

Generalized Hypercementosis: Report of a Rare Case and Review of the Possible Role of Two Identified Predisposing Factors

***Ifeyinwa E. UCHE, ** Babatunde Olamide
BAMGBOSE* Jamiu Adetunji ABANIKANND**

**Department of Preventive Dentistry, Faculty of
Dentistry, Bayero University; Department of Preventive
Dentistry, Aminu Kano Teaching Hospital, Kano,
Nigeria.*

***Department Oral Diagnostic Sciences, Faculty of
Dentistry, Bayero University; Department of Oral and
Maxillofacial Surgery and Radiology, Aminu Kano
Teaching Hospital, Kano, Nigeria.*

Correspondence

*Dr. Jamiu A. Abanikannda
Department of Preventive Dentistry
Faculty of Dentistry, Bayero University
Kano, Nigeria
Email: jaabanik@gmail.com*

Received: 27th July, 2020

Revision: 16th October, 2020

Accepted: 20th June, 2021

ABSTRACT

Objective: In this paper, a case is presented of a female adult patient with generalized hypercementosis (GHC) which could only be attributed to two rarely reported predisposing factors. It also seeks to critically review the literature concerning the possible role of two identified predisposing factors.

Case Description: A case of a 50-year-old female trader with generalized hypercementosis discovered as an accidental radiographic finding in the process of assessing her periodontal status. She presented with complaint of gradually increasing mobility leading to loss of some anterior teeth in the upper left quadrant resulting in poor facial aesthetics and the desire to have the lost teeth replaced. Examination revealed a case of oral neglect and generalized periodontitis. Patient's facial features revealed disfigurement and increased facial height due to generalized excessive eruption. The patient was systemically healthy except for bilateral knee osteoarthritis.

Conclusion: Periodontitis and osteoarthritis can be associated with the development of hypercementosis. The dental surgeon should, therefore consider these as possible risk factors in patients presenting with hypercementosis.

Keywords: Generalized hypercementosis, periodontitis, osteoarthritis

Ifeyinwa E. Uche

<https://orcid.org/0000-0002-3605-7004>

Babatunde O Bamgbose

<https://orcid.org/0000-0002-1287-8406>

Jamiu Adetunji Abanikannda

<https://orcid.org/0000-0002-1852-051x>

Citation: Uche IE, Bamgbose BO, Abanikannda JA. Generalized hypercementosis: Report of a rare case and review of the possible role of two identified predisposing factors. Nig J Periodontal Res 2021; 2(2):58-65.

CLINICAL IMPLICATIONS

The roughness of the root surface in hypercementosis may favour the bacterial contamination leading to localized bone loss with marked gingival recession and overall poor prognosis of the affected tooth. Generalized hypercementosis always suggest the possibility of the presence of osteitis deformans or Paget's disease. On rare occasions, hypercementosis can be extensive as to cause the fusion of two or more adjacent teeth, by a layer of cementum, a condition known as acquired concrescence. One of the only practical clinical implications of hypercementosis is the difficulty encountered during extraction of affected teeth. This may suggest the true biologic significance of hypercementosis, is to anchor the tooth in the socket more firmly. This may warrant the need of sectioning the tooth in certain cases to aid removal during tooth extraction.

INTRODUCTION

Hypercementosis (HC) also called cemental hyperplasia is defined as excessive laying down of non-neoplastic cementum contiguous with the normal root cementum of one or more teeth in quantities beyond physiological requirements. The excess cementum could be limited to either a portion or the entire root surface, resulting in abnormal thickening of the affected root portion, thereby altering its shape macroscopically.¹⁻⁵ Hypercementosis may be localized or generalized and may exist either in the typical or atypical forms.⁶ Typically, it is age related and more commonly observed in older patients.^{6,7} The atypical form is virtually seen in young individuals and carries a high suspicion of hereditary link and morphologically appears as spikes and outgrowths^{2,5,6,8} seen in focal areas on the root surface as external cementum projections.⁹

