

MANDIBULAR JUVENILE MONOSTOTIC FIBROUS DYSPLASIA, A COMMON LESION WITH UNUSUAL PRESENTATION; CASE REPORT AND LITERATURE REVIEW.

Akinniyi A. T.^{1*}, Adejobi A. F.¹, Ugwu E. I.¹, Aforka E. E.²,
Ajinuowo A. I.¹, Alimi P. M.²

¹Department of Oral and Maxillofacial Surgery, Obafemi Awolowo University Teaching Hospitals Complex, Ile Ife, Osun State, Nigeria.

²Department of Oral Pathology and Oral Medicine, Obafemi Awolowo University Teaching Hospitals Complex, Ile Ife, Osun State, Nigeria

Corresponding Author: Taofeek A. Akinniyi, Department of Oral and Maxillofacial Surgery, Obafemi Awolowo University Teaching Hospitals Complex, Ile Ife, Osun State, Nigeria

Correspondence: drtaofeek@yahoo.com

DOI: <https://dx.doi.org/10.4314/gdj.v22i2.16>

ABSTRACT

BACKGROUND: A diagnostic dilemma exists between lesions with similar clinical and radiographic features. Aim: We present a case of a dilemma between Garre's osteomyelitis and juvenile monostotic fibrous dysplasia (FD) in a 13-year-old boy with toothache from a carious tooth.

CASE REPORT: This case study reports a rare instance of mandibular monostotic fibrous dysplasia in a 13-year-old male patient, which presented alongside a carious tooth (number 37). While fibrous dysplasia commonly affects the maxilla, the aetiology in this case remains unknown. The patient exhibited symptoms of toothache and left-sided mandibular swelling, with more buccal and slight lingual cortical expansion—sparing the lower border—and radiographic features initially suggestive of Garre's osteomyelitis—treatment involved osseous recontouring of the expanded buccal cortex.

RESULT: The histopathologic report of the shaved bones revealed the lesion to be juvenile monostotic fibrous dysplasia (FD). Over a five-year follow-up period, the patient showed no signs of recurrence.

CONCLUSION: To the best of our knowledge, this is the first reported case of a dilemma between Garre's osteomyelitis and monostotic fibrous dysplasia in Nigeria.

KEYWORDS: Juvenile, monostotic, fibrous dysplasia, mandible.

INTRODUCTION

Fibrous dysplasia is a benign developmental fibro-osseous lesion characterized by the replacement of normal bone with excessive proliferation of fibrous tissues, resulting in underdeveloped and inadequately calcified bones.^{1,3} It can affect a single bone (monostotic type) or multiple bones (polyostotic type).^{4,5} When polyostotic, it may present as a component of syndromes such as McCune-Albright syndrome or Jaffe Lichtenstein syndrome.^{3, 6} Fibrous dysplasia can involve craniofacial bones as well as the long bones. When seen in the head and neck region, monostotic fibrous dysplasia more commonly affects the maxilla (occurring twice as often as in the mandible).⁵ Fibrous dysplasia of the mandible is rare and typically presents with downward displacement of the lower border of the mandible and expansion of the lingual and buccal cortices.^{7,8} Fibrous dysplasia accounts for about 2.5-5% of benign bony lesions, with monostotic cases representing approximately 75-80% and polyostotic cases accounting for about 20-25%.^{9,10} In craniofacial bones, about 10% of cases are monostotic while 50-100% are polyostotic.^{11,12}

The aetiology of fibrous dysplasia remains unclear,¹³ though some reports suggest it can be initiated or reactivated during pregnancy, indicating a possible role of female sex hormones.^{2,7} The pathogenesis is mainly due to a mutation in the GNAS1 gene, which encodes the alpha subunit of a transmembrane signaling G protein (G α). This mutation leads to inappropriate overproduction of cyclic adenosine monophosphate (cAMP).^{14,15} The mutation occurs on chromosome 20q13.2-13.3, substituting the amino acid arginine at position 201 with cysteine or histidine in osteoblastic cells.¹⁶⁻¹⁹ This mutation affects the proliferation and differentiation rates of fibroblasts and osteoblasts, which

are involved in the lesion.

