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Cementoblastoma: An Updated Review of Clinicopathological Features, Molecular Pathogenesis, and Clinical Management

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Abstract

Cementoblastoma (CB) is a rare benign odontogenic mesenchymal neoplasm defined by the deposition of cementum-like mineralized matrix in true continuity with the root of an affected tooth. We conducted a narrative review of the literature indexed in the China National Knowledge Infrastructure (CNKI), supplemented by PubMed/PMC and CNKI Scholar, and verified the bibliographic details of every source ultimately cited. This review synthesizes current evidence on the epidemiology, clinical and imaging features, histopathology and differential diagnosis, molecular pathogenesis, treatment and prognosis of the disease. CB predominantly affects patients in their second and third decades, with an approximately equal sex distribution, and 70%–90% of lesions arise in the mandibular molar–premolar region, most often in association with the first permanent molar. A well-circumscribed radiopaque mass fused to the root and surrounded by a thin, uniform radiolucent rim is virtually pathognomonic; cone-beam computed tomography (CBCT) adds clinically decisive information on cortical perforation, root resorption and involvement of the inferior alveolar canal, and increasingly supports preoperative risk stratification. Histologically, CB is indistinguishable from osteoblastoma, and true attachment to the root remains the only reliable discriminator. The most substantive advance of the past decade has been in molecular pathology: recurrent FOS/FOSB rearrangements were identified in osteoid osteoma and osteoblastoma in 2018, FOS rearrangement and c-FOS overexpression were demonstrated in CB in 2021, and the fifth edition of the WHO Classification of Head and Neck Tumours incorporated this update, reopening the question of whether CB belongs within the osteoid osteoma–osteoblastoma spectrum. A novel FOS::FN1::FOS fusion in a jaw lesion was reported in 2025. Standard treatment is complete excision of the tumor together with extraction of the involved tooth and thorough curettage or peripheral ostectomy. The recurrence estimate has been revised downward from 37.1% to 11.8%; bone expansion and cortical perforation are the best-supported risk factors, and long-term imaging follow-up is essential because recurrence may appear five to seven years after surgery. The Chinese-language literature consists largely of case reports and radiographic reviews, with no published molecular validation or multicenter cohorts. Priorities for the next phase include standardized tissue procurement and molecular testing, imaging-based risk stratification, an integrated diagnostic pathway, and prospective long-term follow-up registries.

Keywords

Cementoblastoma; odontogenic tumors; FOS rearrangement; c-FOS; cone-beam computed tomography; differential diagnosis; recurrence.

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1. INTRODUCTION

Cementoblastoma (CB), historically also termed benign cementoblastoma or "true cementoma", is a rare benign odontogenic neoplasm characterized by the formation of cementum-like mineralized matrix in direct continuity with the root of an affected tooth. Dewey is usually credited with the first report, published in 1927 under the label "osteoma of a molar" [1]. A rigorous historical reappraisal by Ide et al. in 2023, however, traced the earliest complete morphological description to 1888 and the first radiographic documentation to 1906, and reconstructed the somewhat convoluted process by which cementoblastoma came to be accepted as a distinct pathological entity [2]. The 2005 WHO classification assigned CB to the group of odontogenic mesenchymal and/or ectomesenchymal tumors, with or without odontogenic epithelium; the fourth (2017) and fifth (2022) editions retained its designation as a benign mesenchymal odontogenic tumor [4]. The fifth edition introduced, for the first time, explicit "essential" and "desirable" diagnostic criteria for each entity, and in doing so placed unequivocal emphasis on true fusion between the tumor and the tooth root as the indispensable diagnostic requirement [4].

Despite this apparently secure nosological position, the past decade has unsettled several long-held assumptions. Molecular evidence now links the lesion to the osteoid osteoma–osteoblastoma family; pooled analyses have revised the recurrence rate substantially downward; and high-resolution three-dimensional imaging has shifted radiology from simple lesion characterization toward preoperative risk assessment [7,8,17,26,27]. These developments have not yet been integrated into a single account, and the English-language literature contains almost no discussion of the sizable Chinese-language case experience. This narrative review therefore synthesizes verified evidence on the epidemiology, clinical and imaging presentation, histopathology, differential diagnosis, molecular pathogenesis, treatment and prognosis of CB, and identifies the questions that remain open.