Hypercementosis is considered to be essentially idiopathic. However, several factors have been hypothesised as possibly aetiological including local and systemic factors.^{2,6} Presence of local infection was hypothesized as a factor in the aetiopathogenesis of hypercementosis,¹ and several authors^{1,10,11} pointed out that inflammation of low intensity is a more likely stimulus for the excess deposition of cementum. Though both Pulpal and periodontal infections have been indicted in literature as locally associated risk factors, most available literature have failed to recognize the existence of a possible association between periodontitis and hypercementosis, thus pulpal

infection is commonly cited. Most cases of generalized hypercementosis (GHC) are reported in association with systemic conditions such as Paget's disease.

In this paper, a very rare case is presented of generalized hypercementosis associated with generalized periodontitis in a postmenopausal woman with bilateral knee osteoarthritis.

CASE REPORT

A 50-year-old female trader, presented to the periodontology clinic of Aminu Kano teaching hospital, in Kano Nigeria with a complaint of gradual loss of teeth which started over five years ago and the desire for prosthetic replacement. There was no history of trauma of any sort. She experienced gradually increasing mobility of the teeth, with no associated pain, followed by eventual loss of the affected teeth. She noticed increasing spacing in-between her teeth over the past few years with discomfort during mastication due to food particles hanging in-between the teeth, prompting the patient to use wooden tooth picks. There was history of bleeding during tooth cleaning.

Patient had no history of past dental visit or treatment. No history of tumour or radiation therapy. The only remarkable systemic condition was bilateral knee osteoarthritis which had been symptomatic for about four years without medical attention. She was not on any medication at presentation. She cleans her teeth with chewing stick only. She rarely consumes refined carbohydrate diets, does not smoke nor consume alcoholic beverages. Her last menstrual period was over 5 years.

On general physical examination, patient was healthy looking, not in any obvious distress, not pale, anicteric, acyanosed, not dehydrated. No acral enlargement was seen. However, she is obese. Her BMI was 39.64 Kg/M²

Extraoral examination revealed facial asymmetry occasioned by some degree of upper labial collapse on the left, increased anterior facial height all leading to poor facial aesthetics. There was no detectable abnormality of the temporomandibular joints. The submandibular lymph nodes were palpably enlarged but not tender.

On intraoral examination, there was generalized inflammation of the gingiva with loss of stippling, soft consistency, redness, rounded and blunted gingival margin, bulbous interdental papillae and generalized bleeding on gentle probing. There was gross accumulation of debris, plaque and calculus.

The oral hygiene was poor with simplified oral hygiene index ¹⁴ of 4.6. There was generalized periodontal destruction, bimaxillary proclination, generalized spacing of the teeth and extensive loss of clinical attachment. In addition, there was generalized gingival recessions, furcation involvement of teeth 14, 16, 17, 24, 36, 27, 36 and 37 and mobility of teeth 11, 12, 13, 17, 24, 31, 32, 33, 42, 44, 45 and 46. The missing teeth were 21, 22, 23 and 41. Pulp vitality testing of all standing teeth with an electric pulp tester was done, followed by thermal (cold) pulp testing, using endo ice which revealed all teeth to be vital.

True bitewing radiograph and periapical series revealed generalized peri radicular radiopacities continuous and indistinguishable from the original roots' cementum giving them club shaped appearance. The radiopacities involve almost the entire root length of most teeth, including the anterior teeth. The lamina dura was preserved and the periodontal ligament spaces were otherwise unaffected, except for its widening around teeth 36, 37, 46 and 47. There were generalized moderate to severe horizontal pattern alveolar bone loss. However, and interestingly, the degree of mobility of the teeth was less compared to the extent of bone loss around them.

There was no ocular, dermatological or oral manifestations of Vitamin A, or other vitamin deficiency. Laboratory investigations including Fasting blood glucose level, electrolyte, urea and creatinine, serum proteins, thyroid function tests, were within normal limits. There was moderately elevated serum inorganic phosphate and a minimally reduced serum calcium level. Rheumatoid factor test was negative.