There is no gender predilection for monostotic fibrous dysplasia, but polyostotic fibrous dysplasia is more common in females due to the role of female sex hormones.^{2,20} Fibrous dysplasia usually begins in the first and second decades of life and tends to stabilize as patients approach skeletal maturity.²¹ Most fibrous dysplasia cases are asymptomatic. Clinical features depend on the lesion's location and may include facial deformity and asymmetry, nasal congestion or obstruction, malocclusion, paraesthesia, and pain. Associated teeth are usually firm.

Radiographic appearances of fibrous dysplasia can vary and include cystic, sclerotic, and mixed radiolucent-radiopaque patterns.^{2,3,22,23} It typically appears as a poorly defined, fusiform area of dysplastic bone with a smooth, homogeneous ground-glass appearance. In the mandible, there is often an upward displacement of the inferior alveolar canal and loss of lamina dura, which is typical of monostotic fibrous dysplasia by Petrikowski.²⁴

Ossifying fibroma and chronic osteomyelitis (Garre's sclerosing osteomyelitis) are considered primary differential diagnoses for monostotic fibrous dysplasia.^{8,25,26} Diagnosis of monostotic fibrous dysplasia is based on a combination of findings from the clinical features, radiographic presentation, and histopathologic appearance.

Histopathologic evaluation reveals a moderately cellular fibrous connective tissue stroma with irregularly shaped trabeculae of immature bone, resembling the Chinese character for "bone." Fibroblasts exhibit uniform, spindle-shaped nuclei devoid of mitotic figures. Endocrinologists should evaluate Patients with fibrous dysplasia due to the potential association with endocrinopathies.²⁷

Treatment depends on the patient's age, signs, symptoms, and cosmetic concerns. Treatment options include observation, medical treatment, and surgery. Asymptomatic small lesions may only require periodic follow-up, while large lesions causing cosmetic or functional problems may need bone recontouring or, in extreme cases, jaw resection and reconstruction.²⁹ Bisphosphonates have been reported to provide pain relief and slow lesion growth.^{30, 31}

Lifelong follow-up is recommended to monitor disease progression or recurrence after surgery.^{3, 8, 11} We present a case of mandibular fibrous dysplasia in a 13-year-old boy, initially diagnosed as Garre's osteomyelitis. The patient exhibited a slowly progressive, painful swelling on the left side of his mandible, which had persisted for three months. Clinically, the lesion was diagnosed as Garre's sclerosing osteomyelitis. Under general anaesthesia, surgical excision and recontouring of the affected area were performed. The excised tissue was sent for histopathologic examination, which revealed fibrous dysplasia. This unusual presentation posed a diagnostic challenge. The patient has been followed up with no signs of recurrence and demonstrated improved facial aesthetics.

CASE PRESENTATION

A 13-year-old boy was referred to the outpatient clinic of the Department of Oral and Maxillofacial Surgery at Obafemi Awolowo University Teaching Hospitals' Complex (OAUTHC), Ile-Ife, with a three-month history of left-sided, painful mandibular swelling that had gradually increased in size to its present dimensions of 8cm by 5.5cm at presentation. There was no history of trauma to the region; however, there was a history of toothache before the jaw swelling in the same region. He was initially being managed for a dentoalveolar abscess at a private facility, which necessitated placement on antibiotics (Augmentin tablets) and analgesic (paracetamol), which gave him temporary relief, but the swelling persisted. Hence, the reason for the referral. The preoperative clinical photograph is shown in Figure 1.

