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Systematic Review

Immunoexpression of P53 in ameloblastomas: a systematic review

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Abstract – Objective: The objective of this systematic review is to evaluate the available evidence on P53 immunoexpression in ameloblastomas and to investigate its potential association with tumor behavior in order to clarify its diagnostic significance and prognostic implications. **Methods:** Following PRISMA guidelines, research in PubMed, Web of Science, Scopus, and ScienceDirect databases yielded 17 studies meeting the inclusion criteria. **Results:** Results indicate that P53 overexpression is frequent in ameloblastomas, particularly in solid and multicystic types, and correlates with increased tumor aggressiveness and recurrence. A relationship was observed between P53 expression and other key molecular markers, including Bcl-2 and Ki-67, emphasizing its role in disrupted apoptotic pathways. **Conclusions:** These findings support the potential of P53 as a prognostic marker and suggest further research into targeted therapies for better management of ameloblastomas.

Introduction

Ameloblastoma (AMB) is a benign odontogenic tumor that accounts for approximately 10% of all craniofacial tumors. Although histologically benign, it is characterized by persistent growth, local invasiveness, and a high risk of recurrence, with recurrence rates reaching up to 70%, and in some cases, they may transform into malignant forms [1,2]. The tumor's clinical behavior is influenced not only by its histological subtype but also by underlying molecular alterations [3,4].

Among the molecular pathways involved in ameloblastoma pathogenesis, the *P53* tumor suppressor gene has emerged as a key focus of interest due to its critical role in cell cycle regulation, DNA repair, and apoptosis. Overexpression of P53 protein has been reported in various odontogenic tumors, including ameloblastomas, and appears to be associated with more aggressive histological subtypes. [5,6]

Several narrative reviews and original studies have addressed P53 expression in ameloblastomas, sometimes in relation to other markers such as Ki-67, Bcl-2, or MDM2 [3,6–8]. However, these studies remain limited by small sample sizes, inconsistent methodologies, and a lack of standardized synthesis. To our knowledge, no systematic review has yet applied PRISMA methodology to provide a structured analysis of P53 expression across different histological variants of ameloblastomas.

The objective of this systematic review is to evaluate the available evidence on P53 immunoexpression in ameloblastomas and to investigate its potential association with tumor behavior in order to clarify its diagnostic significance and prognostic implications.

Materials and methods

This systematic review was performed in accordance with the PRISMA 2020 guidelines (preferred reporting items for systematic reviews and meta-analyses) and is registered in the PROSPERO database under the registration number CRD42022355941.

Focused question

We intended to answer the following focused question: Is there an overexpression of P53 in patients with ameloblastoma?

According to the study hypothesis, the PECOS criteria were defined as follows:

Population:
- Healthy individuals without systemic diseases.

Exposure:
- Patients diagnosed with ameloblastomas.

Comparison:
- Ameloblastomas (AME) of varied histological typology,
- Dentigerous cyst (DC),

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- Odontogenic keratocyst (OKC),
- Adenomatoid odontogenic tumors (AOT),
- Ameloblastic carcinoma (AMECA).

Outcomes:
- Expression of p53 protein,

Setting:
- Immunohistochemical (IHC) studies,
- Polymerase chain reaction (PCR).

Study selection criteria

Inclusion criteria

Studies were considered eligible for inclusion if they met all of the following criteria:
- Original research articles focusing on the expression of p53 in odontogenic tumors, particularly ameloblastomas.
- Studies conducted on human tissue samples, with data obtained through IHC or PCR.
- Case reports and case series providing original IHC or PCR data on p53 expression.
- Articles published in English.
- Studies published between 2010 and 2024.
- Studies involving patients without systemic diseases or comorbidities that could influence gene expression.

Exclusion criteria

Studies were excluded if they met one or more of the following criteria:
- Articles focusing primarily on the treatment (surgical, radiotherapeutic, or medical) of ameloblastomas, without p53 expression data.
- Studies conducted on animal models or *in vitro* cell cultures.
- Review articles, letters to the editor, or conference abstracts without original data.
- Descriptive case reports without molecular analysis.
- Articles published in languages other than English.
- Studies involving patients with systemic conditions (*e.g.*, autoimmune diseases, cancer syndromes) potentially affecting molecular markers.
- Studies not reporting quantitative or qualitative data on p53 expression using IHC or PCR methods.

