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RESEARCH ARTICLE

PERIODONTAL DISEASES IN CHILDREN – A LITERATURE REVIEW

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ARTICLE INFO	ABSTRACT
Article History: Received 16 th February, 2017 Received in revised form 11 th March, 2017 Accepted 20 th April, 2017 Published online 31 st May, 2017	An attractive smile is the one that has both harmonious correlations between the shape and colors of the teeth and a good proportion between lip and gingival. When it comes to children, it becomes more important because now a day's children are more aware of their surroundings and are very conscious about their appearance and smile. Children and adolescents are subject to a wide variety of gingival infections. In 1996, Albandar <i>et al.</i> assessed the prevalence of gingivitis among large group of adolescents in the United States and found that 82.1% of the participating subjects were having gingivitis. Similar findings of high prevalence of gingivitis among children and adolescents were reported by other studies worldwide. In another study, he assessed the prevalence of early-onset forms of periodontitis among group of US adolescents and reported that 0.6% of the subjects were having juvenile periodontitis at the age of 13–15 years, and 2.75% of the subjects were having chronic periodontitis at the age of 16-17years.
Key words:	
Gingivitis, Periodontitis, Periodontal abscess.	

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INTRODUCTION

Periodontal diseases constitute a group of conditions that are considered nowadays ubiquitous among children, adolescents, and adults. The term "periodontal diseases" includes any inherited or acquired disorders of the tissues that are investing and supporting the teeth (gingiva, cementum, PDL, and alveolar bone) (Gusberti et al., 1983). The periodontium is defined as those tissues supporting and investing the tooth which comprises of root cementum, periodontal ligament, bone lining the tooth socket (alveolar bone), and that part of the gingiva facing the tooth (dentogingival junction) (Gisele et al., 2014). In 1996, Albandar et al. assessed the prevalence of gingivitis among large group of adolescents in the United States and found that 82.1% of the participating subjects were having gingivitis. Similar findings of high prevalence of gingivitis among children and adolescents were reported by other studies worldwide.2 In another study, he assessed the prevalence of early-onset forms of periodontitis among group of US adolescents and reported that 0.6% of the subjects were having juvenile periodontitis at the age of 13-15 years, and 2.75% of the subjects were having chronic periodontitis at the age of 16-17 years. Low prevalence of periodontitis among

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children and adolescents was reported by other studies in different populations (Oh et al., 2002). Many researchers have observed larger amount of plaque and less inflammation in relation to the amount of plaque in children compared to the adults.

Furthermore, experts and clinicians noted that most of the periodontal diseases that affect children and adolescents are reversible and cause little tissue damage compared to the adults (Parimalakilkarni et al., 2011). Periodontal disease may progress to cause exposure of the roots, mobility, and premature loss of the teeth. In 1989, the American Academy of Periodontology set criteria in order to distinguish various forms of periodontal diseases. Those criteria are

- Age at onset,
- Distribution of the sites affected by the disease,
- Presence or absence of the systemic diseases, •
- Rate of the disease progression,
- Response to treatment, and
- Presence or absence of specific host or microbial factors.

The most recent classification of periodontal diseases was introduced in 1999 by the international workshop of periodontology and includes greater variety of periodontal disease categories accepted by American academy of periodontology (Valerie Clerehugh, 2000).

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AGGRESSIVE PERIODONTITIS

Aggressive periodontitis which is also called "juvenile periodontitis" is considered to be prevalent in children and adolescents during circumpubertal period. It is characterized by rapid loss of connective tissue attachment and alveolar bone with familial aggregation. It is caused by both pathogenic microflora and abnormality in host defense mechanisms.

Table 1. Classification of periodontitis by American Academy of Periodontology



ACCORDING TO EUROPEAN SOCIETY OF PERIODONTOLOGY (1993)

- Early onset periodontitis
- Adult periodontitis
- Necrotizing periodontitis

ACCORDING TO INTERNATIONAL WORKSHOP FOR CLASSIFICATION OF PERIODONTAL DISEASES (1999)

- Chronicperiodontitis (Generalized/Localized)
- Localized aggressive periodontitis
- Generalized aggressive periodontitis

Periodontitis as a manifestation of systemic diseases associated with haematological disorders:

- Acquired neutropenia
- Leukemia
- Associated with genetic disorders
- Familial and cyclic neutropenia
- Down's syndrome
- Leucocyte adhesion deficiency syndrome
- Papillon Lefevre syndrome
- Chediak-Higashi syndrome
- Langerhans cell disease
- Glycogen storage disease
- Chronic granulomatous disease
- Infantile granulomatous agranulocytosis
- Cohen syndrome
- Ehlers-Danlos syndrome
- Hypophosphatasia
- Crohn disease
- Marfan disease

- Necrotizing ulcerative periodontitis
- Abscesses of periodontium
- Combined periodontic endodontic lesions

Aggressive periodontitis can be subdivided into localized (LAgP) and generalized form (GAgP) (SigrunZachrisson *et al.*, 1972).

