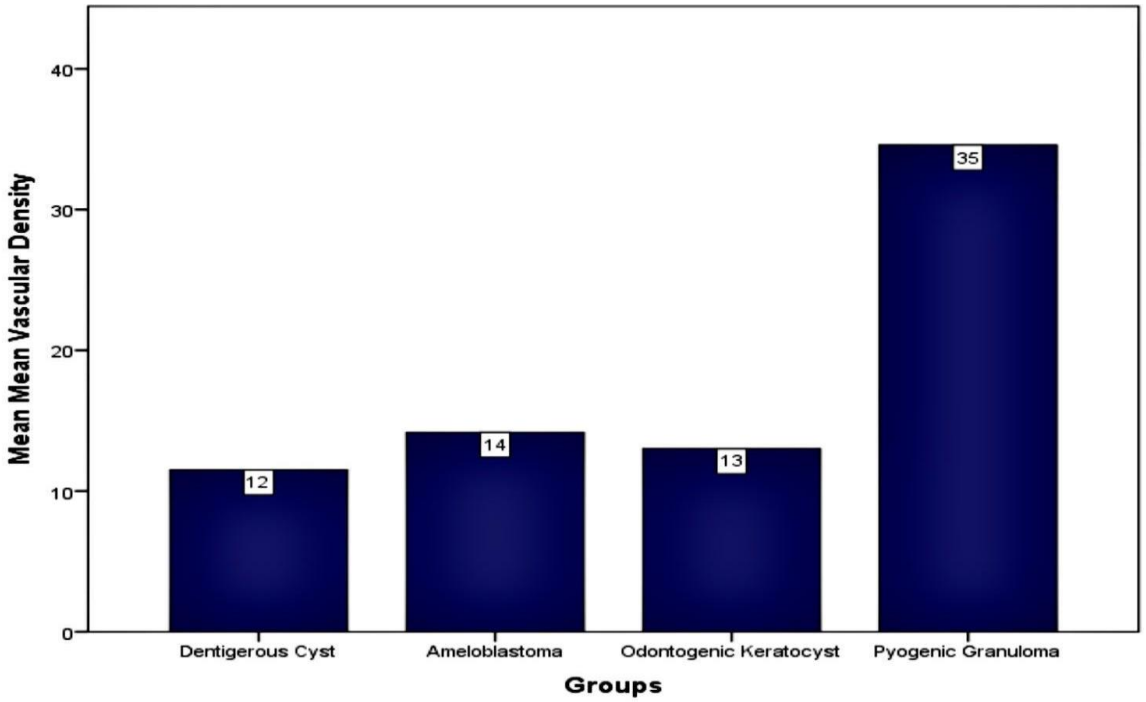

Figure 4. Mean Vascular Density (MVD) of AM, OKC and DC comparison with PG

Table 2. Summary of notable findings from literature analysis on CD105 in odontogenic pathologies

Author, Year	Location	Odontogenic pathology assessed (Sample size)	IHC marker used	Results of CD105	Findings
Hande et al, 2011 [9]	Maharashtra	AM (20), UAM (15), Normal (10)	CD105	MVD: AM = 35 ± 12.93 UA = 31.13 ± 10.54 N = 15.80 ± 5.67	No significant difference in MVD between AM and UA reflecting the fact that though clinical behaviour, histopathological presentation and prognosis of AM and UA differ, the process of angiogenesis is not different
Santos et al, 2011 [22]	Brazil	OKC (20), DC(20), RC (20)	NF-B, MMP, CD105	MVC of RCs = 16.9, range 2.0-44.0, DCs = 12.1, range 0.0-48.0 OKCs (10.0, range 1.7-39.0	MMP-9 and NF-B high expression confirmed the more aggressive biologic behavior of OKCs, and the differences in the biologic behavior of the lesions studied were not associated with the angiogenic index.