A diagnosis of generalized periodontitis with generalized hypercementosis in a patient with bilateral knee osteoarthritis was made.

Treatment

Periodontal: She was informed about the finding of generalized hypercementosis and the clinical implication, as well as the periodontitis. She was educated with emphasis on oral hygiene care, proper brushing methods, use of floss and interdental brush and the need for regular dental checkup.

Full mouth scaling and polishing was done, followed by subgingival scaling and root planing of all teeth, at one quadrant per week. Patient was placed on the following oral medications: Doxycycline capsules 100mg once daily for two weeks, Metronidazole

400mg 8 hourly for a week, Ascorbic acid 100mg 8 hourly for two weeks, and also 0.2% Chlorhexidine gluconate mouth rinse, 10mls 12 hourly for two weeks. She was scheduled for regular 3 monthly periodontal reviews.

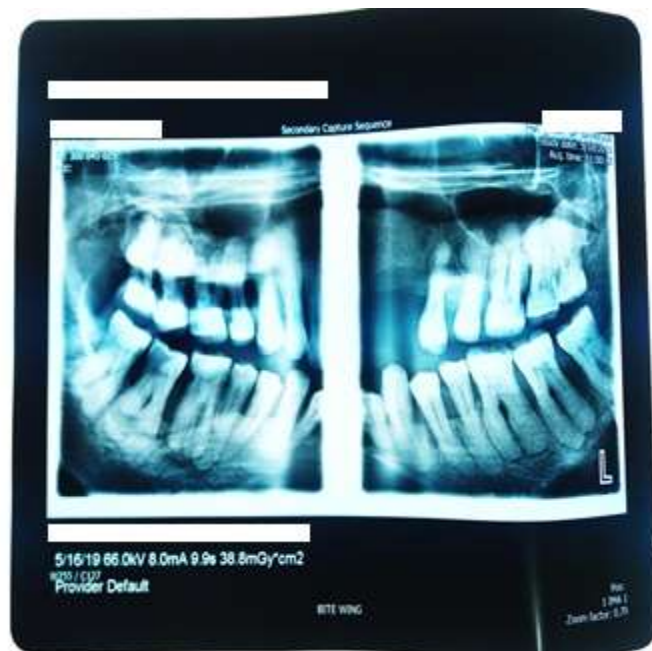
Oral surgery: She was subsequently referred to the oral surgery clinic for extraction of teeth 11, 24, 31, 32, 33 and 46. Patient consented to the extraction of only teeth 31 and 36 which were grossly mobile at presentation. The extracted teeth were sent for histopathological analysis. They displayed bulbous roots, consistent with the radiographic observation. Histopathology result revealed thickened cementum layers, confirming the radiological diagnosis of hypercementosis.

Restorative: She was also referred to the restorative dentistry clinic, where she was reviewed and planned for prosthetic replacement with flexible removable partial denture, with flange to camouflage the gingival recession.

Medicine: Patient was referred to the rheumatologists, who reviewed her and confirmed the diagnosis of bilateral knee osteoarthritis, worse on the right knee. She was placed on medications, advised on need for regular exercise for weight reduction and appropriate lifestyle modification.

DISCUSSION

Although the exact aetiology is obscure, some authors^{6, 7, 8, 16, 17} see HC (especially the typical form) as an idiopathic, age-related phenomenon. Some investigators concede that certain cases of HC in humans were observed to be unassociated with any remarkable finding when evaluated for presence of an attributable systemic condition.^{6, 7} Despite these, other researchers^{1, 10, 11, 15, 16} have implicated certain factors in the aetiology of hypercementosis, including continuous eruption of the teeth, presence of inflammatory reaction, functional stress resulting from excessive occlusal forces, inclusion of periodontal cementicles during normal cemental deposition and conservative cemental deposition in reaction to periapical inflammation. Thoma and Goldstein¹⁰ affirmed that continuous eruption of the teeth and presence of inflammatory reaction are responsible for secondary cementum deposition. The other factors included formation of spicules and root repair. Shafer et al.¹¹ in agreement with Thoma and Goldman¹⁰ proposed that low-intensity inflammation, may be responsible for cementum deposition.