On examination, he was a healthy-looking young boy with vital signs within the normal range (Blood pressure-100/60mmHg, pulse rate – 88 beats/minute, Respiratory rate – 18 cycles/minutes, Temperature – 37.1°C). There was obvious facial asymmetry, evidenced by a discrete swelling on the left side of the body and an angle of the mandible measuring approximately 8 x 6 cm in size anteroposteriorly and superoinferiorly, respectively. The swelling was bony, hard in consistency globally, and tender on light palpation. The ipsilateral submandibular lymph nodes were palpably tender, enlarged, discrete, and freely mobile. On intraoral assessment, the mouth opening was 3.2, with a maximal interincisal distance. The occlusion was unaffected, with a class 1 molar relationship. The swelling was limited to the buccal cortex with slight obliteration of the buccal vestibule. The swelling spanned from the mesial of tooth 36 to the distal of tooth number 37. The overlying mucosa appeared clinically healthy. Tooth number 37 had a gross class 1 carious lesion involving the pulp with associated grade I mobility. The orthopantomogram was used to assess the lesion radiographically and revealed coronal radiolucency in relation to tooth number 37, which was communicating with the pulp. There was also periapical radiolucency which measures 4mm in relation to the distal root and 2mm in relation to the mesial root, there is a widening of the periodontal ligament space in relation to 37. There is a mixed radiolucent and radiopaque lesion (not a classical

ground glass appearance or orange-peel appearance) that extends from tooth 36 to the horizontal ramus not involving the posterior border, the margin blends imperceptibly with the surrounding bone as shown in the orthopantomograph in Figure 2. The inferior border of the mandible was preserved, this is evident in the intraoperative clinical photograph shown in Figure 3. A provisional diagnosis of Garre's osteomyelitis was made based on the clinical presentation. The patient had surgical recontouring of the affected bone and extraction of tooth number 37 with curettage of the mesial and distal root sockets under general anaesthesia via extraoral access using a submandibular incision, as shown in Figure 4. The post-operative period was uneventful. The healing was satisfactory, and all the symptoms abated.

The shaved bone, Figure 5, was sent for histopathologic examinations. The specimen sent to the pathology lab consisted of six pieces of irregularly shaped, brownish-to-greyish, bony-hard tissue. The dimensions of all the specimens combined were 3.1cm by 2cm by 1.5cm. Microscopic examination of the sections revealed curvilinear trabeculae of woven bone within a fibrous connective tissue stroma, which resembled the so-called "Chinese-letter" appearances typical of fibrous dysplasia. The trabeculae of bone had artefactual clefts around them, termed "peri-trabecular clefts". The stroma did not have significant inflammatory cell infiltrates. The histologic diagnosis was eventually made, based on the patient's age and involvement of the mandible, as juvenile monostotic fibrous dysplasia of the mandible. The photomicrograph is shown in Figure 6. The patient has undergone several reviews over the last five years, with no signs of recurrence and improved facial aesthetics. The follow-up schedule included monthly reviews in the first post-operative year, once every three months for the next two years, and currently, annual follow-ups.



Figure 1: An extraoral preoperative clinical photograph showing discrete swelling of the left side of the mandible.

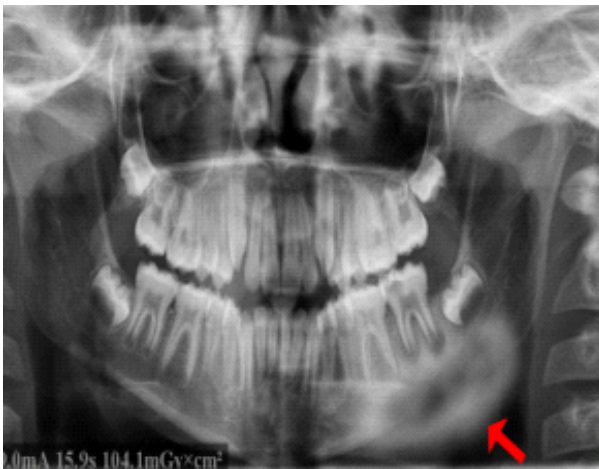


Figure 2: An orthopantomogram of the patient showing a mixed radiolucent and radiopaque lesion of the left side of the mandible in relation to the lower left tooth numbers 6, 7, and unerupted 8. Margin blends with surrounding normal bone.