Lima et al, 2011 [23]	Brazil	RC (24), PG (24)	CD105, CD34, Tryptase	CD105-positive vessels in 50% of RCs, 70.8% of PGs showed areas of close association with MCs.	Highest concentration of tryptase-positive MCs showed association with CD34-positive vessels rather than CD105-positive vessels, although there was no difference in angiogenesis and MC density between RC and PG, and it suggests that MCs have a role in vascular compartment maintenance.
Gadbail et al, 2011 [24]	Maharashtra	OKC (38), DC (27)	CD105, Ki67	MVD: OKC = 51.67 ± 14.81, DC = 5.99 ± 6.59, Normal = 15.25 ± 13.67	CD105 was strongly expressed in micro vessels of OKC compared to that of Dentigerous cyst and normal oral mucosa, suggesting that angiogenesis might be associated with locally aggressive biological behavior of OKC.
Jamshidi et al, 2014[25]	Iran	OKC (10), AM (30)	CD105, CD34	MVD: AM = 14.47 ± 3.8, OKC = 9.6 ± 2.9	The MVD was significantly higher in ameloblastoma than odontogenic keratocyst, which suggests that angiogenesis is one of the potential mechanisms involved in the more aggressive biologic behavior of ameloblastoma compared to OKC.
Kumar et al, 2014 [26]	Maharashtra	AM (20), DC (20), OKC (20)	CD105	MVD: AM = 7.98 ± 2.70, OKC = 6.25 ± 2.88, DC = 3.75 ± 1.42	AM and OKC demonstrated a higher mean value of MVD than DC, suggesting that angiogenesis possibly contributes to different biological behaviors of OKC, DCs and solid AMs
Chandrangsu et al, 2016 [27]	Thailand	OKC (39)	p53, p63, p73, CD105	MVD: 26.7 ± 15.8	Three members of the p53 protein family were expressed in OKCs, and their expression relates to angiogenesis in these tumors
Galvánet	Mexico	Odontogenic myxoma (18), dental follicles	CD105	MVD: OM =	The odontogenic myxoma smaller than 3 cm showed a

al, 2016 [28]		(18)		0.29 ± 0.32, DF = 1.27 ± 1.06	greater MVD than those larger than 3 cm in size, and it was lower in large OMs than dental follicles, suggesting that the vascular proliferation has a limited role in the growth mechanisms and the aggressive behavior of this neoplasm.
Lotfy et al, 2020 [29]	Egypt	RC (15), DC (15), OKC (15)	CD 105,	MVD: RC = 18.10 ± 8.38, DCs = 12.38 ± 5.42, OKC = 17.88 ± 7.11.	Increase in angiogenesis of OKC confirms that aggressive lesions require a larger number of blood vessels, and highlights the concept of unique clinical behavior of this cyst.
Ali et al, 2020 [30]	Pakistan	AM (16), OKC (16), CGCL (16), PG (16)	CD105	CGCL = 32.99 ± 0.77, OKC = 7.21 ± 0.75 AM = 8.07 ± 0.36 PG = 14.7 ± 0.96	CGCL was the most aggressive, with highest MVD among the investigated odontogenic lesions
Ibrahim et al, 2022 [31]	Egypt	AM (15), Ameloblastic fibroma (15)	AgNORs, CD105	AM = 27.52 ± 7.85 Ameloblastic fibroma = 18.2 ± 3.57	Higher MVD reflects a higher proliferative activity and a more locally aggressive biologic behavior of ameloblastoma when compared to ameloblastic fibroma
Anjali et al, 2022 [32]	Mumbai	OKC (25), DC (25), PG (10)	CD105	OKC = 13.25 ± 2.94, DC = 12.87 ± 2.4, PG = 13.02 ± 1.07	There was no statistically significant difference in the mean MVD values of OKC, DC and PG.

AM: Ameloblastoma, UA: Unicystic ameloblastoma, CD105: Cluster differentiation 105, MVD: Mean vascular density, OKC: Odontogenic Keratocyst, DC: Dentigerous cyst, RC: Radicular cyst, MMP-9: Matrix metalloproteinase-9, NF-B: MVC: Micro Vessel Count, PG: Pyogenic granuloma, CD34: Cluster differentiation 34, MC: Mast cells, OM: Odontogenic myxoma, CGCL: Central giant cell lesions.

DISCUSSION

Newly formed blood vessels were precisely evidenced by the expression of CD105 (Endoglin), a key indicator of angiogenesis used in this study. The mean vascular density in ameloblastoma was found to be higher, averaging 14.17 ± 2.552 than in OKC and DC which had mean values of 13.00 ± 3.593 and 11.5 ± 5.419 respectively. According to the study's findings, ameloblastoma's high MVD is indicative of its greater neovascularization, which is linked to its more aggressive behavior, while cysts show less aggression.