Weinberger¹⁶ in his review of the clinical significance of hypercementosis was hesitant in accepting hypercementosis to be the result of inflammation from diseased pulp. His ground of doubt was the observation that evidence for root enlargement in the periapical area of teeth with diseased pulp is lacking.

Consolaro et al.¹⁸ deduced that the commonest causes of HC are increased occlusal demand, absence of an antagonist, passive eruption leading to extrusion and chronic periapical lesions.¹⁸

Unlike localized hypercementosis involving one or a few teeth, which is usually associated with a localized cause, most cases of GHC are linked with one form of systemic conditions or the other, with symmetrical enlargement of entire roots. Such systemic conditions include atherosclerosis, deforming arthritis (osteoarthritis), rheumatoid arthritis, exophthalmic goiter, acromegaly, calcinosis, vitamin A deficiency and Paget's disease.^{2,16,18,19} It is very important that a possible systemic association be ruled out in every case of

GHC to ensure appropriate and adequate management of such patient.

There are quite a few cases of GHC reported in the literature.¹⁹⁻²¹ Lieder and Garbarino² reported a case of GHC in a 76-year-old female with extensive hypercementosis involving nearly all that remained of the patient's dentition. The case was associated with rheumatoid arthritis, diagnosed 58 years earlier; although the time the GHC commenced could not be ascertained. All the remaining teeth, except three were vital. The patient had no significant periodontal problem.

Seed and Nixon (2004)²² also reported a case of GHC quite similar to the present case in that the patient also presented with a complaint of poor aesthetics, was an irregular dental attendee and had periodontal disease resulting from dental neglect with no remarkable medical history. Therefore, periodontitis is the only identifiable factor attributable with the reported GHC.

Patil and Yadav (2015)²¹ reported a case of GHC in a 20-year-old lady that was associated with multiple missing teeth of idiopathic origin. Even though radiographic and laboratory investigations were done without any positive finding, it is possible the cause could be an unidentified genetic anomaly, moreso that evidence abounds in the literature associating most cases of hypercementosis in young age groups with a genetic link.^{2,6,8, 23, 24}

Shah et al (2018)²⁵ reported a case of GHC in 56-year-old Indian woman, where they diagnosed idiopathic GHC. In their report however, only radiographic investigations were mentioned. They did not mention laboratory or other investigations to prove convincingly that a substantive systemic or local factor does not exist in relation to the case. However oral neglect, as mentioned by these authors, was the only factor put forward which may suggest poor periodontal health.

Butala and Patel (2018)²⁰ also reported a 58-year-old woman with hypercementosis involving 15 out of 26 remaining teeth in her mouth. Their presentation was silent about an underlying cause as no investigation was taken to rule out a systemic link. Interestingly, generalized accumulation of plaque and calculus, severe gum bleeding, recession of gingivae and periodontal pockets were observed in the patient pointing to the presence of periodontitis. In the present case, systemic conditions such as Paget disease, rheumatic fever, hyperthyroidism, acromegaly, Vitamin A deficiency, calcinosis were all ruled out with thorough clinical examination and appropriate laboratory/ radiological investigations.

The only remarkable medical condition was a diagnosed bilateral knee joint osteoarthritis. Ruling out these conditions are essential to conclude that a case is idiopathic. The probable explanation for the generalized hypercementosis in this present case is partly the presence of periodontitis resulting from oral neglect. This is in agreement with the observation of other authors.^{8,20,22,25}

