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International Journal of Current Research
Vol. 10, Issue, 06, pp.70606-70608, June, 2018

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

ISSN: 0975-833X

RESEARCH ARTICLE

A CASE OF AGGRESSIVE PERIODONTITIS ASSOCIATED WITH ACHONDROPLASIA

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ARTICLE INFO

Article History:
Received 16th March, 2018
Received in revised form
25th April, 2018
Accepted 20th May, 2018
Published online 30th June, 2018

Key words:
Achondroplasia, Dwarfism,
Fibroblast growth Factor Receptor-3 gene,
Periodontal Disease, Periodontitis

ABSTRACT

Achondroplasia is a non-lethal form of chondrodysplasia. It is transmitted as an autosomal dominant trait, with complete penetrance. It results in a disturbance in the endochondral bone formation which causes dwarfism and other associated features. A young female patient suffering from achondroplasia reported with oral manifestations showing features of aggressive periodontitis, hypoplasia of the mid-face, clinical attachment loss, and mobility of teeth. This case report brings to light the possible association of achondroplasia and aggressive periodontitis and the importance of early referral of such cases.

Citation: Ayushya Warang, Swapna Mahale, Dipali Chaudhari and Lavanya Kalekar. 2018. "A Case of Aggressive Periodontitis associated with Achondroplasia", *International Journal of Current Research*, 10, (06), 70606-70608.

INTRODUCTION

Achondroplasia is a disorder of bone growth that causes the most common type of dwarfism. The term achondroplasia was first used by Parrot in 1878. It belongs to a group of disorders called chondrodystrophies or osteochondrodysplasias. It may be inherited as an autosomal dominant trait, which means that if one parent has achondroplasia, the infant has a 50% chance of inheriting the disorder and if both parents have the condition, the infant's chances of being affected increase to 75%. However, most cases appear as spontaneous mutations which mean that an achondroplastic child can have normal parents (Shiang *et al*., 1994). There is limited literature about the association of achondroplasia with periodontitis. This article presents a case of a female patient with achondroplasia and development of aggressive periodontitis.

Case report: A 24-year-old female patient reported to the Department of Periodontics and Oral Implantologyof our hospital, with the chief complaint of many loose and missing teeth. Patient was born after a term uncomplicated pregnancy. Patient reports of late eruption of some permanent teeth. Family history showed that the patient's parents had a consanguious marriage.

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DOI: https://doi.org/10.24941/ijcr.30981.06.2018

The patient also reported of her mother having exfoliation of many teeth due to mobility since a young age.

General examination: On general examination, the patient had short stature (132 cm height), long trunk, short extremities, particularly in the proximal segments (rhizomelic shortening), deformity of legs (Figure 1); short mid-face and maxilla, flattening of nasal bridge, and relatively broad forehead (Figure 2). Third and fourth fingers showed medial inclination known as the 'Trident hand'. Extension and rotation were limited at the elbow.

Intraoral examination (Figure 3). Intraoral examination revealed that the patient's oral hygiene was fair with Oral Hygiene Index (OHI) score of (2.1). The gingiva had mild inflammation and was associated with bleeding on probing. Full mouth probing depths, mobility and furcation involvements were recorded. Grade I mobile were 11, 12, 17, 21, 22, 41 and 15,31 were grade III mobile.

There was grade I furcation involvement in relation to 36. Generalized clinical attachment loss and mucogingival problems were present. The following teeth were missing- 16, 14, 23, 24, 25, 26, 44, 45 and 46. 32 and 42 were congenitally missing. Pathologic migration and traumatic occlusion was present. The patient had bilateral posterior cross-bite.