According to a study by Sefi et al., there was a substantial statistical significance (p-value of less than 0.001) and a significant difference in the MVD between OKC and DC [33], and MVD in OKC was significantly lower than that in ameloblastoma. These results are consistent with the current study's findings, which show that AM has a greater MVD than both OKC and DC. The consistent results across the studies highlight the AM's aggressive nature, which is demonstrated by its increased vascularization. The main conclusions of a survey of the literature on CD105 in odontogenic diseases are compiled in Table 2.The elevated MVD seen in AM is probably caused by the overexpression of angiogenic factors like CD105. As oxygen and nutrients are required to sustain the rapid growth of the tumor the metabolic needs are supported by the improved blood vessel formation in AM [26]. Furthermore, the increased expression of other factors, such as matrix metalloproteinases (MMP-2 and MMP-9), transforming growth factor-beta (TGF-β), fibronectin, tenascin, and stromal myofibroblasts (MF), which are known to promote neovascularization and metabolic activity in the connective tissue, may also contribute to this increase in MVD. By improving vascular supply of tumor, the aforementioned molecules support vital processes like signaling cascades, extracellular matrix remodeling, and cell adhesion. Hence, in comparison to OKC and DC, AM had a greater MVD [22]. Additionally, this increased vascularization has been found to be influenced by anastomosed micro vessels, which are more frequently seen in AM cases [30]. The growth and metabolic needs of the tumor are supported by these interconnected networks of blood vessels, which enhance blood flow and nutrition supply. The results of the current study are in line with earlier research by Jamshidi et al., where angiogenesis was observed to be higher in ameloblastoma than in OKC and implied that angiogenesis is crucial for both tumor growth and cellular metastasis. It could also be used to forecast patient survival, metastasis, and tumor progression [34-36].

In contrast to AM, OKC and DC had lower MVD, which suggests less neovascularization and less aggressive behavior. Despite the fact that both DC and OKC are cystic lesions, the higher MVD in OKC than DC might be due to its higher MMP expression. Browne et al. were among the first to propose that the connective tissue wall of OKC plays a substantial role in the pathophysiology of the lesion [29, 37]. MMPs are enzymes that promote angiogenesis and tissue remodeling by breaking down different extracellular matrix components. The inducement of new blood vessel formation and a rise in MVD are directly associated with the increased expression of MMPs in OKC [32]. Hence, the clinically aggressive behavior of OKC has been linked to increased MMP expression, which could be the reason for its greater invasiveness and chance of recurrence in comparison to DC.

Furthermore, compared to DC and normal oral mucosa (NOM), CD105-positive juvenile micro vessels in OKC have slightly aberrant shape, with convoluted and dilated lumens. A high Ki-67 labeling index may imply a quick fluid transfer to maintain the OKC epithelium, which is highly proliferative [24]. According to a study by Kouhsoltani et al., radicular cysts had a greater MVD than OKC, which was linked to a high level of inflammatory infiltration in these lesions [38]. Additionally, inflammatory cells might encourage angiogenic activity in these cysts, according to Graziani et al., Nonaka et al., and Tete et alB[39–41]. However, in OKC, high blood vessel concentrations are observed in both locations with low inflammation as well as substantial inflammation. The higher tissue metabolism in OKC, which suggests a more aggressive biological behavior, may be the cause of increased angiogenesis in OKC relative to DC [29]. Therefore, our data supports the classification of OKC as aggressive lesion, as evidenced by CD105 expression and angiogenesis, regardless of whether they are categorized as cysts or tumors.

Compared to other groups, PG has a higher mean MVD (CD105 - 34.58 ± 21.108, VEGF - 19.83 ± 2.78); however, this does not always signify that the lesion is aggressive. The different pathogenic mechanisms that encourage angiogenesis can be the cause of this [26]. In addition to angiogenesis, the underlying mechanisms that cause it also affect how aggressive the lesion is [42].The drawbacks of the current investigation include a limited sample size and the fact that it is a retrospective analysis, which restricts how broadly the findings can be applied to this particular demographic [43]. Multicenter randomized

controlled trials (RCTs) with standardized methodology should be carried out to evaluate CD105 in both benign and malignant odontogenic lesions in order to obtain a thorough understanding of the function of angiogenesis in the biological behavior of odontogenic lesions [44, 45].

Hence, more aggressive growth and a higher chance of recurrence are associated with enhanced neovascularization in odontogenic lesions [46]. Higher vascular density in lesions indicate a worse prognosis and require more intensive therapy with close observation. Anti-angiogenic treatments might also be useful in treating certain cases of aggression. In general, the prognosis and therapeutic approach for these pathologies are greatly influenced by neovascularization. The findings of the study, which used the CD105 marker to compare the mean vascular density of ameloblastomas, OKCs, and dentigerous cysts, indicate that when evaluating the prognosis of lesions based on angiogenesis, factors like the underlying process, precise classification, lesion variants, and the presence of inflammatory reactions should be taken into account. Therefore, employing CD105 to assess the lesion aggressiveness through angiogenesis helps clinicians in grouping patients into high- and low-risk categories. This might ultimately influence the treatment planning and patient outcome.