Zhou et al.⁸ were the first authours to explicitly attribute HC to periodontitis. Even though their reported case was that of localized HC. The explanation was that the HC could be a natural attempt at stabilizing the periodontally compromised teeth against occlusal load by augmenting the total surface area of the roots for better distribution of occlusal forces, which is in agreement with explanation by Consolaro et al.¹⁵ Humerfelt et al.⁶ explained that absorption and distribution of forces acting on the teeth by the periodontium may put periodontal fibres in motion and possibly induce a cytoskeletal deformation in the cementoblasts, resulting in activation of cellular stress. The increased stress then cause release of mediators and synthesis of cementum matrix on the root surface.^{6,18} This buttresses the explanation of HC being an adaptive response that comes to play when periodontal functional demands are increased such as when affected by periodontitis. This hypothesis may explain the clinical observation in our patient who presented with a long-standing generalized periodontitis with loss of three teeth to the disease process. Most of the remaining teeth had advanced loss of bony support and were all hypercementosed. The HC was able to stabilize the affected teeth despite their advanced alveolar bone loss. Since she has no parafunctional habit, the probable explanation for this is that the ordinary forces of mastication have become increasingly traumatic for the compromised periodontium and as such constituting a functional occlusal stress. The excess cemental apposition may be compensatory of periodontal tissue loss in order to increase area of support and distribution of occlusal forces in an attempt to stabilize the tooth.^{8,18} It is therefore pertinent to mention that the association of periodontitis with HC though grossly under reported in the literature is fairly plausible and the possibility of periodontitis predisposing to hypercementosis should not be overlooked.

With regards to the vitality of hypercementosed teeth, most reported cases involve teeth with preserved vitality.^{2,6,8} Contrary to this was a study where hypercementosis was reported to have

occurred 4.2 times more in teeth with necrosed pulp compared with those with vital pulp.¹ The observation in the present case conforms with studies in which the hypercementosis were found on vital teeth. The bone of contention therefore is resolving the fact that pulpal infection leading to necrosis and periapical inflammation was the more commonly indicted type of infection in the literature, with little mention of infection of periodontal origin.^{1, 6, 10, 26} In view of a low-grade inflammation as a local aetiological factor of HC as agreed by many authors,^{1, 10, 11} periodontitis affecting vital teeth seem to be more descriptive of a low-grade infection than periapical pathoses.

Concerning gender predilection, most authors agreed that HC has no gender predilection.^{19, 27} However, considering GHC in particular, all reported cases^{2, 20, 21, 22, 25, 30} affected female individuals similar to our experience. It is therefore necessary to determine the tendency for gender predilection concerning GHC in particular.

In relation to age of patients with HC, most reported cases involved patients within the 5th and 6th decade of life. The present case report involved a 50 year old woman, which fairly corroborates earlier studies. Hurzeler and Zander²⁶ observed from measurements of cementum from cut sections of randomly selected human teeth of ascending age range that root cementum, especially in the apical portion, increases gradually in a manner commensurate with aging. This seem to corroborate the hypothesis of HC being an age-related phenomenon,^{6, 7, 8, 16, 17} However, there are no defined yardstick to delineate what stands for age-related cementum thickness and/or functional demand and what can be called hypercementosis as a diagnosis.¹⁸

Most reported cases of hypercementosis were reported to spare the incisors, especially the lower incisors.^{2, 14, 28, 29} All teeth including incisors and canines were involved in the present case which is one of the most extensive hypercementosis case to be reported. The patient has advanced periodontal disease affecting the whole of the dentition.

A possibility of systemic association of osteoarthritis was present in our case, similar to an observation in some studies^{2, 16} in which generalized hypercementosis was linked with degenerative bony changes in some joints. Though some authors^{1, 2, 16} mentioned rheumatoid arthritis as a systemic condition possibly associated with HC, our patient tested negative to rheumatoid factor. She complained of knee pains and movement difficulties for which she was diagnosed of bilateral knee

osteoarthritis with presence of Baker's cyst in the right knee joint. The diagnosis was confirmed by radiologic findings of osteophytic outgrowths involving both medial and lateral femoro-tibial condyles as well as the superior pole of the patella. Weinberger classified HC into two types on a basis of radiographic study. The first type was identified as that in which the radiographic appearance of the excess cementum is indistinguishable from the original cementum, while in the second type, it is distinguishable. The first type was associated with hypertrophic arthritis (osteoarthritis), traumatic occlusion, Paget disease and acromegaly; while the second type was linked to rheumatic fever. In our case, considering this classification the excess cementum could not be distinguished from the original cementum, thereby supporting the existence of a link with patient's osteoarthritis. Also, the periodontal ligament space and lamina dura were preserved and there was no acral sign nor biochemical evidence of growth hormone derangement, thus ruling out Paget's disease and acromegaly.