Search strategy

The initial search was performed online by two independent reviewers (Y.A. and S.A.) from the start of the study up to November 2024. Duplicate records were then removed. Preliminary research was conducted in databases including PubMed, Scopus, Web of Science, ScienceDirect, and the Cochrane Library to identify relevant systematic reviews and articles. These searches helped establish terms and synonyms related to the primary concepts of interest (P53 and amelobastoma) and to evaluate and develop the most optimal search strategy. In addition to database searches, manual search was also conducted.

The following strategy was used: (ameloblastoma or ameloblastomas) and (p53 antigen or P53 tumor suppressor protein or cellular tumor antigen p53 or oncoprotein p53).

Study selection

The initial selection of records from the first hit was conducted by independently reviewing their titles. In the second round, abstracts of the remaining records were reviewed, followed by full-text assessments in the third round. All records underwent independent evaluations by the same reviewers to determine their eligibility. Any disagreements between the two reviewers during the abstract and full-text screening were resolved through consultation with a third reviewer (S.C.). Studies that did not satisfy the inclusion criteria were excluded from the analysis.

Data collection process

For each study included in the final review, relevant data were independently extracted by three reviewers (Y.A., S.A., N.A.). Any discrepancies among the three reviewers during the data extraction process were resolved through discussion with a fourth reviewer (S.C.). The extracted information included the first author's name and the year of publication, methods used (immunohistochemical technique or PCR technique), types of lesions, number of samples, and reported outcomes. All extracted data were organized in tables using Office software by two reviewers (Y.A., G.E.). The data extraction forms, along with the findings from each included study, were collectively reviewed to ensure the accuracy and reliability of the process.

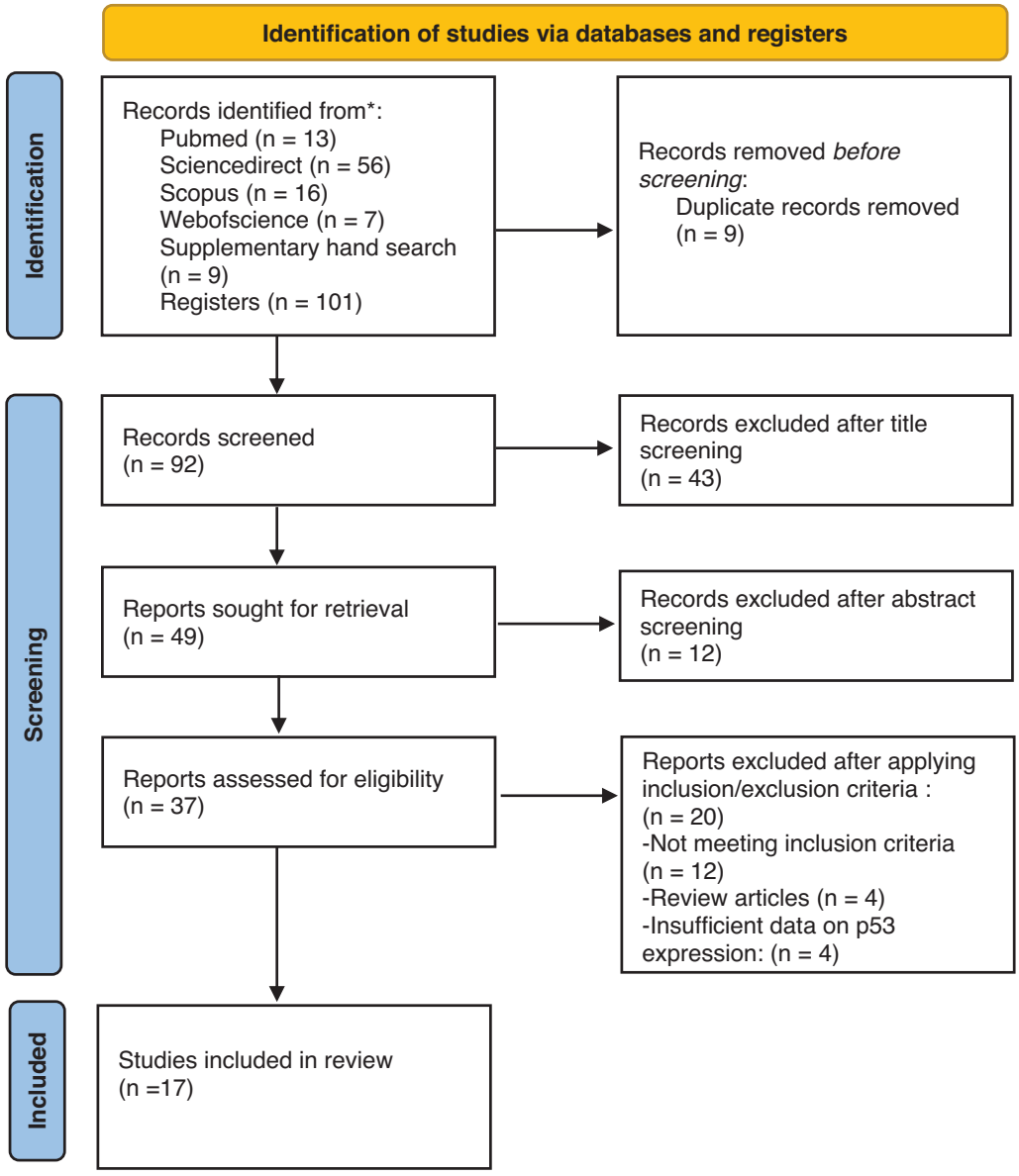
Quality assessment of the included articles