LOCALIZED AGGRESSIVE PERIODONTITIS

Localized aggressive periodontitis patients have interproximal attachment loss on no more than two teeth other than first permanent molars and incisors. At the microbiological level, up to date, no single species of microorganism has been found in all cases of LAgP. However, aggregatibacter (Actinobacillus) species in combination with Bacteroides-like species and Eubacterium species has been isolated from most of LAgP cases. It is well documented that LAgP is associated with a variety of functional defects in neutrophils. A robust serum antibody response to infecting agents is frequently detected (Albandar *et al.*, 1997).

Clinical features

- Usually it occurs around the age of puberty and is localized to first molars and incisors.
- Circumpubertal onset with periodontal damage
- Gingival inflammation, edematous, bleeding, pocketing.
- Rapid attachment loss and bone destruction (Kelly *et al.*, 2004).

GENERALIZED AGGRESSIVE PERIODONTITIS

There is generalized interproximal attachment loss affecting at least three permanent teeth other than the first molars and incisors. Attachment loss occurs in pronounced episodic periods of destruction. The disease is frequently associated with the periodontal pathogens, Actinobacillus actinomy cetemcomitans and Porphyromonasgingivalis and neutrophil function abnormalites. A poor serum antibody response to infecting agents is frequently detected.

Management

In general, treatment methods for the aggressive periodontal diseases should include oral hygiene instruction and reinforcement and evaluation of the patient's plaque control, supra and sub gingival scaling and root planing to remove microbial plaque and calculus, control of other local factors, occlusal therapy, periodontal surgery and maintenance are necessary.

In addition to the parameters for chronic periodontitis, the following should be considered for patients who have aggressive periodontitis:

• A general medical evaluation may determine if systemic disease is present in children and young adults who exhibit severe periodontitis, particularly if aggressive periodontitis appears to be resistant to therapy. Consultation with the patient's physician may be indicated to coordinate medical care in conjunction with periodontal therapy. Modification of environmental risk factors should be considered.

- Initial periodontal therapy alone is often ineffective. However, in the early stages of disease, lesions may be treated with adjunctive antimicrobial therapy combined with scaling and root planing with or without surgical therapy. Microbiological identification and antibiotic sensitivity testing may be considered. In very young patients, the use of tetracycline may be contraindicated due to the possibility of staining of teeth. Alternative antimicrobial agents or delivery systems may be considered.
- The long-term outcome may depend upon patient compliance and delivery of periodontal maintenance at appropriate intervals, as determined by the clinician. If primary teeth are affected, eruption of permanent teeth should be monitored to detect possible attachment loss.
- Due to the potential familial nature of aggressive diseases, evaluation and counselling of family members may be indicated (Zacy Carola Nualart Grollmus *et al.*, 2007).

CHRONIC PERIODONTITIS

Chronic periodontitis is defined as inflammation of the gingiva and the adjacent attachment apparatus. The disease is characterized by loss of clinical attachment due to destruction of the periodontal ligament and loss of the adjacent supporting bone. Although this form of periodontitis is considered more prevalent in adults, it can be seen occasionally in children and adolescents. Comparing to aggressive periodontitis, chronic periodontitis is characterized by a low to moderate rate of progression that may include episodes of rapid destruction. It is subdivided according to the percentage of the involved sites into localized (<30%) and generalized (>30%) (Vivek Singh Chauhan, 2012).

Clinical Features

It includes combinations of the following signs and symptoms: edema, erythema, gingival bleeding upon probing, and suppuration. Chronic periodontitis with advanced loss of periodontal support is characterized by a loss of greater than one third of the supporting periodontal tissues. Loss of clinical attachment, in the furcation, if present, will exceed Class I (incipient). Advanced destruction is generally characterized by periodontal probing depths greater than 6 mm with attachment loss greater than 4 mm. Radiographic evidence of bone loss is apparent. Increased tooth mobility may be present. Chronic periodontitis with advanced loss of periodontal supporting tissues may be localized, involving one area of a tooth's attachment, or more generalized, involving several teeth or the entire dentition. A patient may simultaneously have areas of chronic periodontitis with slight, moderate, and advanced destruction. Management

Initial Therapy

• Contributing systemic risk factors may affect treatment and therapeutic outcomes for chronic periodontitis. These may include diabetes, certain periodontal bacteria, aging, gender, genetic predisposition, systemic diseases and conditions (immunosuppression), stress, nutrition, pregnancy, HIV infection, substance abuse, and medications. Elimination, alteration, or control of risk factors which may contribute to adult periodontitis should be attempted. Consultation with the patient's physician may be indicated.