Figure 3: Intraoperative photograph showing submandibular incision marking to access the lesion.



Figure 4: Intraoperative clinical photograph of the lesion surgically exposed.

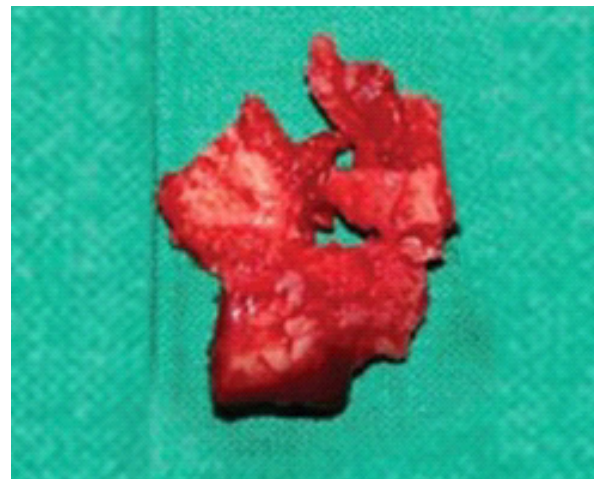


Figure 5: Intraoperative photograph of the shaved bone.

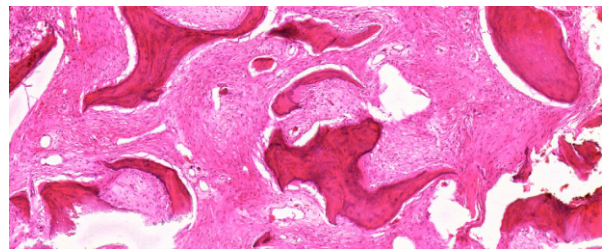


Figure 6: Photomicrograph of the surgical specimen showing curvilinear trabeculae of woven bone described as "Chinese-letter" in a fibrous connective tissue stroma. (H and E, X40)

DISCUSSION

Monostotic FD accounts for 80-85% of all FD cases, with the jaw being one of the most commonly affected sites.⁷ Monostotic FD can be categorized into three types: juvenile, aggressive juvenile, and adult FD. Juvenile FD is usually a painless, slowly progressive swelling diagnosed within the first to second decade of life. The patient in this report was in this age range but presented with toothache and swelling limited to the affected teeth. This may have masked the usually painless expansile swelling of FD. It is also worth noting that the growth in juvenile FD is generally slow, and parents or patients may find it difficult to recall when it was first noticed. It is also possible that the lesion has been present unnoticed until pain drew the patient's attention to it.

Fibrous dysplasia is a slow-growing benign fibrous-osseous lesion that can affect one or more bones in the human skeleton. Typically, FD is characterized by hamartomatous replacement of medullary bone by immature and poorly calcified bone. Its growth usually abates at the end of somatic growth, and such should not be considered a tumour. In contrast, osteomyelitis is regarded as an infection. Osteomyelitis is often accompanied by pain, and when it involves proliferative periostitis, it is referred to as Garre's osteomyelitis.³² This condition is characterized by a distinct gross thickening of the periosteum (the membrane surrounding the bone) due to mild irritation or infection.³² It is a type of chronic osteomyelitis that typically affects children and young adults.^{32, 33} The clinical features can be mistaken for FD, and this may have contributed to our misdiagnosis. FD is usually painless, but it may cause discomfort, especially

when extensive swelling leads to nerve compression. Pain in mandibular monostotic fibrous dysplasia is often attributed to early involvement of the inferior alveolar nerve within the inferior alveolar canal. In the presented case, the pain was due to a carious tooth associated with the lesion.

Monostotic FD does not usually have sex predilection. Fibrous dysplasia tends to affect females more than males. However, our case involved a 13-year-old African boy, which aligns with De Melo et al.²⁹, who reported monostotic fibrous dysplasia in a 15-year-old boy from South America with monostotic fibrous dysplasia.