1.1. Literature search strategy

The search was completed on 11 November 2026. For Chinese-language sources, CNKI was queried with cementoblastoma as a topic term, which returned 175 records; restricting the same term to the title field returned 14 records, comprising 13 journal articles and one master's thesis. English-language and other foreign-language sources were identified through PubMed/PMC and CNKI Scholar using "cementoblastoma". Eligible sources were systematic reviews and large pooled analyses, clinicopathological or radiographic case series containing at least ten cases, original molecular pathology studies, authoritative reviews of the WHO classification, and case reports published since 2021 that contributed new information. Records with unverifiable bibliographic data, duplicate publications, and studies whose true subject was the biology of normal cementoblasts were excluded. Thirty-four references with verified bibliographic details were ultimately included; the Chinese-language contributions appeared chiefly in the West China Journal of Stomatology, Stomatology, the Journal of Dental Prevention and Treatment, the Journal of Oral Science Research, and the Journal of Clinical Stomatology. This is a narrative review rather than a systematic review: no meta-analysis was attempted and completeness of ascertainment was not formally assessed.

2. EPIDEMIOLOGY AND CLINICAL PRESENTATION

CB accounts for approximately 1.68%–2.6% of all odontogenic tumors [3,5,6], although individual series have reported figures ranging from under 1% to 6.2% [9]. Two large epidemiological surveys of odontogenic tumors in Chinese populations — 759 and 1,309 cases, respectively — provide useful local context for interpreting this rarity [5,6].

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Brannon et al. reported 44 new cases in 2002 and reviewed 74 previously published ones: age at diagnosis ranged from 8 to 44 years (mean 20.7 years), with 30 male and 14 female patients; 70.4% of lesions were mandibular, and the mandibular first molar was the most frequently affected tooth. More than 90% presented as a well-defined radiopaque or mixed-density mass fused to the root and surrounded by a radiolucent band [7]. The updated analysis by Chrcanovic and Gomez, which pooled 258 cases from 141 publications, confirmed a near 1:1 sex ratio, a peak incidence in the second and third decades, and a strong predilection for the posterior mandible, particularly the first molar. In descending order of frequency, associated findings were bone expansion (74.9%), the presence of symptoms (70.2%), a vital pulp in the affected tooth (78%), root resorption (59.8%), inferior displacement of the mandibular canal (23.6%), and cortical perforation (16.3%) [8].

Chinese series are broadly consistent with these figures but show a wider age range. Xu et al. analyzed 14 cases with a median age of 21.0 years and a mean of 27.6 years, an equal sex distribution, mandibular involvement in 12 cases (85.7%), and a premolar–molar location in 13 (92.9%) [3]. Fang et al. reported 12 cases aged 13–58 years (mean 33.8 years), with mandibular lesions in 66.7% and posterior lesions in 91.7%; jaw swelling (75%) and pain (25%) were the most common presenting complaints [10]. In a 20-case series from two Shandong University hospitals, Qi documented ages of 7–69 years (mean 36.70 ± 15.22 years), with 60% of patients aged 20–40 years, mandibular involvement in 90%, and presentation within one year of onset in 75% [11].

Early lesions are frequently asymptomatic and are discovered incidentally on routine radiographs. As the tumor enlarges, patients develop localized jaw expansion, pain, tooth mobility or displacement, a gingival sinus tract, and facial asymmetry. The affected tooth is usually intact, with a vital pulp but possible tenderness to percussion — a valuable clue distinguishing CB from periapical inflammatory disease [3,7,12]. The lesion grows slowly but persistently, at an estimated rate of approximately 0.5 cm per year, and pediatric cases may display comparatively aggressive local behavior [3,16]. Mandibular lesions occasionally produce lower-lip paresthesia, compression and displacement of the inferior alveolar canal, or even pathological fracture; maxillary lesions may extend into the maxillary sinus and nasal cavity [3,7,15,18]. Unusual presentations involving primary teeth, impacted teeth, multiple teeth, or multiple synchronous lesions add considerably to the diagnostic and surgical challenge [9,13,14,16,18].