- Instruction, reinforcement, and evaluation of the patient's plaque control should be performed.
- Supra- and sub gingival scaling and root planning should be performed to remove microbial plaque and calculus.
- Antimicrobial agents or devices may be used as adjuncts. Sub gingival microbial samples may be collected from selected sites for analysis, possibly including antibiotic-sensitivity testing.
- Local factors contributing to chronic periodontitis should be eliminated or controlled. To accomplish this, the following procedures may be considered:
- Removal or reshaping of restorative overhangs and over-contoured crowns;
- Correction of ill-fitting prosthetic appliances
- Restoration of carious lesions
- Odontoplasty
- Tooth movement;
- Restoration of open contacts which have resulted in food impaction;
- Treatment of occlusal trauma
- Extraction of hopeless teeth.

For reasons of health, lack of effectiveness or non-compliance with plaque control, patient desires, or therapist's decision, appropriate treatment to control the disease may be deferred or declined.

Compromised Therapy

In certain cases, because of the severity and extent of disease and the age and health of the patient, treatment that is not intended to attain optimal results may be indicated. In these cases, initial therapy may become the end point. This should include timely periodontal maintenance.

Periodontal Surgery

In patients with chronic periodontitis with advanced loss of periodontal support, periodontal surgery should be considered. A variety of surgical treatment modalities may be appropriate in managing the patient.

- Gingival augmentation therapy
- Regenerative therapy (Barr, 1995).

PERIODONTAL ABSCESS

A localized purulent infection within the tissues adjacent to the periodontal pocket that may lead to the destruction of periodontal ligament and alveolar bone.

Clinical features

It includes combinations of the following signs and symptoms: a smooth, shiny swelling of the gingiva, pain, with the area of swelling tender to touch, a purulent exudate, and/or increase in probing depth.

The tooth may be sensitive to percussion and may be mobile. Rapid loss of periodontal attachment may occur. A periodontal abscess may be associated with endodontic pathosis.

Management

It includes irrigation and debridement of the necrotic areas and tooth surfaces, oral hygiene instructions and the use of oral rinses, pain control, and management of systemic manifestations, including appropriate antibiotic therapy, as necessary. Patient counselling should include instruction on proper nutrition, oral care, and appropriate fluid intake. A comprehensive periodontal evaluation should follow resolution of the acute condition (Kubar *et al.*, 2005).

NECROTIZING PERIODONTAL DISEASES

Necrotizing periodontal diseases occur with varying but low frequency in North American and European children. It is seen with greater frequency in certain populations of children and adolescents from developing areas of Africa, Asia, and South America. It shows the presence of interproximal necrosis and ulceration and the rapid onset of gingival pain and become febrile. Necrotizing ulcerative periodontitis sites have high levels of spirochetes and P. intermedia and invasion of the tissues by spirochetes has been shown to occur. Factors that predispose include viral infections (including HIV), malnutrition, emotional stress, lack of sleep, and a variety of systemic diseases (Armitage, 1999).

Clinical Features

It includes combinations of the following signs and symptoms: necrosis and ulceration of the tips of the interdental papillae or gingival margin; and painful, bright red marginal gingiva which bleed on slight manipulation. The mouth may have a malodor, poor nutrition and systemic manifestations may be present.

Management

Treatment involves mechanical debridement, oral hygiene instructions, and careful follow-up. Debridement with ultrasonics has been shown to be particularly effective and results in a rapid decrease in symptoms. If the patient is febrile, antibiotics may be an important adjunct to therapy. Metronidazole and penicillin have been suggested as drugs of choice (Genco *et al.*, 1988).

COMBINED PERIODONTAL AND ENDODONTIC LESIONS

Combined periodontal and endodontic lesions are localized, circumscribed areas of infection originating in the periodontal and pulpal tissues. The infections may arise primarily from pulpal inflammatory diseases which expresses itself through the periodontal ligament or the alveolar bone to the oral cavity. They also may arise primarily from a periodontal pocket communicating through accessory canals of the tooth and / or apical communication and secondarily infect the pulp. In addition, they may arise as a sequel of a fractured tooth.