CONCLUSION

According to the study's findings, there was a statistically significant difference between the study groups and the control group when comparing the mean vascular density (MVD) of DC, OKCs, and AM using the CD105 marker. Ameloblastoma had a higher mean vascular density than DC and OKC. Despite being statistically insignificant, the differences between the study groups showed a good association with the biological behavior of odontogenic lesions. For additional research, more thorough investigations with bigger sample sizes and more precise criteria are required.

DECLARATIONS

Funding
This research received no external funding or financial support.

Conflict of Interest

The authors declare no conflict of interest.

Ethical Approval

This study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Institutional Medical Ethics Committee.

Acknowledgments
none

REFERENCES

1. Savithri V, Suresh R, Janardhanan M, Aravind T, Mohan M. Prevalence of odontogenic cysts and its associated factors in South Indian population. J Oral Maxillofac Pathol. 2020;24(3):585. doi: 10.4103/jomfp.JOMFP_171_20.
2. Vivekbalamithran V, Ramalingam K, Ramani P, et al. Odontogenic keratocyst with moderate epithelial dysplasia: a rare entity. Cureus. 2024;16(3):e56702. doi:10.7759/cureus.56702.
3. Pandiar D, Ramani P, Krishnan RP, Thamilselvan S, Ramya R. Dysplastic epithelial changes in odontogenic keratocyst: a rare histological presentation with immunohistochemical cognizance. OralOncol.2021;122:105580. doi:10.1016/j.oraloncology.2021.105580.
4. Sudarsan R, Abilasha R. Prevalence of odontogenic tumours in association with age and gender: an institutional study. J Popul Ther Clin Pharmacol. 2023;29(4):222–31. doi:10.47750/jptcp.2022.1004.
5. Hendra FN, Van Cann EM, Helder MN, et al. Global incidence and profile of ameloblastoma: a systematic review and meta-analysis.Oral Dis.2020;26(1):12–21.
6. Aldelaimi AAK, Enezei HH, Berum HER, et al. Management of a dentigerous cyst: a ten-year clinicopathological study. BMC Oral Health. 2024;24:831. doi:10.1186/s12903-024-04607-w.
7. Amin T. Aggressiveness of odontogenic keratocyst. J Health Allied Sci. 2022:11–22. doi:10.1055/s-0042-1758036.
8. Kademani D, Lewis JT, Lamb DH, Rallis DJ, Harrington JR. Angiogenesis and CD34 expression as a predictor of recurrence in oral squamous cell carcinoma. J Oral Maxillofac Surg. 2009;67:1800–5.
9. Hande AH, Gadbail AR, Sonone AM, Chaudhary MS, Wadhwan V, Nikam A. Comparative analysis of tumour angiogenesis in solid multicystic and unicystic ameloblastoma by using CD 105 (endoglin). Arch OralBiol.2011;56(12):1635-40. doi:10.1016/j.archoralbio.2011.06.007.

10. Li C, Guo B, Wilson PB, Stewart A, Bryne G, Bundred N, et al. Plasma levels of soluble CD105 correlate with metastasis in patients with breast cancer. Int J Cancer. 2000;89(2):122–6.

11. Saad RS, Liu YL, Nathan G, Celebrezze J, Medich D, Silverman JF. Endoglin (CD105) and vascular endothelial growth factor as prognostic markers in colorectal cancer. Mod Pathol. 2004;17(2):197–203.

12. Ho JW, Poon RT, Sun CK, Xue WC, Fan ST. Clinicopathological and prognostic implications of endoglin (CD105) expression in hepatocellular carcinoma and its adjacent non-tumorous liver. World J Gastroenterol. 2005;11(2):176–81.

13. Tao X, Huang Y, Li R, Qing R, Ma L, Rhodus NL, et al. Assessment of local angiogenesis and vascular endothelial growth factor in patients with atrophic erosive and reticular lichen planus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.2007;103:661–9.

14. Derakhshan S, Mahdavi N, Kardouni Khoozestani N, et al. Assessment of the association of OCT3/4 with GLUT1 and CD105 in oral squamous cell carcinoma using dual immunohistochemistry. BMC Oral Health.2022;22:300.doi:10.1186/s12903-022-02332-w.