3. IMAGING

3.1. Conventional radiography and the "halo" sign

The classic radiographic appearance is a round or ovoid, densely radiopaque mass fused to the root of the affected tooth, sharply demarcated and encircled by a thin, fairly uniform radiolucent band — the so-called "halo" sign. Loss of the periodontal ligament space, obscuration of the root outline, root resorption and displacement of adjacent teeth are common accompanying findings [3,7,10,12]. Early lesions may present only as a periapical radiolucency and be mistaken for apical periodontitis, with the result that the tooth is subjected to unnecessary pulp extirpation or root canal treatment [3,7]. Xu et al. also described secondary changes — a disorganized trabecular pattern around the lesion, narrowing or obliteration of the marrow space, and thickening of the inferior mandibular cortex — which they attributed to local secondary infection or reactive bone formation [3].

3.2. Cone-beam computed tomography and three-dimensional assessment

Because planar images cannot demonstrate buccolingual extension, cortical integrity or the spatial relationship to the inferior alveolar canal, CBCT has become central to preoperative

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evaluation. Xu et al. classified their 14 cases by internal density into a homogeneous type (6 cases, 42.9%) and a heterogeneous type (8 cases, 57.1%), the latter showing lacunar or spoke-like radiolucent areas. All six patients examined with CBCT showed buccolingual expansion with thinning and focal discontinuity of the cortex; ten demonstrated perilesional trabecular disorganization and reactive sclerosis; panoramic radiographs showed inferior displacement of the mandibular canal in seven cases; and both maxillary cases showed discontinuity of the sinus floor [3]. Three of the eight heterogeneous lesions recurred, raising the possibility that imaging phenotype carries prognostic information, although the authors explicitly cautioned that any relationship to histology, surgical approach or outcome remains to be validated [3].

Preto et al. described an aggressive CB in a 31-year-old man in whom multiplanar CBCT reconstructions demonstrated a mixed-density, irregularly marginated mass fused to the root, together with obliteration of the periodontal ligament space, external resorption of the distal root, expansion and perforation of the buccal plate, and invasion of the roof of the mandibular canal; these findings directly altered the planned resection [17]. Noviyanti et al. reported a giant CB in a 19-year-old woman in which CT showed a densely ossified mass extending widely in the buccolingual dimension, continuous with the apex of the first molar on sagittal views and surrounded by a well-defined low-density rim; the size of the lesion and cortical perforation ultimately necessitated hemimandibulectomy [9]. More recently, Riahi et al. described an asymptomatic recurrence detected incidentally on CBCT performed for implant planning seven years after extraction, underscoring the value of three-dimensional imaging in long-term surveillance and pre-prosthetic assessment [19].

3.3. From characterization to risk stratification

The role of imaging in CB has thus expanded from qualitative diagnosis to risk stratification. In the pooled analysis of Chrcanovic and Gomez, bone expansion and cortical perforation were significantly associated with recurrence [8], and CBCT is the most sensitive and readily available means of detecting both [17]. Preoperative CBCT assessment of cortical integrity and lesion margins should therefore be regarded as mandatory whenever conservative surgery or tooth preservation is contemplated. Magnetic resonance imaging can help delineate soft-tissue involvement and bone marrow edema and can assist in excluding a malignant bone tumor, but the available evidence is largely anecdotal and no systematic evaluation has been published.

4. HISTOPATHOLOGY AND DIFFERENTIAL DIAGNOSIS

4.1. Gross and microscopic features

Grossly, the specimen is a spherical calcified mass attached to the root, enveloped by a complete fibrous capsule, truly fused with the root, and hard on sectioning [2,7]. Microscopically, abundant cementum-like (or osteoid-like) mineralized matrix is arranged in sheets and rounded or ovoid lobules, with conspicuous basophilic reversal lines that produce a Paget-like "jigsaw" pattern. The matrix is rimmed by plump, active cementoblast-like cells within a richly vascular fibrous stroma, and multinucleated osteoclast-like giant cells are commonly present [7,10,21,22]. The center of the lesion is densely mineralized and relatively paucicellular, whereas the periphery contains a cellular, actively growing zone with radially arranged unmineralized osteoid; root resorption is frequently seen [7,10]. Neoplastic cementoblasts are morphologically indistinguishable from osteoblasts, may show hyperchromatic and variably sized nuclei, and, when interpreted without the clinical and radiographic context, are readily mistaken for osteosarcoma [7,10].