Clinical features

It includes the combinations of the following signs and symptoms: smooth, shiny swelling of the gingiva or mucosa, pain, with the area of swelling tender to touch and a purulent exudate. The tooth may be sensitive to percussion and mobile. A fistulous track may be present. Rapid loss of the periodontal attachment and peri radicular tissues may occur. Facial swelling and cellulitis may be present. 7

Management

Establish drainage by debriding the pocket and by incising the abscess. Other treatments may include endodontic therapy, irrigation of the pocket, limited occlusal adjustment, the administration of antimicrobials, and management of patient discomfort. A surgical procedure for access, debridement may be considered. In some circumstances, an endodontic consultation may be required. In other circumstances, extraction of the tooth may be necessary. In any case, a comprehensive periodontal and endodontic examination should follow resolution of the acute condition (Johnson, 1986).

PERIODONTITIS ASSOCIATED WITH SYSTEMIC CONDITIONS

It is a group of rare diseases that predispose the affected individual to highly destructive periodontal infections. These diseases are characterized by defective functions of neutrophils and other immune cells (Zambon, 1985).

Patient Evaluation

- A comprehensive periodontal evaluation should be performed.
- Conditions which are suggestive of systemic disorders should be identified.
- Physical disabilities.
- Signs or symptoms of xerostomia, mucocutaneous lesions, gingival overgrowth, excessive gingival haemorrhage, or other indicators of undetected or poorly-controlled systemic disease.
- Therapeutic drug use,
- Signs or symptoms of smoking, chemical dependency, and other addictive habits.
- History of recent or chronic diseases,
- Evidence of psychological/emotional factors,
- History of familial systemic disease.
- Request laboratory tests as appropriate
- Referral to or consultation with other health care providers should be made and documented when warranted (Leggott *et al.*, 1987).

INSULIN DEPENDENT DIABETES MELLITUS (IDDM)

Decrease in insulin secretion or availability caused by genetic defect in pancreatic beta-cells.A vast majority of studies have concluded that the incidence of chronic gingivitis in patients with type 1 diabetes is significantly higher than that in the healthy population and it increases with in a age group of diabetic children aged 5-9 years. In a Swiss clinical trial on experimental gingivitis induced by refraining from oral hygiene for three weeks, there were no differences in the plaque index scores or in the composition of bacterial plaque between the type 1 diabetics and healthy controls, but the diabetics responded to plaque irritation by an earlier developed and more severe gingival inflammation, which corresponded to a significantly higher levels of some inflammatory biomarkers in crevicular fluid. The onset and progression of periodontitis in diabetic patients are probably induced by diabetic microangiopathy, impaired immune response and a lower resistance to infections, different oral microflora, and disorders in collagen metabolism.

Clinical features

Gingivitis, attachment loss, and bone loss are more prevalent in poorly controlled cases. Reduced PMNs functions (chemotaxis, adhesion and phagocytosis). Decreased collagen synthesis and increased collagenase activity, delayed wound healing, and increased susceptibility to infections may be seen (Genco *et al.*, 1988).

Management

- Identification of signs and symptoms of undiagnosed or poorly controlled diabetes mellitus.
- Consultation with the patient's physician as necessary.
- Consideration of diagnosis and duration of diabetes, level of glycaemic control, and medications and treatment history.
- Recommendation that diabetic patients take medication as prescribed and maintain an appropriate diet on the day of periodontal therapy.
- Consideration of adjunctive systemic antibiotics for periodontal procedures if the diabetes is poorly controlled.
- Attempts to reduce stress or anxiety.
- Preparation to diagnose and manage medical emergencies associated with diabetes.16

HIV/AIDS: Majority of the parents were HIV-positive highly suggestive that mother-to-child transmission was the dominant mode of transmission of the virus to infected children. Mode of transmission were child circumcision and blood transfusion. It develops as a result of infection with human immunodeficiency virus. The progression of the disease is faster and more severe in children due to their developmental stage and to the immaturity of their immune system.

Clinical features

- Linear gingival erythema
- Acute necrotizing ulcerative gingivitis
- Acute necrotizing periodontitis1

Management

- Consultation and coordination of treatment with patient's physician as necessary.
- Controlling associated mucosal diseases and acute periodontal infections.
- Administration of systemic or local medications (for example, antibiotics) only if indicated and administered in a manner that avoids opportunistic infections and adverse drug interactions (Valerie Clerehugh and AradhnaTugnait, 2000).