15. Di Paolo V, Russo I, Boldrini R, et al. Evaluation of endoglin (CD105) expression in pediatric rhabdomyosarcoma.BMCCancer.2018;18:31. doi:10.1186/s12885-017-3947-4.

16. Li DY, Sorensen LK, Brooke BS, Urness LD, Davis EC, Taylor DG, et al. Defective angiogenesis in mice lacking endoglin. Science. 1999;284(5419):1534–7.

17. Seon BK, Haba A, Matsuno F, Takahashi N, Tsujie M, She X, et al. Endoglin-targeted cancer therapy. Curr Drug Deliv. 2011;8(1):135–43.

18. Behrem S, Zarkovic K, Eskinja N, Jonjic N. Endoglin is a better marker than CD31 in evaluation of angiogenesis in glioblastoma. Croat Med J. 2005;46(3):417–22. 2010;10:2367–84.

19. Shenoi R, Devrukhkar V, Chaudhuri, Sharma BK, Sapre SB, Chikhale A. Demographic and clinical profile of oral squamous cell carcinoma patients: a retrospective study. Indian J Cancer. 2012;49:21–6.

20. Mahapatra N, Uma Rao KD, Ranganathan K, Joshua E, Thavarajah R. Study of expression of endoglin (CD105) in oral squamous cell carcinoma. J Oral Maxillofac Pathol. 2021;25(3):552. doi:10.4103/jomfp.jomfp_13_21.

21. Weidner N, Semple JP, Welch WR, Folkman J. Tumour angiogenesis and metastasis: correlation in invasive breast carcinoma. N Engl J Med. 1991;324:1–8.

22. de Andrade Santos PP, de Aquino AR, Oliveira Barreto A, de Almeida Freitas R, Galvão HC, de Souza LB. Immunohistochemical expression of nuclear factor κB, matrix metalloproteinase 9, and endoglin (CD105) in odontogenic keratocysts, dentigerous cysts, and radicular cysts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;112(4):476–83. doi:10.1016/j.tripleo.2011.04.022.

23. Lima SC, Rizo VH, Silva-Sousa YT, Almeida LY, Almeida OP, León JE. Immunohistochemical evaluation of angiogenesis and tryptase-positive mast cell infiltration in periapical lesions. J Endod. 2011;37(12):1642–6. doi:10.1016/j.joen.2011.08.024.

24. Gadbail AR, Hande A, Chaudhary M, Nikam A, Gawande M, Patil S, Tekade S, Gondivkar S. Tumor angiogenesis in keratocystic odontogenic tumor assessed by using CD-105 antigen. J Oral Pathol Med. 2011;40(3):263–9. doi:10.1111/j.1600-0714.2010.00962.x.

25. Jamshidi S, Zargaran M, Baghaei F, Shojaei S, Mahmoodabadi R, Dehghan A, Moghimbeigi A. An immunohistochemical survey to evaluate the expression of CD105 and CD34 in ameloblastoma and odontogenic keratocyst. J Dent (Shiraz). 2014;15:192–8.

26. Kumar DV, Hemavathy S, Kulkarni D, Rudraiah PM, Sidramayya Mathpati SK, Priya S. Expression of CD105 in tumor angiogenesis: a comparative study (ameloblastoma, keratocystic odontogenic tumor and dentigerous cyst). J Int Oral Health. 2015;7(6):23–7. PMID: 26124595

27. Chandrangsu S, Sappa-yatosok K. p53, p63, and p73 expression and angiogenesis in keratocystic odontogenic tumors. J Clin Exp Dent. 2016;8(3):e319–24. doi:10.4317/jced.52843.

28. del Carmen González-Galván M, Aguirre-Urizar JM, Bologna-Molina R, et al. Assessment of CD-105 as an angiogenic modulator in odontogenic myxomas and dental follicles. Int J Surg Pathol. 2016;24(4):315–9. doi:10.1177/1066896916632588.

29. [Accessed 2024 Oct 16]. Available from: https://mjd.journals.ekb.eg/article_200152_cb32d8ea2f8671a47c9e19653d218d4a.pdf.

30. Ali K, Zeb Khan S, Sultana N, Alghamdi O, Muhammad S, Mokeem SA, Ali S, Abduljabbar T, Vohra F. Assessment of tumor angiogenesis by expression of CD 105 in ameloblastoma, odontogenic keratocyst, and central giant cell lesion. Asian Pac J Cancer Prev. 2020;21(11):3373–9. doi:10.31557/APJCP.2020.21.11.3373.