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Table 1. Differential diagnosis of cementoblastoma and other radiopaque periapical lesions of the jaws

Entity	Relationship to the root	Pulp vitality	Key distinguishing features
Cementoblastoma	True fusion; loss of the root outline and periodontal ligament space	Usually vital	Round or ovoid radiopaque mass with a thin, uniform "halo"; slow growth; may expand the jaw
Hypercementosis	Root contour preserved; localized thickening of the root surface	Vital	No discrete mass and no jaw expansion; associated with trauma, occlusal loading or aging
Periapical cemental dysplasia / cemento-osseous dysplasia	No fusion; often multifocal	Vital	Middle-aged women, mandibular anterior region; irregular radiolucent rim; lesions mature spontaneously
Condensing osteitis	No fusion	Usually non-vital or a history of pulpitis	Evidence of caries or pulpal disease; ill-defined radiopacity, no capsular rim, no expansion
Odontoma	No fusion; usually pericoronal	Vital	Tooth-like structures or dental hard-tissue density; often associated with an impacted tooth; anterior region
Periapical bone island	No fusion	Vital	No radiolucent rim; well defined with brush-like margins; intact periodontal ligament; no expansion [20]
Osteoblastoma	No fusion; arises within the medullary cavity	Unrelated to the tooth	Histologically near-identical to CB; distinguished by imaging and intraoperative attachment to the root [21,22]
Osteosarcoma	No fusion	Variable	Rapid growth, ill-defined margins, periosteal reaction, soft-tissue mass, atypical mitoses [23,24]

4.2. The boundary with osteoblastoma

Slootweg's comparative study of 1992 established just how closely CB and osteoblastoma resemble one another histologically [21]. Subsequent WHO classifications and authoritative reviews have consistently held that true continuity with the tooth root is the only reliable feature separating the two [4,7,22]. The diagnosis of CB is therefore, by definition, an integrated clinico-radiological-pathological judgment: a small biopsy that does not include the root–tumor interface can at most be reported as "consistent with an osteoblastoma-like lesion" and cannot independently establish or exclude CB [7,10].

4.3. Differential diagnosis

The differential diagnosis of a radiopaque periapical lesion is broad, and the most consequential distinction is from osteosarcoma. Active cementoblasts may exhibit

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pleomorphism, and recurrent lesions can contain epithelioid cells with large, bizarre nucleoli; morphology alone can therefore lead to an erroneous diagnosis of malignancy [10,24]. Oya et al. reported a 29-year-old woman who developed a recurrencence five years after initial enucleation, with a well-circumscribed multifocal radiopaque lesion and marked cytological atypia. The absence of atypical mitoses and low proliferative activity, together with c-FOS positivity, supported a diagnosis of benign recurrent CB [24]. The principal entities to be considered, and the features that distinguish them, are summarized in Table 1.

Li et al.'s radiographic analysis of jaw bone islands provides directly citable Chinese-language evidence for distinguishing periapical bone islands from CB [20]; Estrela et al. systematically reviewed the differential diagnosis and management of periapical radiopaque or hyperdense jaw lesions, emphasizing the combined use of pulp vitality testing and three-dimensional imaging [23]; and Sankari and Ramakrishnan reiterated, through an illustrative case, the need for radiological and histological findings to be interpreted together [25].

5. MOLECULAR PATHOGENESIS

5.1. FOS/FOSB rearrangements: from bone tumors to odontogenic tumors

In 2018, Fittall et al., using whole-genome and transcriptome sequencing, identified recurrent FOS rearrangements — and, less commonly, FOSB rearrangements — in osteoid osteoma and osteoblastoma, and confirmed by fluorescence in situ hybridization (FISH) and immunohistochemistry that these alterations were near-universal in a validation cohort of 55 cases. Their work effectively redefined this group of benign osteoblastic tumors as a molecular entity driven by FOS/FOSB dysregulation [26]. Given the striking morphological overlap between CB and osteoblastoma, Lam et al. examined 16 CBs in 2021: immunohistochemistry showed strong, diffuse nuclear and cytoplasmic c-FOS expression in 71% of cases, and all three cases in which FISH was evaluable carried an FOS rearrangement; the remaining specimens failed FISH because of decalcification-induced nucleic acid damage [27]. The authors proposed that CB and osteoblastoma may represent different manifestations of a single disease spectrum [27]. This study remains the most consequential advance in the field: it provided the first reproducible molecular marker for CB and made c-FOS immunohistochemistry a practical ancillary tool in diagnostically difficult cases [24,27].