LEUKOCYTE ADHESION DEFICIENCY (LAD): It is inherited as autosomal recessive condition in which glycoprotein adhesion in leukocyte molecules is severely reduced.

Clinical features

- Poor immune response to bacterial infections.
- Acute inflammation and rapid bone loss.

- Recurrent bacterial infections.
- Poor wound healing.
- Associated with prepubertal periodontitis (Newell *et al.*, 2005).

LEUKEMIA

It is a malignant disease caused by the proliferation of the white blood cells (WBC) forming tissues, especially those in the bone marrow. It may be acute or chronic and can affect any of the WBC – granulocytes (myeloid), lymphocytes, or monocytes. Acute types of leukemia were frequent in people under 20 years of age. Acute lymphoblastic leukemia mainly occurs in children under 10 years. Factors that have been implicated to be of etiologic significance are radiation injury, chemical injury, genetic factors – Down's syndrome, immune deficiency and viral infections.

Clinical features

Gingiva appears as swollen, glazed, and spongy tissue which is red-deep purple in appearance with gingival bleeding.

- Enlargement may appear as a diffuse enlargement of the gingival mucosa, an oversized extension of the marginal gingiva, or a discrete tumor like interproximal mass. It is moderately firm in consistency, but there is a tendency toward friability and hemorrhage, occurring either spontaneously or on slight irritation15.
- Lethargy, malaise, sore throat, fever, skin infections that fail to heal, purpura, cervical lymphadenopathy, spleenomegaly, hepatomegaly and petechiae.

Management

- Coordination of treatment with the patient's physician.
- Minimization of sites of periodontal infection by means of appropriate periodontal therapy prior to the treatment of leukaemia and transplantation.
- Avoidance of elective periodontal therapy during periods of exacerbation of the malignancy or during active phases of chemotherapy.
- Consideration of antimicrobial therapy for emergency periodontal treatment when granulocyte counts are low.
- Monitoring for evidence of host-versus-graft disease and of drug-induced gingival overgrowth following bone marrow transplantation.
- Periodontal therapy, including surgery, for patients with stable, chronic leukemia.19

Neutropenia

The number of PMNs in peripheral blood is below 1000/mm3 in infants and 1500/mm3 in children. Gingiva appears as swollen, glazed, and spongy tissue which is red-deep purple in appearance with gingival bleeding. Enlargement may appear as a diffuse enlargement of the gingival mucosa, an oversized extension of the marginal gingiva, or a discrete tumor like interproximal mass. It is moderately firm in consistency, but there is a tendency toward friability and hemorrhage, occurring either spontaneously or on slight irritation.

Clinical features

• Lethargy, malaise, sore throat, fever, skin infections that fail to heal, purpura, cervical lymphadenopathy, spleenomegaly, hepatomegaly and petechiae.

• Severe gingivitis, gingival ulcerations, and periodontitis. Recurrent infections such as otitis media and upper respiratory infections (Gusberti *et al.*, 1983).

Management

It includes scaling along with periodontal therapy and antimicrobial therapy which treats the periodontal disease related to this condition (ZacyCarolaNualartGrollmus, 2007

ACRODYNIA

Acrodynia is caused by mercurial toxicity reaction (mercury poisoning or idiosyncrasy to mercury).

Clinical features

- Gingival and mucosal hyperplasia.
- Alveolar bone loss.
- Early loss of primary teeth.
- Profuse salivation and sweating (Gaurav Gupta *et al.*, 2012).

HISTIOCYTOSIS

Disturbance of the reticulo endothelial system includes defects in PMNs and monocytes. Gingiva appears as swollen, glazed, and spongy tissue which is red-deep purple in appearance with gingival bleeding.

- Enlargement may appear as a diffuse enlargement of the gingival mucosa, an oversized extension of the marginal gingiva, or a discrete tumor like interproximal mass. It is moderately firm in consistency, but there is a tendency toward friability and hemorrhage, occurring either spontaneously or on slight irritation.
- Lethargy, malaise, sore throat, fever, skin infections that fail to heal, purpura, cervical lymphadenopathy, spleenomegaly, hepatomegaly and petechiae may occur.

Clinical features

• Punched out necrotic ulcers, with granulation tissues, tissue necrosis with marked bone loss. Increased susceptibility to bacterial infections.

Management

Biopsy of granulation tissue associated with deep lesions.