Figure 1. Short stature, long trunk and deformity of legs

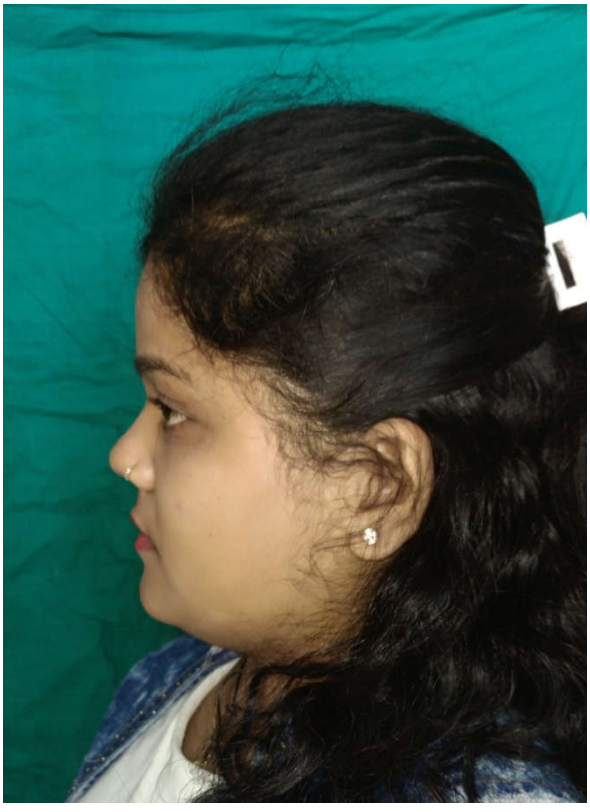

Figure 2. Short midface and flattening of nasal bridge

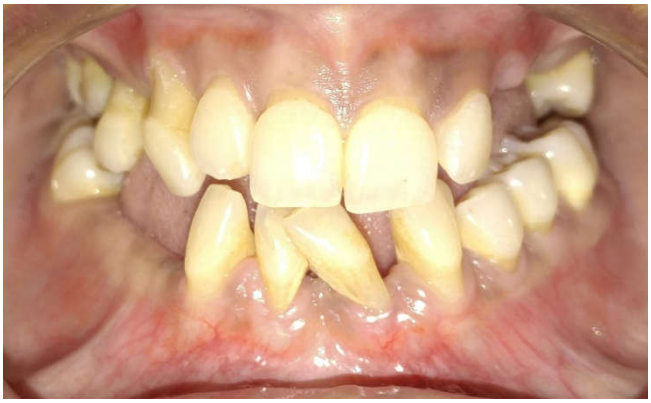

Figure 3. Intra-oral view

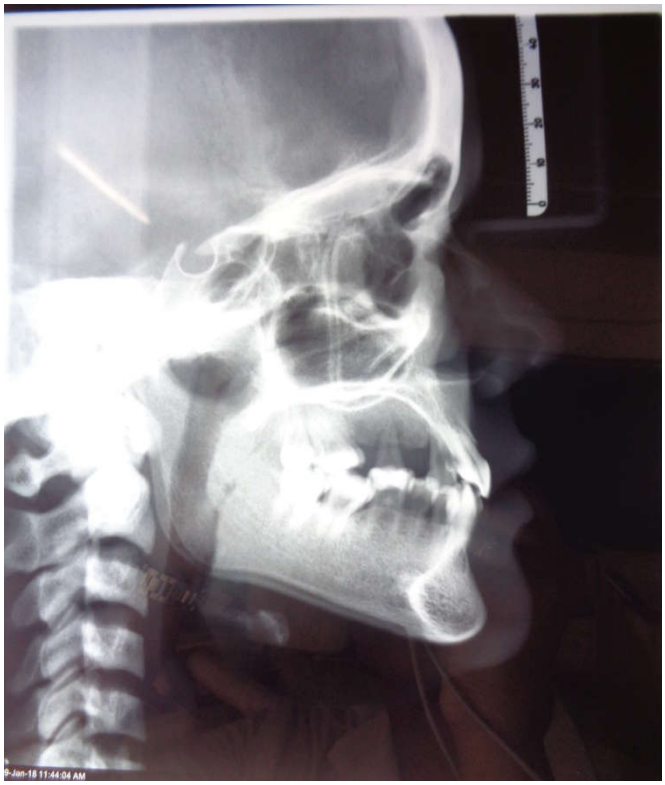

Figure 4. X-ray lateral view

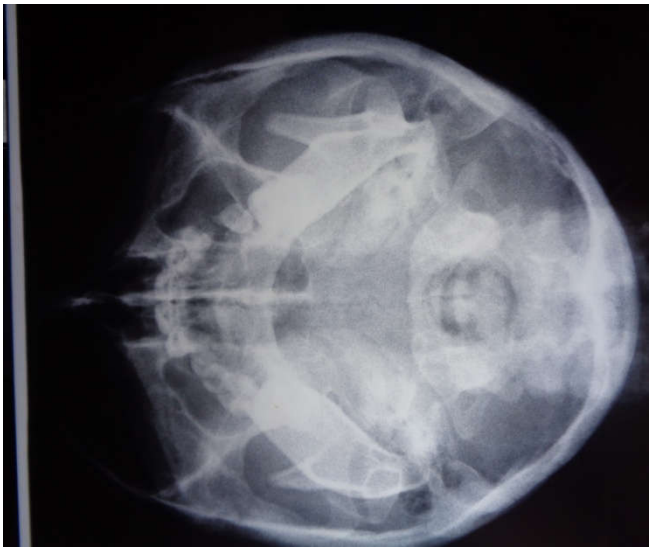

Figure 5. X-ray depicting small foramen magnum

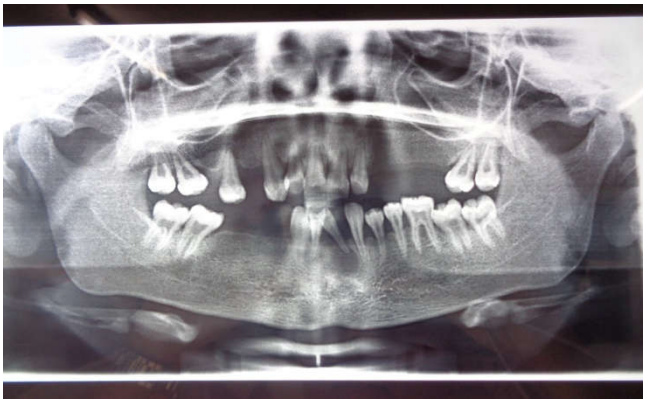

Figure 6. OPG

Radiographic findings: X-ray skull (lateral view) showed relatively broad forehead and flattening of nasal bridge, short maxilla and mid-face, Class III tendency, protrusive chin and short base of skull (Figure 4). X-ray base of skull revealed short foramen magnum, which is a cardinal feature of achondroplasia (Figure 5). An Orthopantamogram (OPG) was done which revealed generalized moderate bone loss associated with all teeth and periapical radiolucency associated with 31(Figure 6).

Investigations: Complete blood count of the patient was normal. Serum alkaline phosphatase was raised (160 U/l). Myeloperoxidase (MPO) staining was done to aid in making the diagnosis of the type of periodontitis (Singh *et al*., 2011). Increased intensity of staining MPO granules in neutrophilswas suggestive of Aggressive periodontitis.

Diagnosis

Based on findings such as early onset of disease, rapid attachment loss and bone destruction, familial aggregation of cases, amount of microbial deposits inconsistent with the severity of disease (Baer 1971) and intense MPO granules staining the periodontal diagnosis of Aggressive periodontitis was established. The general features, radiographic and laboratory findings are indicative of the patient having achondroplasia (Wheeles textbook of orthopaedics).

Treatment: Phase I therapy was started in the patient which included full mouth scaling and root planing with detailed oral hygiene instructions. The patient was prescribed chlorhexidine digluconate mouth rinse (0.2%), twice daily. 31 and 15 were extracted as theywere grade III mobile with hopeless prognosis. Restorative and prosthetic treatment as initiated. The patient was placed on a proper maintenance programme to keep the aggressive periodontitis in check.

DISCUSSION