5.2. The fifth-edition WHO update and the ensuing debate

In their authoritative account of the changes introduced in the 2022 fifth edition, Soluk-Tekkesin and Wright noted explicitly that CB had acquired a molecular update — c-FOS expression and the same FOS rearrangement found in osteoblastoma — and identified the unresolved question this raises: is CB an independent odontogenic tumor, or merely a gnathic bone tumor within the osteoid osteoma–osteoblastoma spectrum [4]. The fifth edition also separated bone- and cementum-forming lesions peculiar to the jaws from the general bone tumor classification, placing them in a dedicated chapter on odontogenic and maxillofacial bone tumors, and added essential and desirable diagnostic criteria for every entity [4,28]. A 2026 update on the molecular pathology of jaw lesions by Guimarães, Odell and Gomes adopted a more cautious position: genetic findings in bone- and cementum-forming odontogenic tumors are "not consistent" across studies, molecular evidence supports the current classification in most instances but leaves areas of uncertainty, and jaw lesions differ from morphologically similar lesions at other skeletal sites [28].

5.3. Emerging evidence and its diagnostic limits

In 2025, Tsuchiya et al. reported an osteoblastic/cementoblastic tumor of the jaw in a 20-year-old man that was difficult to classify because psammomatous or dot-like calcifications and

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scant osteoid matrix mimicked CB. Whole-genome sequencing and nanopore methylation analysis revealed a previously undescribed FOS::FN1::FOS fusion; the methylation profile clustered with osteoblastoma and the copy-number profile was essentially flat, leading the authors to designate the lesion as a new morphological subtype of osteoblastoma [29]. The case is instructive in two respects: the jaws can generate unusual morphological spectra, and molecular characterization is increasingly becoming the arbiter of classification in this group of lesions [28,29]. In the same year, Lam et al. showed in a cell-of-origin model of osteoid osteoma and osteoblastoma that truncated FOS impairs osteogenic differentiation and cell proliferation while upregulating prostaglandin and NF-κB inflammatory signaling—findings that offer a mechanistic explanation for woven bone formation, indolent biological behavior, and the characteristic analgesic response to nonsteroidal anti-inflammatory drugs [30]. It must be emphasized, however, that this model was not derived from CB and the results cannot be extrapolated directly.

In diagnostic practice, c-FOS immunohistochemistry is most useful in small biopsies, recurrent lesions, and cases in which osteosarcoma is a concern [24,27]. Its limitations are equally clear. Specimens containing dental hard tissues usually require decalcification, which severely compromises nucleic acid quality and produces high failure rates for FISH and sequencing [27]; c-FOS positivity is not specific to CB either, being shared by osteoblastoma, osteoid osteoma and some epithelioid hemangiomas [26,27]. Molecular markers should therefore be treated as one component of an integrated diagnosis rather than a substitute for the combined clinical, radiographic and histological assessment.

6. TREATMENT, RECURRENCE AND FOLLOW-UP

6.1. Surgical principles

The prevailing approach is complete excision of the tumor with simultaneous extraction of the involved tooth, supplemented by thorough curettage of the tumor bed or peripheral ostectomy to remove any residual lesional tissue [7,8,10]. Brannon et al. specifically argued that retained tumor continues to grow and gives rise to recurrence, and accordingly recommended en bloc removal together with the tooth plus meticulous curettage or peripheral ostectomy as the standard operation [7]. For small, well-circumscribed lesions, enucleation with extraction of the affected tooth, or tumor removal at the time of minimally invasive extraction, may suffice: Guo et al. described a case of multiple CB treated by enucleation, extraction and grafting of the defect with a mixture of bone substitute and autologous bone marrow [13], while Zheng and Zhao used sectioning of an impacted third molar and ultrasonic bone surgery to create space, removed the tooth and tumor atraumatically, and reported an uneventful recovery [14]. Large lesions, those with cortical perforation, and those involving several teeth require partial or segmental jaw resection, or even hemimandibulectomy with reconstruction [3,9,32]. In recurrent disease adjacent to the inferior alveolar nerve, piezosurgery can improve osteotomy precision and reduce operative trauma [19].