HYPOPHOSPHATASIA

Genetic disorder characterized by low level of serum alkaline phosphatase and excretion of phosphor ethanolamine in urine.

Clinical features

- Premature loss of deciduous teeth and skeletal deformity.
- Defective bone/tooth mineralization.
- Cementum hypoplasia/aplasia.

Management

Chemical and mechanical control of bacterial plaque.

CHEDIAK-HIGASHI SYNDROME

- It is an autosomal recessive disorder characterized by impaired function of cytoplasmic microtubules in PMNs.
- It has frequently been linked with severe periodontitis.
- It is a rare autosomal recessive immunodeficiency disorder characterized by large lysosomal granules in granulocytes, partial oculocutaneous infections and intermittent febrile episodes.

Clinical features

- Recurrent infections.
- Severe gingivitis and periodontitis.
- Intraoral ulcerations.
- Mobility of teeth.

Management

- Functional defects in Chediak- Higashi syndrome leukocytes are corrected by ascorbic acid.
- Other treatments consisted of management regimens such as vincristine - corticosteriods, etoposide – corticosteroids intrathecal methotrexate and high dose of intravenous globulin, including a transient remission.
- Rigorous bacterial plaque control.

PAPILLON-LEFEVRE SYNDROME

It is an autosomal recessive condition associated with impaired neutrophil functions and characterized by palmoplantar keratosis and severe early onset periodontitis. Skin lesions can also be found on the knees and elbows. It has been classified as a type IV ectodermal dysplasia. A variety of other clinical findings including cranial calcifications and an increased susceptibility to infections have been reported, mutations of the cathepsin C gene are responsible for papillon-Lefevre syndrome, and is also due to mutations of the cathepsin C gene.

Clinical features

- Palmoplantar hyperkeratosis.
- Early-onset periodontitis affecting both primary dentition and permanent dentition.
- Swollen gingival, migration and mobility of teeth, periodontal pockets, fetor oris and exfoliation of teeth.

Management

The treatment of the periodontal component of Papillon-Lefevre Syndrome is very difficult. Many authors suggested the importance of the professional dental care and the use of prophylactic antibiotics, but usually this procedure is not enough. It has been shown that acitretin therapy is a safe and effective treatment in paediatric cases of inherited keratinization disorders. Oral retinoids plus antibiotics have been reported with good response. The use of oral retinoids for prolonged period is suggested. It seems a beneficial drug to prevent loss of permanent teeth in children. Antibiotic prophylaxis is advised (Guideline of periodontal therapy, 2003).

DOWN SYNDROME

Trisomy 21, mongolism, and autosomal chromosomal anomaly associated with impaired PMNs functions, connective tissue disorders, and gingival hyper innervation. It is a frequently occurring genetic disorder with a frequency of approximately 1 in 700 live births. It is characterized by a typical orofacial appearance including flattened facial profile with epicanthic folds, expanded bridge of the nose, opened nose, and a protruding, fissured tongue. Growth of the middle face is stunted while the mandible is oversized with a protrusive dentition. Affected children also demonstrate a myriad of other difficulties including growth retardation, mental retardation, mental deficiency, muscle hypotonia, joint hyperflexibility and congenital heart disease. The affected individuals frequently manifest aggressive form of periodontal disease affecting both primary and permanent dentition. The periodontal destruction is characterised by the formation of deep periodontal pockets associated with heavy plaque accumulation and intense gingival inflammation.

Clinicalfeatures

- Gingivitis and periodontitis especially in lower anteriors.
- Enamel hypoplasia.
- Microdontia.
- Macroglossia.
- Fissured tongue.

Management

Rigorous bacterial plaque control (Treatment of Plaqueinduced Gingivitis, 2004).

EHLERS-DANLOS SYNDROME

Ehlers-Danlos syndrome (EDS) is a clinically and genetically heterogeneous connective-tissue disorder characterized by articular hypermobility, skin hyperextensibility, and tissue fragility collagen disorder affecting joints and skin. Type VIII is autosomal dominant and has periodontal implications. Pathogenic mutations in the genes encoding collagen types I, III, and V, and the collagen-processing enzymes hydroxylase and collagen N-peptidase have been found to underlie several EDS variants which suggests that EDS is a disorder of fibrillar collagen metabolism More recently, mutations in Tenascin-X, a large extracellular-matrix protein of unknown function, were reported in patients with EDS, indicating that defects outside the classic collagen family have potential to cause EDS phenotypes. The primary feature that discriminates EDS-VIII from other forms of EDS is severe early-onset periodontitis. Periodontitis is characterized by irreversible destruction of the periodontal tissues (periodontal ligament, alveolar bone, and connective tissue) and has many heterogeneous causes. It is estimated to occur in 15%-20% of the adult population and 0.5%–3% of children.