A risk of bias assessment for the included studies was performed in accordance with the guidelines outlined in the Cochrane Handbook for Systematic Reviews of Interventions (2008), Chapter 8: Assessing Risk of Bias in Included Studies [9].

An adapted checklist based on the Cochrane framework was used to evaluate the methodological quality of each study. The tool comprised five key domains:
- Clearly defined study objective,
- Adequate description of inclusion criteria,
- Clearly defined protocol,
- Adequate statistical analysis,
- Clearly described main outcomes.

Each domain was rated as follows:
- Yes: Low risk of bias
- No: High risk of bias
- Uncertain: Insufficient information or ambiguity regarding potential bias

This adaptation ensured methodological consistency across the included observational and descriptive studies.



*Consider, if feasible to do so, reporting the number of records identified from each database or register searched (rather than the total number across all databases/registers).

**If automation tools were used, indicate how many records were excluded by a human and how many were excluded by automation tools.

Fig. 1. Flow diagram.

Results

Literature search

Figure 1 presents a PRISMA flow diagram illustrating the study selection process based on the specified inclusion criteria. An extensive search across the selected databases yielded 101 references for potential inclusion in this systematic review. After removing 9 duplicate records, 92 unique articles remained for screening. Following title and abstract screening, 55 articles were excluded for not meeting the inclusion criteria. The remaining 37 full-text articles were assessed for eligibility. Among these, 20 were excluded after thorough examination due to reasons such as non-human samples, lack of P53 analysis, or being review articles. Consequently, 17 studies were included in the final systematic review.

Main outcomes

The key findings from the included studies are summarized in Table I.

Across the 17 included studies, the majority (approximately 76%, 13 out of 17) reported moderate to high levels of P53 expression in ameloblastomas, suggesting a potential involvement in tumor pathogenesis. However, four studies

Table I. Features and main outcomes of the included studies.