Most cases of monostotic fibrous dysplasia occur in the maxilla, making mandibular cases, such as ours, relatively rare. The frequent location of mandibular monostotic fibrous dysplasia is the body and angle region. This is similar to the site of occurrence in our case. In the mandible, fibrous dysplasia frequently affects the body and angle region, similar to our case. Although the literature suggests pain in mandibular fibrous dysplasia results from compression of the inferior alveolar nerve, our patient's pain was due to pulpal inflammation following an untreated carious lesion. The patient's diffuse mandibular swelling expanded the lingual and buccal cortices but did not involve the lower border of the mandible. This contrasts with the De Melo et al.²⁹ case, where the lower border was expanded, potentially due to our patient's early presentation prompted by tooth pain.

The initial clinical diagnosis of Garre's osteomyelitis was made on account of the age, location of the lesion, presence of pulpal-involved carious tooth number³⁷ in the region of the swelling, and radiographic presentation. This is similar to the case presented by Kugushev et al.³⁴ case. The reason for our initial misdiagnosis may be due to the presence of carious tooth number³⁷ with periapical radiolucency, as well as the clinical resemblance to mandibular fibrous dysplasia and Garre's osteomyelitis. Functional impairment is less of a problem with monostotic fibrous dysplasia of the mandible, as seen in our case, where masticatory, speech, and swallowing functions were preserved.

The radiographic finding of extensive bone involvement confirmed that the lesion is not a dentoalveolar abscess, raising suspicion of other possible bone lesions. The radiographic controversy between FD and osteomyelitis has been reported in the literature. Gupta and Deepak³⁵ reported a case of chronic osteomyelitis with an unusual radiographic appearance typical of FD. The negative history of trauma or systemic disease in their report added to the dilemma. However, a previous history of tooth extraction in their case support osteomyelitis. In this study, a pre-existing grossly carious lesion informed our diagnosis of Garre's osteomyelitis. In a recent article, Campell et al.³⁶ reported a case of chronic non-bacterial osteomyelitis masquerading as FD.

The radiographic presentation of monostotic fibrous dysplasia is characterized by a mixed radiopaque and radiolucent appearance, with the margin blending into adjacent normal bone. The degree of ossification varies based on the lesion's bony and fibrous components, contributing to different clinical presentations and potential misdiagnosis. This could explain the initial diagnosis of Garre's osteomyelitis in the presented case. This variability aligns with Kugushev et al.'s³⁴ report.

Petrikowski et al.²⁴ attempted to radiographically differentiate between osteogenic sarcoma, osteomyelitis, and FD of the jaws by assessing radiographs of 30 cases of these diseases. Despite using trained observers and strict criteria, differentiating these lesions radiographically

proved difficult, and it was concluded that radiographic characteristics alone were insufficient to differentiate them.

The diagnosis dilemma is worsening because histopathologic analysis often fails to separate these lesions consistently. In a case report on the diagnosis dilemma of juvenile FD and chronic osteomyelitis involving the posterior mandible³⁷, the authors reported findings of radiographic features of osteomyelitis in a 4-year-old girl months after being diagnosed with histopathological diagnosis of FD. The histopathologic appearance of FD is a typical 'Chinese letter' trabecular pattern, as seen in the index case. However, other studies have shown that the histological appearance of osteomyelitis can feature fibrous tissue accompanied by a sparse inflammatory cell infiltrate, new bone formation, and a sequestrum.^{38, 39}

The suspicion of reactive periosteal bone formation due to low-grade infection and repeated hospitalization due to pain and swelling has been attributed to early treatment in Garre's osteomyelitis.⁴⁰ This prompted the need for early intervention in the index case. Therefore, it is important to emphasize that treatment of jaw FD is usually performed in the late teens when bone maturity is assured unless gross functional and/or aesthetic concerns exist. The associated symptoms in the patient precluded treatment delay, and the diagnosis of FD has not been confirmed. Treatment of FD is usually carried out when the jawbones have attained maturity, based on the patient's sex, age, and tumor size, to prevent recurrence.