Achondroplasia is caused by quantitatively defective endochondral bone formation. Most cases (80%) result from a spontaneous mutation of a single gene on chromosome 4. The remaining 20% of cases are transmitted in an autosomal dominant fashion. The genetic defect results in a reduction in functional fibroblast growth factor receptor-3 (FGFR-3) (Shiang *et al*., 2011). FGFR3 gene provides instructions for making a protein called fibroblast growth factor receptor 3, which is a part of fibroblast growth factor receptors. This protein is involved in the development and maintenance of bone and brain tissue. It limits the formation of bone from cartilage (a process called calcification), particularly in the long bones. These proteins also play a role in the regulation of cell growth and division, determination of cell type, formation of blood vessels, wound healing and embryo development. Two specific mutations in the FGFR3 gene are responsible for almost all cases of achondroplasia. Researchers believe that these mutations cause the protein to be overly active, which interferes with skeletal development and results in decreased endochondral ossification, inhibited proliferation of chondrocytes in growth plate cartilage, decreased cellular hypertrophy, and decreased cartilage matrix production[5].Thus, the defective FGFR3 might hamper the growth and function of fibroblasts which are the predominant cells found in the periodontal ligament leading to increased susceptibility of the achondroplastic patients to periodontitis. Several odontostomatologic manifestations of achondroplasia have been previously reported, including skeletal and dental class III malocclusion, a narrow maxilla, macroglossia, and an open bite between the posterior teeth (Celenk *et al*., 2003). Stafne (1950) reported retarded eruption of many permanent teeth in a 30-year-old affected male (Stafne, 1950). There are sparse reports on association of achondroplasia with periodontitis. Chawla K. *et al* (2012) reported a case of a female patient with achondroplasia and early development of periodontal disease[8]. Till date, to the best of our knowledge none of the cases of achondroplasia have been reported to have aggressive periodontitis. This report could pave way for further research of the FGFR3 mutation and its implications on periodontal health and disease.

Conclusion

When the case was reported, periodontal disease had already advanced and the patient had lost many teeth. With this case report, we emphasize the need for early referral of each achondroplastic patient to the periodontist. Regular oral health check-ups and timely intervention in these patients can help in prevention and reducing the morbidity of periodontal disease[8].

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Wheeles textbook of orthopaedics

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