Author's	Geographic origin	Method	Type of lesions	No. of samples	Outcomes
Nakarin Kitkumthon *et al.* (2010) [6]	Thailand	PCR	Group 1 : AMB SMA UA Group 2 : Healthy controls	78 5325 94	The *P53* Arg allele increases susceptibility to AMB, suggesting its importance in tumor etiology.
Noorieh Sharifisistani *et al.* (2011) [7]	Iran	IHC	AMB follicular plexiform UA KOT	39 15 15 9 15	p53 was expressed in 77.8% of AMB and 100% of KOTs, while MDM2 was detected in 74.8% and 80% of cases, respectively. No statistically significant difference in p53 expression was observed among AMB subtypes or between AMB and KOTs ($P > 0.05$). A positive correlation between p53 and MDM2 expression was reported.
Amol Ramchandra Gadbail *et al.* (2011) [8]	India	IHC	AMB SMA UA KCOT DC NOM FOM	23 14 9 32 30 12 10	No significant difference between UA and SMA ($P = 0.388$). p53 significantly higher in AMB compared with KCOT, DC, NOM, and FOM ($P < 0.001$).Positive correlation between Ki-67 and p53 ($P < 0.001$).
Olusegun Michael Adesina *et al.* (2022) [10]	Nigeria	IHC	AMB AOT OKC	69 23 23	p53 immunoreactivity was significantly higher in AMB than in AOT and OKC ($P < 0.05$). The highest expression was observed in plexiform ameloblastoma, indicating increased proliferative activity
Zulfin Shaikh *et al.* (2015) [11]	India	IHC	AMB Follicular Plexiform Unicystic AOT	25 12 4 9 25	p53 expression was observed in both AMB and AOT with no statistically significant difference between the two tumors ($P = 0.554$). The average percentage of p53-positive cells was 75.97% in AMB and 69.87% in AOT. In contrast, survivin expression was significantly higher in AMB than in AOT ($P = 0.002$), suggesting differences in apoptotic regulation and tumor behavior.
Adriano Mota Loyola *et al.* (2016) [12]	Brasil	IHC	AMECA + atypical AMB	17	Strong p53 nuclear expression was observed in AMECA and was associated with a high Ki-67 proliferative index, supporting the role of p53 alterations in malignant transformation.
Zhu You *et al.* (2019) [13]	China	IHC	AM-BC	6	p53 expression was positive in all AM-BC cases and significantly higher than in other ameloblastoma variants ($P < 0.05$), suggesting increased proliferative activity.
Jefferson-da Rocha Tenório *et al.* (2018) [14]	Brasil	IHC	AMB OKC AOT	20 20 20	p53 expression was detected in all lesions, predominantly with low expression in AMB. No significant difference was observed between AMB, OKC, and AOT ($P = 0.108$). Non-significant positive correlation with Bcl-2 ($r = 0.200$) and negative correlation with Bax ($r = -0.100$).
Abhishek Singh *et al.* (2020) [15]	India	IHC	SMA OKC UA	20 20 20	p53 expression was detected in all AMBs. No significant difference was observed between UA and SMA ($P = 0.388$), while expression was significantly higher in AMBs compared with other odontogenic lesions ($P < 0.001$). Positive correlation with PCNA was reported.

Table I. (continued).

Author's	Geographic origin	Method	Type of lesions	No. of samples	Outcomes
Jahanshah Salehinejad *et al.* (2011) [16]	Iran	IHC	AMB Plexiform Follicular Acanthom-tous AOT	30 15 12 3 12	No statistically significant difference in p53 expression among ameloblastoma subtypes ($P = 0.589$). A significant difference in p53 expression was observed between ameloblastomas and AOTs ($P < 0.001$). The intensity of p53 staining was significantly higher in ameloblastomas. A positive correlation between PCNA and p53 expression was also reported.
Takeshi Beppu *et al.* (2015) [17]	Japan	IHC	AMECA secondary type	1	An unusual case of AMB progressing to AMECA was documented, with increased Ki-67 and p53 staining, alongside changes in cytokeratin expression.
Hisashi Kato *et al.* (2012) [18]	Japan	IHC	PA AMB	1 4	Immunohistochemical analysis showed absence of p53 expression in both peripheral and intraosseous AMBs, while p63 was positively expressed in all samples. The Ki-67 labeling index was low (2.22% in PA and 1.37% in intraosseous AMBs), indicating low proliferative activity and supporting the benign biological behavior of these lesions.
Alma Florescu *et al.* (2012) [19]	Romania	IHC	AMB	17	p53 expression was detected in 52.9% (9/17) of AMBs, with weak to moderate nuclear staining predominantly in peripheral columnar cells. The positivity index exceeded 50% in peripheral cells but remained below 10% in stellate reticulum cells. A significant difference in marker reactivity between epithelial compartments was observed ($P < 0.001$), while no association with histological subtype was found ($P > 0.05$).
Juan-Carlos de Vicente *et al.* (2010) [20]	Spain	IHC	OKC DCs 10 RCs AMB	11 10 10 10	P53 immunoexpression was detected in 64% of OKCs, 40% of DCs and RCs, and 30% of AMBs. Immunolabelling for P53 was scattered and only occasionally clustered in AMB.
Daniela Adorno-Farias *et al.* (2018) [21]	Brasil	IHC	AA	8	Weak to moderate nuclear p53 expression was detected in AA, associated with high Ki-67 proliferative activity, suggesting aggressive tumor behavior.
Thasvir Singh *et al.* (2016) [22]	India	IHC	SMA UA	33 6	UA showed significantly higher p53 expression than SMA ($P = 0.03$), with many cases demonstrating strong positivity (>50% tumor cells).
Adriano Mota Loyota *et al.* (2015) [23]	Brasil	IHC	AA	5	Immunohistochemical analysis demonstrated weak to moderate nuclear p53 staining in all cases, with additional weak cytoplasmic expression in three cases. The tumors also showed a high proliferative activity with a mean Ki-67 index of 72.4 ± 24.9 positive cells per high-power field, supporting the aggressive biological behavior and high recurrence potential of AA.