CONCLUSION

There is lack of scientific studies investigating the role of periodontitis in the development of HC. The present case being reported appears to be an age-related occurrence of HC having osteoarthritis and possibly periodontitis as risk factors. However, this appear to be the first reported case in Africa of generalized HC existing concurrently with generalized periodontitis. Similarly, association between osteoarthritis and HC is being mentioned in the literature. It is however unclear the relative contributions of either of periodontitis or osteoarthritis in HC development. Further scientific research is recommended to establish and better explain more these observations, whether they are mere coincidence, association, or aetiological.

Financial Sponsorship

No external source of funding

Conflict of Interest

None declared

REFERENCES

1. Gardner BS, Goldstein H: The significance of hypercementosis. *Dental Cosmos* 1931; 73:1065-1069.

2. Leider AS, Garbarino VE. Generalised hypercementosis. *Oral Surg Oral Med Oral Pathol* 1987; 53:375-380.
3. Pappen FG, Fagonde CD, Martos J, Silveira LFM. Hypercementosis: a challenge for endodontic therapy. *RSBO* 2011; 8(3):321-328.
4. Pinheiro BC, Pinheiro TN, Capelozza ALA, Consolaro A. A scanning electron microscopic study of hypercementosis. *J Appl Oral Sci* 2008; 16(6):380-384.
5. Souza NL, Lima Junior SM, Garcia Santos Pimenta FJ, Rodrigues Antunes Souza AC and Santiago Gomez R. Atypical hypercementosis versus cementoblastoma. *Dentomaxillofac Radiol* 2004; 33:267-270.
6. Humerfelt A, Reitan K. Effects of hypercementosis on the movability of teeth during orthodontic treatment. *Angle Orthod* 1966; 36(3):179-189.
7. Azaz B, Michaeli Y, Nitzan D. Aging of tissues of the roots of nonfunctional human teeth. *Oral Surg, Oral Med, Oral Pathol* 1977; 43(4):572-578.
8. Zhou J, Zhao YF, Xia CY, Jiang L. Periodontitis with hypercementosis: report of a case and discussion of possible aetiologic factors. *Aust Dent J* 2012; 57:511-514.
9. Lilliana B, Bethania P, Rogerio A, Alberto C, Tiago P. Root apical third and canal morphology of teeth with hypercementosis. *Dent Press Endod* 2013; 3(3):23-31.
10. Thoma KH, Goldman HM. The pathology of dental cementum. *J Am Dent Assoc* 1939; 26: 1451-1461.
11. Shafer WG, Hine MK, Levy BM. A textbook of Oral Pathology. 4th ed. Philadelphia: Saunders; 1983.
12. Rushton MA. Anomaly of cementum in cleidocranial dysostosis. *Br Dent J* 1956; 100: 81-83.
13. Gardner AF. A survey of periapical pathology. *D Digest* 1962; 68:260-263.
14. Greene JC, Vermillion JR. The simplified oral hygiene index. *J Am Dent Assoc* 1964; 68:7-13.
15. Consolaro A, Consolaro RB, Francischone LA. Cementum, apical morphology and hypercementosis: A probable adaptive response of the periodontal support tissues and potential orthodontic implications. *Dental Press J Orthod* 2012; 17(1):21-30.
16. Weinberger A. The clinical significance of hypercementosis. *Oral Surg Oral Med Oral Pathol* 1954; 7(1):79-87.
17. Buch B, Matthee MJ. Radiological diagnosis IX. Hypercementosis. *J Dent Assoc S Afr* 1985; 40(1):23.
18. Consolaro A, Consolaro RB, Francischone LA. Hypercementosis and increase cementum thickness over the age: Clinical implications and meanings. *Dental Press Implantol* 2012; 6(1) 20-32.
19. Bender IB. Paget's disease. *J Endod* 2003; 29:720-723.
20. Butala PB, Patel PS. Generalized hypercementosis-A report of an accidental finding. *Int J Maxillofac Imag* 2018; 4(1):36-38.
21. Patil SR, Yadav N. Generalized hypercementosis with multiple missing teeth in a young female: A rare case report. *Int J Health Allied Sci* 2015; 4:178-180.
22. Seed R, Nixon PP. Generalized hypercementosis: A case report. *Prim Dent J* 2004; 119-122.
23. Jeddy N, Radhika T, Saravanan R, Prabakar R. Localised multiple cemental excrescences: A rare presentation of hypercementosis. *J Clin Diagn Res* 2014; 8(5):ZD16-ZD17.
24. Israel H. Early hypercementosis and arrested dental eruption: heritable multiple ankylodontia. *J Craniofac Genet Dev Biol* 1984; 4:243-246.
25. Shah D, Sutaria SP, Shah S and Dave B. Idiopathic generalized hypercementosis: A case report. *J Med Sci Clin Res* 2018; 6(12): 982-986.
26. Hurzeler B, Zander A. Determination of cementum thickness. *J Dent Res* 1958; 37(96):44.
27. Bürklein S, Jansen S, Schäfer E. Occurrence of hypercementosis in a German population. *J Endod* 2012; 38(12):1610-1612.
28. Consolaro A, Oliveira LU, Vasconcelos MHF. Determination of the prevalence of hypercementosis and its etiopathogenic implications. *Odontol Mod* 1987; 14(3):6-14.
29. Satish K, Surabhi R, Sanjay J, Veerendra P, Ratnakar P. Endodontic challenges presented by a hypercementosis tooth. *Int J Res Dent* 2015; 5(2):42-46.
30. Basdra EK, Stellzig A, Komposch G. Generalized hypercementosis in a young female patient. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 83(4):418-419.
31. Suter VG, Reichart PA, Bosshardt DD, Bornstein MM. Atypical hard tissue formation around multiple teeth. *Oral Surg Oral Med Oral Path Oral Radiol Endod* 2011; 111:138-145/