FD tends to stabilize and essentially stop enlarging when the patient reaches skeletal maturity; some continue to grow, albeit slowly. The intervention performed in this case was for both diagnosis and cosmetics purposes. Cosmetics, functional deformity, and pain are factors that determine the need for treatment.³ Treatment modalities for FD may include paring down, surgical resection, and medical treatment with bisphosphonate therapy. The index patient underwent surgical recontouring with extraction of the involved tooth under general anesthesia via an extraoral approach. Treatment options for monostotic fibrous dysplasia include radical resection with mandibular reconstruction or conservative surgical management involving osseous shaving via intraoral or extraoral approaches. Our choice of treatment was informed by the patient's age and the size of the lesion. The index case was managed conservatively via an extraoral approach for better lesion access, contrasting with De Melo et al.'s²⁹ intraoral approach. Although the extraoral approach can result in facial scars, it provides better access to lesions, especially in the posterior body and angle of the mandible, thereby reducing the risk of recurrence and minimizing the psychological and financial impacts on patients. Studies have reported a recurrence prevalence of 25% to 50% after treatment for FD following shaving.³ We do not record a recurrence 5 years post-intervention. We assumed that our choice of access allowed for thorough recontouring, and the age-limiting nature of the lesion may have been responsible. Repeated interventions increase the risk of malignant transformation, making a "once and for all" mindset crucial for the best chance of cure. Patient selection in choosing the approach is essential. Medical treatment was not considered in this case. The effective use of bisphosphonates has been confirmed mostly in polyostotic FD, especially in craniofacial FD in the maxilla, where resection may result in both cosmetic and functional defects, providing pain relief and improving skeletal strength.

Follow-up is critical for detecting early recurrence. Our patient benefited from lifelong follow-up, similar to De Melo et al.'s protocol.²⁹ Follow-up was facilitated by the patient's proximity to our facility.

This case report highlights the importance of a multidisciplinary approach in the early diagnosis of jaw lesions in children. The potential for increased periosteal bone formation in children could account for the exaggerated bone formation.³⁷ The presence of dental infection raised the suspicion of Garre's osteomyelitis, which could have blurred the clinical presentation of FD in this patient. Therefore, clinicians must be cognizant of the possibility of different lesions with similar presentations occurring at the same time when making a diagnosis, especially in children. Details of history and collaboration with oral pathologists and radiologists are essential in the early diagnosis and treatment of such controversial cases.

CONCLUSION

This case highlights the diagnostic challenges posed by monostotic fibrous dysplasia of the mandible, especially in pediatric patients, where clinical and radiographic features can closely mimic those of other conditions, such as Garre's osteomyelitis. The painful swelling associated with carious lesions in the index case may contribute to our dilemma. A high index of suspicion, supported by thorough imaging and histopathological evaluation, is essential for accurate diagnosis. Early identification and appropriate surgical intervention can lead to favorable outcomes and prevent unnecessary treatments. To the best of our knowledge, this is the first reported case of a dilemma between Garre's osteomyelitis and juvenile monostotic fibrous dysplasia in Nigeria.