AMB: ameloblastoma, SMA: solid ameloblastoma, UA: unicystic ameloblastoma, AM-BC: ameloblastomas with basal cell features, AA: adenoid ameloblastoma, PA: peripheral ameloblastoma, DC: dentigerous cyst, OKC: odontogenic keratocyst, AOT: adenomatoid odontogenic tumors, AMECA: ameloblastic carcinoma, RC: radicular cyst, NOM: normal oral mucosa, FOM: fetal oral mucosa.

Table II. Expression patterns of P53 and associated markers in histological subtypes of ameloblastoma.

Study	Histological variant	P53 expression rate	Co-expressed markers	Potential prognostic implications
Noorieh Sharifisistani *et al.* (2011) [7]	Follicular/Plexiform AMB	Higher in follicular variant	Ki-67	More aggressive behavior in follicular variant
Amol Ramchandra Gadbail *et al.* (2011) [8]	SMA	High	Bcl-2, Bax	Aggressiveness, recurrence
Zulfin Shaikh *et al.* (2015) [11]	AA	Variable, mostly peripheral staining	Ki-67	Associated with proliferative activity
Adriano Mota Loyola *et al.* (2016) [12]	AMECA	Very high	Ki-67, MDM2	Malignancy
Zhu You *et al.* (2019) [13]	AM-BC	High	Ki-67	Aggressiveness
Jefferson-da Rocha Tenório *et al.* (2018) [14]	Various AMB subtypes (follicular, plexiform, unicystic)	Moderate	Bcl-2, Bax	Anti-apoptotic profile due to Bcl-2/Bax imbalance
Abhishek Singh *et al.* (2020) [15]	SMA/OKC	High in SMA, Higher in OKC	MDM2	Progression potential

reported low, absent, or statistically non-significant expression [14,18,19,21], indicating heterogeneity in the reported findings.

Regarding histological subtypes, most studies (approximately 88%, 15 out of 17) reported moderate to high levels of P53 expression in solid/multicystic ameloblastomas [7,8], whereas lower expression was more commonly observed in unicystic variants [10]. However, one study [22] presented an opposite finding, reporting higher P53 positivity in unicystic ameloblastomas compared with solid/multicystic types.

Several studies demonstrated that P53 expression was frequently higher in follicular subtypes compared with plexiform or unicystic forms [7,11].

In more aggressive variants such as basal cell ameloblastomas and ameloblastic carcinomas, very high P53 expression was reported [12,13], often accompanied by elevated levels of Ki-67 and MDM2, indicating increased proliferative potential and malignant transformation risk.

Studies that explored co-expression with apoptotic markers (Bcl-2, Bax) reported an imbalance favoring cell survival in aggressive variants [8,14], suggesting a mechanistic role for P53 in resistance to apoptosis.

Additionally, Singh *et al.* [15] showed comparative overexpression of P53 in odontogenic keratocysts and solid ameloblastomas, reinforcing its involvement in tumor progression. Salehinejad *et al.* [16] also supported the proliferative role of P53 by demonstrating its co-expression with PCNA in ameloblastomas, further linking P53 to tumor growth dynamics.