32. Rajendran R, Sivapathasundharam B. Shaffer's Textbook of Oral Pathology. 5th ed. New Delhi: Elsevier; 2006:585-587.
33. Vijay R, Chandan S. Hypercementosis: Review of literature and report of a case of mammoth, dumbbell shaped hypercementosis. J Indian Acad Oral Med Radiol 2015; 27:160-163.
34. Langlais RP, Langland OE, Nortje CJ. Diagnostic Imaging of the Jaws. 1st ed. Philadelphia: Williams & Wilkins; 1995:187-189.
35. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial Pathology. 3rd ed. New Delhi: Elsevier. 2009:96-97

INSTRUCTIONS TO AUTHORS

Nigerian Journal of Periodontal Research (NJPR) will publish two issues of annually: in JANUARY-JUNE and JULY-DECEMBER. NJPR accepts reports of original research articles, special review articles, histories and reports of rare and special cases, new sciences, discoveries and innovations in surgical techniques relevant to the study and practice of Periodontology.

Submission of an article for consideration for publication will be held to imply that it has neither been published nor is it under consideration for publication elsewhere. The editor reserves the right to edit manuscripts to ensure brevity and clarity of expression. The publishers also acquire all rights to the work upon acceptance of the manuscript by the Journal. Permission for reproduction must therefore be obtained by application in writing to the Editor-in-Chief. It is assumed that the author has considered ALL ethical aspects of his/her research and taken cognisance of the guidelines of the Helsinki declaration as revised in 1975 or the ethical guidelines of the institution(s) in which the work was done.

Following completion of the peer review process on every article, a decision of acceptance as submitted, acceptance with minor revision, acceptance with major revision or rejection will be communicated.