31. Ibrahim A, Alqalshy E, Abdel-Hafiz AA-S, El-Rahman KA, Alazzazi M. Roles of proliferation and angiogenesis in locally aggressive biologic behavior of ameloblastoma versus ameloblastic fibroma. Diagnostics. 2022;12:392.

32. Anjali AK, Yadav S, Kumar S, Chande M, Jadhav A, Pereira T. Comparison of CD105 (endoglin) expression in odontogenic keratocyst and dentigerous cyst: an immunohistochemistry study. Oral Maxillofac Pathol J. 2022;13(1):32–5.

33. Seifi S, Shafaie S, Ghadiri S. Microvessel density in follicular cysts, keratocystic odontogenic tumors, and ameloblastomas. Asian Pac J Cancer Prev. 2011;12(2):351–6.

34. Siriwardena BS, Tennakoon TM, Tilakaratne WM. Relative frequency of odontogenic tumors in Sri Lanka: analysis of 1677 cases. Pathol Res Pract. 2012;208:225–30.

35. Peltola J, Magnusson B, Happonen RP, Borrman H. Odontogenic myxoma: a radiographic study of 21 tumors. Br J Oral Maxillofac Surg. 1994;32:298–302.

36. Bologna-Molina R, Damián-Matsumura P, Molina-Frechero N. An easy cell counting method for immunohistochemistry that does not use an image analysis program. Histopathology. 2011;59:801–3.

37. Browne RM. The pathogenesis of odontogenic cysts: a review. J Oral Pathol. 1975;4:31–6.

38. Kouhsoltani M, Moradzadeh Khiavi M, Jamali G, Farnia S. Immunohistochemical assessment of mast cells and small blood vessels in dentigerous cyst, odontogenic keratocyst, and periapical cyst. Adv Pharm Bull. 2015;5:637–41.

39. Graziani F, Vano M, Viacava P, Itro A, Tartaro G, Gabriele M, et al. Microvessel density and vascular endothelial growth factor (VEGF) expression in human radicular cysts. Am J Dent. 2006;19:11–4.

40. Nonaka CFW, Maia AP, do Nascimento GJF, Freitas RA, Souza LB, Galvão HC, et al. Immunoexpression of vascular endothelial growth factor in periapical granulomas, radicular cysts, and residual radicular cysts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;106:896–902.

41. Tete S, Mastrangelo F, Grimaldi S, Costanzo G, Salini L, Speranza L, et al. Immunohistochemical evaluation of CD31 in human cystic radicular lesions and in keratocysts. Int J Immunopathol Pharmacol. 2005;18:39–45.

42. Pandiar D, Anbumani P, Krishnan RP. Literature Review, Case Presentation and Management of Non-ossifying Fibroma of Right Angle of Mandible: More Than just a Cortical Defect! Indian J Otolaryngol Head Neck Surg. 2024 Feb;76(1):1054-1061. doi: 10.1007/s12070-023-04110-8. Epub 2023 Aug 7. PMID: 38440574; PMCID: PMC10908682.

43. Krishnan, Reshma Poothakulath MDS; Pandiar, Deepak MDS, FHNP, PhD; Sagar, Sandra MDS. Immunohistochemical Expression of CK14 and Bcl-2 in Odontogenic Keratocyst and Its Variants. Applied Immunohistochemistry & Molecular Morphology 32(3):p 151-156, March 2024. | DOI: 10.1097/PAI.0000000000001182

44. Ardila CM, Yadalam PK. Interpretative Nuances in Risk Prediction of Ameloblastoma Recurrence. Head Neck Pathol. 2025 Apr 8;19(1):42. doi: 10.1007/s12105-025-01777-z. PMID: 40198452; PMCID: PMC11978568.

45. Surana KA, Pandiar D, Krishnan RP. Immunohistochemical Expression of MDM2, Bcl-2, SATB2 and Ki-67 in Histological Variants of Unicystic Ameloblastoma. Head Neck Pathol. 2024 Oct 15;18(1):100. doi: 10.1007/s12105-024-01705-7. PMID: 39404986; PMCID: PMC11480311.

46. Ardila CM, Yadalam PK. Reevaluating Histopathologic and Molecular Insights in Ameloblastoma Management: A Call for Methodological Refinement. Head Neck Pathol. 2025 ;19(1):23. doi: 10.1007/s12105-025-01764-4.