Clinical features

- Aggressive early-onset periodontitis.
- Prolonged bleeding.
- Easily traumatized mucosa.

Management

Chemical and mechanical control of bacterial plaque (Anuj Singh Parihar, 2015).

OCCLUSAL TRAUMA ASSOCIATED WITH PERIODONTITIS

Injury to the periodontium may result from occlusal forces in excess of the reparative or adaptive capacity of the attachment apparatus. Occlusal traumatism affects the supporting structures of the tooth or teeth. The lesion of trauma from occlusion may occur in conjunction with, or independent of, inflammatory periodontal diseases. Although trauma from occlusion and inflammatory periodontal disease may occur concurrently, each condition may be treated separately. The treatment goals and endpoints for each condition may be independent of each other. Occlusal therapy is generally addressed following, or in conjunction with, procedures to resolve the inflammatory lesions.

Clinical Features

Positive diagnosis of occlusal traumatism can be made if some of the signs and symptoms of an injury can be located in some part of the masticatory system. The following represent clinical features of such an injury, but are not pathognomonic for the condition:

- Tooth mobility: Increasing displacement may be of greater concern since a stable pattern of mobility may indicate adaptation.
- Tooth migration.
- Tooth pain or discomfort on chewing or percussion.
- Radiographic changes such as widening of the periodontal ligament space, disruption of the lamina dura, radiolucency in the furcation or at the apex of a tooth that is vital, or root resorption. Just as with mobility, stable radiographic findings may indicate adaptation.
- Tenderness of the muscles of mastication or other signs or symptoms of temporomandibular dysfunction.
- Presence of wear facets beyond expected levels for the patient's age and diet consistency.
- Chipped enamel or crown or root fractures.
- Fremitus.

These clinical signs and symptoms may be indicative of other pathoses. Therefore, differential diagnoses may be established. Use of supplementary diagnostic procedures may be helpful, e.g., pulp vitality tests and evaluation of parafunctional habits.

Management

Treatment of the symptoms of occlusal traumatism is appropriate during any phase of periodontal therapy. Except in the case of acute conditions, treatment is usually first addressed during initial therapy following efforts to reduce or minimize the inflammatory lesion Evaluation of occlusal symptoms should continue throughout the course of therapy.

Treatment may need to be repeated or revised. Efforts are directed toward elimination or minimization of excessive force or stress placed on a tooth or teeth. Occlusal therapy may be accomplished through several different approaches. The choice depends on several factors, such as the characteristics of the forces, the underlying cause of these forces, the amount of periodontal support of the remaining teeth, and the function of the remaining dentition.

Treatment considerations for chronic periodontitis patient with occlusal traumatism may include one or more of the following:

- Occlusal adjustment;
- Management of parafunctional habits
- Temporary, provisional, or long-term stabilization of mobile teeth with removable or fixed appliances;
- Orthodontic tooth movement;
- Occlusal reconstruction;
- Extraction of selected teeth.

In the absence of clinical signs or symptoms, occlusal adjustment to obtain a conceptualized ideal occlusal pattern provides little or no benefit to the patient. Therefore, prophylactic occlusal adjustment appears to be contraindicated. Occlusal relationships may be evaluated as part of periodontal maintenance (PunitVaibav Patel *et al.*, 2011).

REFERENCES

- Albandar, J. M., Brown,L. J. and Loe, H. 1997. "Clinical features of early-onset periodontitis," The Journal of the American Dental Association, vol. 128, no. 10, pp. 1393– 1399.
- Anuj Singh Parihar, VartikaKatoch Furcation involvement & its management: A review. Journal of Advanced Medical and Dental Sciences Research Vol. 3 Issue 1 January-March 2015.
- Armitage, G. 1999. Development of a classification system for periodontal diseases and conditions. Ann Perodontol. 4:1-6.
- Barr, C. E. 1995. "Periodontal problems related to HIV-1 infection," Advances in Dental Research, vol. 9, no. 2, pp. 147–151.
- Gaurav Gupta, Sunil Kumar M.V, Harikesh Rao, PoojaGarg, Rajesh Kumar, Alok Sharma, HarleenSachdeva. Astringents in dentistry: a review. Asian Journal of Pharmaceutical and Health Sciences. Jul-Sep 2012 Vol-2 Issue-3
- Genco, R.J., Zambon, J.J., Christersson, L.A.1988. The origin of periodontal infections. *Adv Dent Res.*,2:245-59.
- Gisele, M.C. Fabri, Cynthia savioli, jose T. Siqueira, Lucia M. Campos. 2014. Periodontal disease in pediatric rheumatic diseases. vol. 54 no. 4.
- Guideline of periodontal therapy. American academy of pediatric dentistry june 2003.
- Gusberti, F.A., Syed, S.A., Bacoon, G., Grossman N., Loesche, W. J. 1983. Puberty gingivitis in insulindependent diabetic children. 1. Cross sectional observations. *J of periodontology* December; 54(12): 714-720.
- Johnson, B., Engel, D. 1986. ANUG. A review of diagnosis, etiology and treatment. *J periodontal*. 57:141-50.