Preparation of Manuscripts It is required that manuscripts be prepared and referenced with the Vancouver system for biomedical manuscripts published by the International Committee of Medical Journal Editors; "Uniform requirements for manuscripts submitted to biomedical journals" Br. Med. J. 1982; 284: 1766-1770.

Manuscripts must be submitted in electronic form, using Microsoft word format Times New Roman on font size 12 via email of the email account of the corresponding author. Contributions must be double-spaced and the recommended outline for the organisation of articles is: **Title page, abstract, clinical implication(s) introduction, materials and methods, results, discussion, conclusions and recommendations (if any), acknowledgements, references, tables and legends.** Each manuscript component should begin on a new page.

Title Page: The title page should have the following: The title of the article which should be concise but

informative; A short running headline of no more than seven words placed at foot of the title page and identified; Â· Full names of each author (Surname last and in Uppercase letters), · Name(s) of department(s)/unit(s) and address of the institution in which the work(s) was/were done; Name and address of author responsible for correspondence about the manuscript.

Abstract and Key Words: The second page should have a structured abstract of no more than 250 words. Below the abstract, identify and provide three to ten key words. **Acknowledgements:** It is the responsibility of author(s) to obtain permission and approval of the wording from anyone acknowledged by name as readers may infer his/her endorsement of the data and conclusions. **References Number:** references consecutively in the order in which they are first mentioned in the text. Identify references in the text, tables and legends by arabic numerals (in superscript). Abstracts, "Unpublished observations", "personal communications" and "unaccepted papers" may not be used as references. However, references to written, not verbal communications may be inserted (in superscript). Examples of correct forms of references are given below: **Journals:** Standard Journal Article. (List all authors when seven or less; when ten or more, list only the first seven and add et. al.) Nwhator S, Opeodu O, Ayanbadejo P, Umeizudike K, Olamijulo J, Alade G, Agbelusi G et al. Could periodontitis affect time to conception? Ann Med Health Sci Res. 2014; 4(5):817-822. **Corporate Author:** The Royal Marsden Hospital Bone-Marrow Transplantation Team: Failure of syngeneic bonemarrow graft without preconditioning in post-hepatitis aplasia. Lancet 1977; 2: 242-244. **Personal Author(s):** Lisen, HN: Immunology: An introduction to molecular and cellular principles of the immune response. 5th ed. New York, Harper & Row, 1974, 406 pp. Chapter in a Book: Weinstein L, Swartz, MN.: Pathogenic properties of invading micro-organisms. In: Sodeman W. A. Jr., Sodeman, WA. eds. Pathologic physiology: mechanism of disease. Philadelphia, W. B. Saunders, 1974, 457-472. **Dissertation or Thesis:** Umoh, AO: Correlation of maternal periodontal status with low birth weight and pre-term deliveries, 2011,45 pp.

Tables: Type each table on a separate page. Number the tables consecutively and supply a brief title for each.

Figures and Illustrations: These must be professionally drawn and or photographed. If photographs of persons are used, either the subjects must not be identifiable or their pictures must be accompanied by written permission to use the photograph. Only one per page is permissible.

Legends for Illustration: Type legends of illustrations double-spaced, starting on a separate page, with Arabic numerals corresponding to illustrations. Explain internal scale and identify method of staining in photomicrographs.

Proof-reading: Proofs will be sent to authors and must be read carefully and returned immediately.

The journal must be reimbursed for unusually extensive or late corrections or revisions due to the authors' negligence. Reprints: Authors of original articles will receive one complimentary copy of the issue and a soft copy in pdf format. Please submit soft copies of manuscripts to: The Editor-in-Chief, Nigerian Journal of Periodontal Research, Department of Periodontology and Community Dentistry, University of Ibadan, Nigeria. editornjprnjpr@gmail.com.

Publication fee: Accepted article will attract a publication of 20,000naira only for local authors and 150 dollars for international authors. Additional fees will apply to coloured pictures.