REFERENCES

- Hartley I, Zhadina M, Collins MT, Boyce AM. Fibrous dysplasia of bone and McCune–Albright syndrome: a bench to bedside review. *Calcified tissue international*. 2019;104:517-29.
- Eversole R, Su L, ElMofty S. Benign fibro-osseous lesions of the craniofacial complex a review. *Head and neck pathology*. 2008;2:177-202.
- Neville BW, Damm DD, Allen CM, Bouquot J. *Oral and maxillofacial pathology* 2nd ed. St Louis: Saunders. 2002:611-9.
- Delilbaşı Ç, Deniz E, Doğan Ekici I. Monostotic fibrous dysplasia of the mandible. *Oral Health and Dental Management*. 2014.
- Brierley DJ, Chee CKM, Speight PM. A review of paediatric oral and maxillofacial pathology. *International Journal of Paediatric Dentistry*. 2013;23(5):319-29.
- Diaz A, Danon M, Crawford J. McCune-Albright syndrome and disorders due to activating mutations of GNAS1. *Journal of Pediatric Endocrinology and Metabolism*. 2007;20(8):853-80.
- MacDonald-Jankowski D. Fibrous dysplasia: a systematic review. *Dentomaxillofacial Radiology*. 2009;38(4):196-215.
- Chandar V. Bilateral fibrous dysplasia of the mandible in a 7-year-old male patient-A rare case. *Journal of Indian Society of Pedodontics and Preventive Dentistry*. 2010;28(2):126-9.
- Lail RA, Majeed A. Clinical Presentation and Outcome of Fibrous Dysplasia in Patients attending Sahiwal Teaching Hospital, Punjab. *Journal of University College of Medicine and Dentistry*. 2022; 1(2).
- Sainudeen S, Asmi M, Pradeep M. Complex pneumosinus dilatans frontalis and CSOM associated with cranial fibrous dysplasia. *Journal of Radiology and Clinical Imaging*. 2021;4(3):113-24.
- Nadaf A, Radhika M, Paremala K, Srinath N. Monostotic fibrous dysplasia with nonspecific cystic degeneration: A case report and review of literature. *Journal of Oral and Maxillofacial Pathology: JOMFP*. 2013;17(2):274.
- Gupta S, Umesh K, Warad N, Ahmed S. Fibrous dysplasia of maxillary bone: A case report. *Al Ameen J Med Sci*. 2011;4(1):92-7.
- Zhou S-H, Yang W-J, Liu S-W, Li J, Zhang C-Y, Zhu Y, et al. Gene expression profiling of craniofacial fibrous dysplasia reveals ADAMTS2 overexpression as a potential marker. *International Journal of Clinical and Experimental Pathology*. 2014;7(12):8532.
- Walther I, Walther BM, Chen Y, Petersen I. Analysis of GNAS1 mutations in myxoid soft tissue and bone tumors. *Pathology-Research and Practice*. 2014;210(1):1-4.
- Lania A, Mantovani G, Spada A. Genetics of pituitary tumors: focus on G-protein mutations. *Experimental Biology and Medicine*. 2003;228(9):1004-17.
- Mangion J, Edkins S, Goss AN, Stratton MR, Flanagan AM. Familial craniofacial fibrous dysplasia: absence of linkage to GNAS1 and the gene for cherubism. *Journal of medical genetics*. 2000;37(11):e37-e.
- DiCaprio MR, Enneking WF. Fibrous dysplasia: pathophysiology, evaluation, and treatment. *JBJS*. 2005;87(8):1848-64.
- Weinstein LS, Shenker A, Gejman PV, Merino MJ, Friedman E, Spiegel AM. Activating mutations of the stimulatory G protein in the McCune–Albright syndrome. *New England Journal of Medicine*. 1991;325(24):1688-95.
- Idowu B, Al-Adnani M, O'donnell P, Yu L, Odell E, Diss T, et al. A sensitive mutation-specific screening technique for GNAS1 mutations in cases of fibrous dysplasia: the first report of a codon 227 mutation in bone. *Histopathology*. 2007;50(6):691-704.
- Nwizu NN, Aguirre A, Chen F. Diagnostic challenges of benign fibro-osseous lesions and psammomatous meningiomas of the craniofacial region: a comparative review of their clinico-pathological features. *N Am J Med Sci*. 2010;3(1):17-23.