Meanwhile, Kitkumthorn *et al.* [6] investigated a *P53* codon 72 polymorphism and its potential association with ameloblastoma susceptibility, offering additional genetic insights into tumor pathogenesis.

Moreover, Beppu *et al.* [17] described a rare and rapidly evolving case of ameloblastoma characterized by markedly increased P53 expression and accelerated cell proliferation, suggesting that P53 dysregulation may contribute to early tumor dedifferentiation and invasiveness in select cases.

Although not all studies reported exact quantitative percentages, the majority identified moderate to strong nuclear P53 staining in 50%–80% of tumor cells, particularly in peripheral epithelial layers.

To provide a clearer comparative overview, Table II summarizes the reported patterns of p53 expression across different histological variants of ameloblastoma, their associated molecular markers, and the potential prognostic implications described in the included studies. This synthesis facilitates the interpretation of the heterogeneity of p53 expression and its possible relevance to tumor behavior.

Quality assessment

The quality assessment of the included studies was conducted based on the criteria outlined in the Cochrane Handbook for systematic reviews of interventions [9].

One study [17] was identified as having a higher risk of bias, with a "No" judgment for adequate statistical analysis and an "Uncertain" judgment for the description of main results. Three studies [15,17,18] received "Uncertain" or "No" responses regarding statistical analysis, indicating a potential moderate risk of bias in this domain.

All other studies answered "Yes" to the questions regarding a clearly defined study objective, adequate inclusion criteria, and a clearly defined protocol, reflecting a generally low risk of bias in these aspects. The question "Was an appropriate statistical analysis used?" was answered affirmatively by 13 out of the 17 studies, showing consistency in the use of robust

Table III. Quality of the included studies.

Author/publication date	Clearly defined study objective	Adequate description of inclusion criteria	Clearly defined protocol	Adequate statistical analysis	Clearly described main outcomes
Nakarin Kitkumthorn *et al.* (2010) [6]	Yes	Yes	Yes	Yes	Yes
Noorieh Sharifi-Sistani *et al.* (2011) [7]	Yes	Yes	Yes	Yes	Yes
Amol Ramchandra Gadbail *et al.* (2012) [8]	Yes	Yes	Yes	Yes	Yes
Olusegun Michael Adesina *et al.* (2022) [10]	Yes	Yes	Yes	Yes	Yes
Zulfin Shaikh *et al.* (2015) [11]	Yes	Yes	Yes	Yes	Yes
Adriano Mota Loyola *et al.* (2016) [12]	Yes	Yes	Yes	Yes	Yes
Zhu You *et al.* (2019) [13]	Yes	Yes	Yes	Yes	Yes
Jefferson-da Rocha Tenório *et al.* (2018) [14]	Yes	Yes	Yes	Uncertain	Yes
Abhishek Singh *et al.* (2020) [15]	Yes	Yes	Yes	Yes	Yes
Jahanshah Salehinejad *et al.* (2011) [16]	Yes	Yes	Yes	Yes	Yes
Takeshi Beppu *et al.* (2015) [17]	Yes	Yes	Yes	No	Uncertain
Hisashi Kato *et al.* (2012) [18]	Yes	Yes	Yes	No	Yes
Alma Florescu *et al.* (2012) [19]	Yes	Yes	Yes	Yes	Yes
Juan-Carlos de Vicente *et al.* (2010) [20]	Yes	Yes	Yes	Yes	Yes
Daniela Adorno-Farias *et al.* (2018) [21]	Yes	Yes	Yes	Yes	Yes
Thasvir Singh *et al.* (2016) [22]	Yes	Yes	Yes	Yes	Yes
Adriano Mota Loyola *et al.* (2015) [23]	Yes	Yes	Yes	Yes	Yes

statistical methods across most studies. Regarding the description of main results, two studies [17,18] provided unclear or "Uncertain" responses, suggesting limitations in how outcomes were reported.

Overall, among the 17 included studies, four presented potential weaknesses related to statistical analysis and reporting clarity, while the remaining 13 were judged to have a low risk of bias overall.

The risk of bias assessment for the studies is summarized in Table III.