6.2. Reappraising the recurrence rate

Of Brannon et al.'s 44 cases, 35 had follow-up (mean 5.5 years) and 13 recurred (37.1%) — a figure far higher than the 2 recurrences among 34 previously published cases with follow-up (5.9%) included in their literature review. Recurrent cases more often showed jaw expansion and cortical perforation, prompting the authors' conclusion that CB is not entirely innocuous [7]. Chrcanovic and Gomez refined this picture using a much larger dataset: among 258 cases, 229 had treatment data and 170 had follow-up, with 20 recurrences (11.8%); the odds ratio for recurrence was 7.875 in lesions with bone expansion ($P = 0.048$) and 3.173 in those with cortical perforation ($P = 0.023$), whereas tooth retention, lesion site and recurrence were not

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significantly associated [8]. In the Chinese series of Xu et al., 3 of 14 cases recurred (21.4%); two were maxillary and one recurred repeatedly, with first recurrences at 7, 12 and 32 months after surgery [3]. The spread among these three estimates indicates that recurrence depends heavily on completeness of excision, case mix, lesion site and duration of follow-up, and that the figures should not be compared directly (Table 2).

Table 2. Major evidence on recurrence after treatment of cementoblastoma

Study	Sample and follow-up	Recurrence rate	Main conclusions
Brannon et al., 2002 [7]	44 cases; 35 with follow-up (mean 5.5 years)	37.1% (13/35)	Recurrence associated with jaw expansion and cortical perforation; complete excision with the tooth plus thorough curettage recommended
Chrcanovic and Gomez, 2017 [8]	258 cases; 170 with follow-up data	11.8% (20/170)	Lower than previously assumed; OR = 7.875 for bone expansion, OR = 3.173 for cortical perforation
Xu et al., 2015 [3]	14 cases with postoperative follow-up	21.4% (3/14)	Recurrences clustered in the heterogeneous imaging type; maxillary site and surgical approach may influence outcome
Oya et al., 2024 [24]; Riahi et al., 2026 [19]	Single cases; recurrence at 5 and 7 years, respectively	—	Recurrence may be late, asymptomatic and multifocal, with cytological atypia; long-term imaging surveillance is required

6.3. The tooth-preservation debate and surveillance

A small number of early, limited lesions have been managed by periapical surgery or endodontic treatment with retention of the tooth [8,9], and the pooled analysis by Chrcanovic and Gomez did not find tooth preservation to increase recurrence significantly [8]. Nevertheless, multiple case series and case reports emphasize that residual tumor is the principal source of recurrence and that recurrent disease may be multifocal, cytologically atypical, or bulky [7,24,31,32]. A defensible position is therefore to recommend en bloc excision with the involved tooth in routine practice, reserving tooth preservation for carefully selected early cases managed with fully informed consent and a guaranteed long-term follow-up. Surveillance should extend over years rather than months: recurrences at five and seven years have been documented [19,24], clinical examination combined with panoramic radiography or CBCT is the mainstay, and CBCT should be repeated routinely before implant placement in any patient with a history of CB [19]. Cytological atypia in a recurrent lesion does not imply malignant transformation; mitotic activity, proliferative index, radiographic margins and c-FOS expression should be weighed together to avoid overtreatment [24].

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7. THE CHINESE-LANGUAGE LITERATURE: A CNKI-BASED APPRAISAL

A title-field search of CNKI for cementoblastoma yielded only 14 records—13 journal articles and one master's thesis—published between approximately 2001 and 2022, the great majority being reports of one or two cases or literature reviews [13,14,33,34]. The studies with the largest case volumes are Fang et al.'s clinicopathological analysis of 12 cases (2006), which summarized the age and site distribution, the characteristic "central radiopacity with a peripheral radiolucent ring", and the basophilic reversal lines seen histologically [10]; Xu et al.'s clinical and radiographic analysis of 14 cases (2015), which proposed the homogeneous/heterogeneous imaging classification and reported a 21.4% recurrence rate [3]; and Qi's 20-case analysis (2019 master's thesis), which documented a notably wide age range of 7 – 69 years and provided a relatively comprehensive discussion of radiological differential diagnosis [11]. The remaining literature consists of a report of multiple CB with bone grafting (Guo et al., 2010) [13], a two-case series with review (Sun et al., 2015) [33], a case report with review (Liu et al., 2008) [34], and the minimally invasive management of an impacted third molar associated with CB described by Zheng and Zhao in 2022 [14]. Collectively these studies have built a valuable body of local evidence on the clinical and imaging features of Chinese patients, and their contributions to imaging classification, minimally invasive technique and differential diagnosis are of genuine regional value.