21. Lee J, FitzGibbon E, Chen Y, Kim H, Lustig L, Akintoye SO, et al., editors. Clinical guidelines for the management of craniofacial fibrous dysplasia. Orphanet journal of rare diseases; 2012: Springer.
22. Riddle ND, Bui MM. Fibrous dysplasia. Archives of pathology & laboratory medicine. 2013;137(1):134-8.
23. Lisle D, Monsour P, Maskiell C. Imaging of craniofacial fibrous dysplasia. Journal of medical imaging and radiation oncology. 2008;52(4):325-32.
24. Petrikowski CG, Pharoah MJ, Lee L, Grace MG. Radiographic differentiation of osteogenic sarcoma, osteomyelitis, and fibrous dysplasia of the jaws. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1995;80(6):744-50.
25. Suma R, Vinay C, Shashikanth M, Reddy VS. Garre's sclerosing osteomyelitis. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2007;25(Supplement):S30-S3.
26. Song S, Jeong HJ, Shin H-K, Kim E, Park S-J, Park JH. Sclerosing osteomyelitis of Garré: A confusing clinical diagnosis. Journal of Orthopaedic Surgery. 2019;27(3):2309499019874704.
27. Riminucci M, Liu B, Corsi A, Shenker A, Spiegel AM, Robey PG, et al. The histopathology of fibrous dysplasia of bone in patients with activating mutations of the Gsa gene: site-specific patterns and recurrent histological hallmarks. The Journal of pathology. 1999;187(2):249-58.
28. Orten SS, Hanna E. Fibrous dysplasia: biology and indications for surgery. Operative Techniques in Otolaryngology-Head and Neck Surgery. 1999;10(2):109-12.
29. De Melo WM, Sonoda CK, Hochuli-Vieira E. Monostotic fibrous dysplasia of the mandible. Journal of Craniofacial Surgery. 2012;23(5):e452-e4.
30. Chapurlat R, Legrand MA. Bisphosphonates for the treatment of fibrous dysplasia of bone. Bone. 2021;143:115784.
31. Chapurlat RD. Medical therapy in adults with fibrous dysplasia of bone. Journal of Bone and Mineral Research. 2006;21(S2):P114-P9.
32. Shah KM, Karagir A, Adaki S. Chronic non-suppurative osteomyelitis with proliferative periostitis or Garre's osteomyelitis. Case Reports. 2013;2013:bcr2013009859.
33. Kim DY. Current concepts of craniofacial fibrous dysplasia: pathophysiology and treatment. Archives of Craniofacial Surgery. 2023;24(2):41.
34. Kugushev A, Lopatin A, Gra-chev N, Dagher S, Kotov V. Clinical and Radiological Features of Primary Chronic Osteomyelitis and Fibrous Dysplasia of the Mandible. Journal of Clinical Physiology and Pathology (JISCP). 2024;3(1):25-34.
35. Gupta D, Garg P, Mittal A. Suppl-1, M7: Computed Tomography in Craniofacial Fibrous Dysplasia: A Case Series with Review of Literature and Classification Update. The open dentistry journal. 2017;11:384.
36. Campbell TN, Frizzell B, MacMullan P. Chronic non-bacterial osteomyelitis masquerading as fibrous dysplasia. Modern Rheumatology Case Reports. 2020;4(2):192-5.
37. Zain-Alabdeen E, Abdelfattah A, Kordi O, Al-Sadhan Re. The dilemma of juvenile fibrous dysplasia versus chronic osteomyelitis of the posterior mandible: A case report. Clinical Case Reports. 2022;10(10):e6379.
38. Orpe E, Lee L, Pharoah M. A radiological analysis of chronic sclerosing osteomyelitis of the mandible. Dentomaxillofacial Radiology. 1996;25(3):125-9.
39. Chang Y-C, Shieh Y-S, Lee S-P, Hsia Y-J, Lin C-K, Nieh S, et al. Chronic osteomyelitis with proliferative periostitis in the lower jaw. Journal of dental sciences. 2015;10(4):450-5.
40. Arslan, S.Y., Bal, Z.S., Ozenen, G.G. (2023). Garre's Osteomyelitis of the Jaw in a Child: Report of a Case Treated with Colchicine. The Journal of Pediatric Research, 10(2), 151-155.