Discussion

This review highlights the relevance of P53 immunoexpression as a potential prognostic marker in ameloblastomas. Majority of studies demonstrated that higher P53 levels were observed in more aggressive histological subtypes, such as Solid/Multicystic ameloblastomas (SMA) and Ameloblastic Carcinomas (AMECA). For instance, Singh *et al.* [15] reported the highest P53 expression in SMA, while Loyola *et al.* [12] found very strong nuclear staining in malignant variants, suggesting a role in tumor transformation and poor clinical behavior.

However, it is important to note that one study [22] reported higher p53 positivity in unicystic ameloblastomas compared with solid/multicystic variants, which contrasts with the general trend. Despite this, approximately 88% of the included studies (15 out of 17) demonstrated higher P53 expression in more aggressive histological subtypes such as SMA and AMECA. This discrepancy may be attributed to methodological variations among studies, including differences in antibody clones, sample sizes, and scoring criteria, which could influence staining intensity and interpretation.

Beyond its association with histological subtype, P53 appears to play a central role in the molecular pathogenesis of ameloblastomas. Co-expression with other markers such as Ki-67, Bcl-2, Bax, and MDM2 suggests that dysregulation of the P53 pathway contributes to increased cellular proliferation, apoptotic resistance, and tumor invasiveness [8,13,14]. Florescu *et al.* [20] similarly reported high expression of P53, Bcl-2, and Ki-67 in follicular solid ameloblastomas, reinforcing its involvement in aggressive biological behavior.

While most included studies did not provide direct outcome data such as recurrence rates or survival, indirect prognostic associations were frequently noted. High P53 expression was consistently found in variants known for their higher recurrence risk, such as follicular and solid ameloblastomas [7,12,19]. These findings highlight the potential use of P53 as a surrogate marker of aggressive clinical behavior, especially in scenarios where histological evaluation may not fully reflect the tumor's biologic potential.

In addition, comparative studies such as that by de Vicente *et al.* [20] highlighted significantly stronger P53 immunoreactivity in odontogenic keratocysts and ameloblastomas versus less aggressive odontogenic lesions, underscoring the marker's relevance for tumor stratification and differential diagnosis.

From a clinical standpoint, the routine immunohistochemical evaluation of P53 expression in ameloblastomas could significantly aid in stratifying patients according to their risk profile. High levels of P53 expression, particularly in solid/multicystic or follicular variants, may indicate a greater likelihood of aggressive behavior, recurrence, or even malignant transformation. In such cases, clinicians might consider performing wider surgical excisions to ensure complete tumor removal and minimize the risk of local relapse. Furthermore, these patients may benefit from a more rigorous postoperative surveillance program, including more frequent clinical evaluations and imaging studies, to detect early signs of recurrence.

In addition, understanding the P53 expression profile could support the selection of patients for future adjuvant or targeted therapies, particularly as molecular-based treatments continue to develop. While not yet standard in clinical practice, such molecular insights could eventually complement histological assessment and improve personalized treatment planning. Thus, integrating P53 assessment into routine diagnostic workflows may enhance prognostic accuracy and help tailor management strategies for improved patient outcomes.

Nevertheless, this review also highlights current limitations in literature. Inconsistent methodologies, heterogeneous antibody clones, small sample sizes, and variable scoring systems limit the ability to generalize results. There is a clear need for larger, multicenter studies with standardized immunohistochemical protocols and outcome tracking to fully validate the prognostic significance of P53 in ameloblastomas.

Conclusion

This systematic review confirms that there is a consistent overexpression of P53 in ameloblastomas, particularly in solid/multicystic variants, compared with other odontogenic lesions. This overexpression correlates with the aggressive biological behavior and higher recurrence potential of these tumors. Therefore, P53 can be considered both a useful prognostic biomarker and a potential therapeutic target. Further standardized studies are needed to validate its clinical utility and explore targeted molecular therapies.

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Conflicts of interest

The authors declare no conflicts of interest.

Data availability statement

The data supporting the findings of this study are available within the article. No additional datasets were generated or analyzed during the current study.

Author contribution statement

*Conceptualization and study design : Y Azzouz, S Chbicheb;
*Data collection and analysis : Y Azzouz, S Abidi;
*Manuscript drafting : Y Azzouz ;
*Critical revision of the manuscript : All authors.

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