The contrast with the topic-field results is instructive. A large share of the 175 records concerned the basic biology of normal cementoblasts — hypoxia, ferroptosis, glutamine metabolism, mineralization and root resorption — and had nothing to do with the tumor itself. Basic research on cementoblast biology in China is thus comparatively active, whereas investigation of CB as a neoplasm remains largely descriptive: no Chinese-language report has yet described validation of FOS rearrangement, molecular subtyping, the diagnostic application of c-FOS immunohistochemistry, or a multicenter prognostic cohort in CB. Closing this gap is arguably the most realistic and potentially original contribution that domestic groups could make in the near term.

8. CURRENT GAPS AND FUTURE DIRECTIONS

The most fundamental limitation is the low level of existing evidence. Almost everything known about CB derives from case reports and retrospective case series; there are no prospective multicenter registries, and questions as consequential as the true recurrence rate, the safety of tooth preservation and the prognostic value of imaging phenotypes lack high-level support [3,8].

A second unresolved issue is the incomplete molecular picture. The recurrent fusion partners of FOS, the downstream AP-1 signaling network, and the similarities and differences between the methylation profiles of CB and osteoblastoma have yet to be clarified in large series [26,27,28,29]. Because decalcification of specimens containing dental hard tissues is a major obstacle to molecular testing [27], standardized tissue procurement — reserving undecalcified tissue or using gentle EDTA decalcification — targeted RNA sequencing, comparative methylation profiling, and multicenter biobanking are urgent priorities.

Nosological uncertainty is a third area requiring attention. Whether CB should be reclassified from an odontogenic tumor to the osteoid osteoma–osteoblastoma spectrum demands convergent evidence from clinical, imaging, molecular and epigenetic studies [4,28,29]. Until such evidence is available, the fifth-edition WHO framework should be retained and pathology reports should state both the molecular result and its limitations.

Diagnostic pathways also need standardization. An integrated sequence of clinical examination, pulp vitality testing, CBCT, histopathology and — where indicated — c-FOS

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immunohistochemistry should be adopted more widely, chiefly to prevent two recurring errors: misinterpreting active cementoblasts in a small biopsy as osteosarcoma, and performing unnecessary pulp extirpation on a vital tooth because of pain [3,10,24,27].

Treatment and surveillance should become more refined. CBCT-based identification of bone expansion, cortical perforation and mandibular canal involvement lends itself to a formal preoperative risk stratification; follow-up should be extended beyond five years, and repeat imaging before implant rehabilitation should become routine [17,19]. Recognition that recurrent lesions may show cytological atypia and multifocal growth should temper any impulse toward indiscriminately radical surgery [24,31].

Finally, an emerging therapeutic hypothesis is worth watching but should not be overstated. The prostaglandin/NF-κB signaling associated with truncated FOS, and its link to the NSAID response, point toward possible adjunctive pharmacological strategies [30]; the evidence, however, comes from osteoid osteoma and osteoblastoma models and remains far from clinical application in CB.

9. CONCLUSION

Cementoblastoma is a rare benign odontogenic tumor with a virtually pathognomonic radiographic appearance, yet one whose diagnosis still depends on the integration of clinical, imaging and histological findings. Two developments over the past decade stand out. Molecular pathology has established FOS rearrangement and c-FOS overexpression, an advance incorporated into the fifth WHO edition that has reopened the question of where the lesion belongs nosologically. In parallel, large pooled analyses have revised the recurrence rate from 37.1% to 11.8% and identified bone expansion and cortical perforation as risk factors that can be recognized on preoperative CBCT. Chinese-language work has contributed useful observations on imaging classification and minimally invasive surgery, but molecular validation and cohort studies are still missing. The next phase should be built on multicenter collaboration, standardized molecular testing, an integrated diagnostic pathway and long-term follow-up, with the aim of converting imaging-based risk stratification into reproducible, widely applicable clinical guidance.

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