- Kelly, K. Hilgers, D.D.S. John W. Dean, D.D.S. 2004. PhD Gregory P. Mathieu. Localized Aggressive Periodontitis in a Six-year-old; A Case Report. Pediatric Dentistry – 26:4.
- Kubar, I. Saygun, A. 2005. Ozdemir, M. Yapar, and J. Slots, "Real-time polymerase chain reaction quantification of human cytomegalovirus and Epstein-Barr virus in periodontal pockets and the adjacent gingiva of periodontitis lesions," Journal of Periodontal Research, vol. 40, no. 2, pp. 97–104.
- Leggott, P.P., Robertson, P.P., Greenspan, D., Wara, D.W., Greenspan, J.S. 1987. Oral manifestations of primary and acquired immunodeficiency disease in children. *Pediaric Dent.*,9:98-104.
- Newell W Johnson Dr Bruce L Pihlstrom, DDS, Bryan S Michalowicz periodontal disease. The lancet. Volume 366, Issue 9499, 19–25 November 2005, Pages 1809–1820
- Oh, J.J., Eber, R., Wang, H.L. 2002. Periodontal diseases in child and adolescents. *J ClinPeriodontol*. 29:400-10.
- Parimalakilkarni, Neeraj Agarwal, Sanjeev Tyagi, HalaswamyKambalimath. A rare case of gingival fibromatosis associated with hypertrichosis and dysmorphic face. The journal of clinical paediatric dentistry vol 35 no.3 2011.
- PunitVaibav Patel, Sheelakumar G, Amirt Patel. Periodontal Abscess: A Review. Journal of Clinical and Diagnostic Research. 2011 Apr, Vol-5(2):404-409
- Shoji Tanaka, Mikako Yoshida, Yukio Murakami, TakakoOgiwara, Masao Shoji, Satoko Kobayashi, Sigeru Watanabe, Mamoru Machino, and Seiichiro Fujisawa (2008) The Relationship of Prevotellaintermedia, Prevotellanigrescens and Prevotellamelaninogenica in the Supragingival Plaque of Children, Caries and Oral Malodor. Journal of Clinical Pediatric Dentistry: April 2008, Vol. 32, No. 3, pp. 195-200.
- SigrunZachrisson, Bjorn U. 1972. Zachrisson. Gingival condition associated with orthodontic treatment. Angle Orthodont. 42(1): 26-34.
- Treatment of Plaque-induced Gingivitis, Chronic Periodontitis, and Other Clinical Conditions. American academy of perdiatric dentistry 2004.
- Valerie Clerehugh and AradhnaTugnait Diagnosis and management of periodontal dieases in children and adolescents. Periodontology 2000 vol 26 2001, 146-168.
- Vivek Singh Chauhan, Rashmi Singh Chauhan, NihalDevkar, AkshayVibhute, Shobha. Gingival and Periodontal Diseases in Children and Adolescents. Journal of Dental & Allied Sciences 2012;1(1):26-29
- ZacyCarolaNualartGrollmus 1, Mariana Carolina Morales Chávez 2, Francisco Javier Silvestre Donat. Periodontal disease associated to systemic genetic disorders. Med Oral Patol Oral Cir Bucal 2007;12:E211-5.
- ZacyCarolaNualartGrollmus, Mariana Carolina Morales Chávez, 2007. Francisco Javier Silvestre Donat. Periodontal disease associated to systemic genetic disorders. Med Oral Patol Oral Cir Bucal., 12:E211-5.
- Zambon, J. J. 1985. "Actinobacillusactinomycetemcomitans in human periodontal disease," Journal of Clinical Periodontology, vol. 12, no. 1, pp. 1–20.
