ROLE OF NUTRITION AND HORMONE IN ORTHODONTICS.

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Abstract- In the field of orthodontics, main area of interest is orthodontic tooth movement to achieve pleasant esthetics with functional efficiency and stable results. For that optimum orthodontic force is applied on tooth which is further transmitted to the adjacent investing tissues leading to some mechanical, chemical and cellular events in these tissues, resulting in desirable tooth movement. nutrition and hormone can reach these tissues though blood circulation and can change the events taking place in these tissues, thereby affecting orthodontic tooth movement. Their effect can be either inhibitory or accelerating. This review highlights the different views by various authors regarding the effects of nutrition and hormones on orthodontic treatment.

Keywords: nutrition, hormone, orthodontic tooth movement.

Introduction

Nutrition according to Council of food and nutrition of the American Medical Association is, "The science of food, the nutrients and other substances there in, their action, interaction and balance in relation to health and diseases and the processes by which the organism ingest, digest, absorbs, transports, utilizes and excretes food substances. The macronutrients include carbohydrate, fat, protein, and water. The micronutrients are minerals and vitamins.

Nutrition plays an important role, especially key vitamins D3 and K2 which are necessary for proper dentofacial development and food consistency influence on crowding and dental arches narrowing. Changes in our dentition and facial appearance are caused by changing our diet from primitive hunter gatherer to a more modern industrialized agriculture. Nutrition and its impact on epigenetically- mediated mechanisms continuously shape our phenotype which impacts overall health and can reverse the path for overall health and facial bone development.

Orthodontics and nutrition both play a role in following nature's path to re-establishing facial balance and dental arches proportions to accommodate all teeth.

Nutritional deficiency occurs due to faulty utilization of the ingested food or insufficient intake, hormonal or enzymatic imbalance, metabolic disturbances, chronic alcoholism, etc. It also causes severe malocclusion, often the main problem is the upsetting of the dental developmental time-table. The resultant premature loss, prolonged retention, poor tissue health and abnormal eruptive paths lead to malocclusion.

Hormones are regarded as the chemical messengers involved in the transmission of information from one tissue to another and from cell to cell.

Hormones have an important influence on the rate of tooth movement, and their consumption is essential to adequately discuss treatment planning with patients. This is especially important in dentistry because many of the patients attending dental clinics face stressful situations. Awareness is therefore necessary on the risks and difficulties that may arise during the dental and orthodontic management of patients with endocrine disorders and most common oral manifestations.

- **Role of nutrition on development of oral tissue**

Nutrient imbalance on developing oral tissues maternal diet and craniofacial development

Deficiency of folic acid, riboflavin and zinc are known to induce clefting ,Suboptimal levels nutrients may potentiate other teratogenic agents, Excessive intake of Vitamin A can act as a teratogen.

Teeth and Salivary Glands

A variety of amino acids, vitamin A, D and C, calcium and phosphorus must be present to insure optimal calcification during the teeth formation and calcifying periods.

Deficiency of essential nutrients results in the following effects on teeth.

Hypoplasia
Hypocalcification
Irreversible changes in salivary glands

Oral Epithelium

Nutritional deficiency may affect obligatory DNA synthesis in the sulcular epithelial tissues and compromise an important component of its defense mechanism of the periodontal tissues.

Skeletal Tissues

Vitamin A, D, E and C deficiencies, along with deficiency of mineral calcium phosphorus may result in inadequate bone growth patterns with malalignment and malocclusion.

- **Role of nutrition on tooth movement**

Role of Lipids in Orthodontic Tooth Movement

Orthodontic tooth movement is accompanied by the appearance of osteoclasts and subsequent alveolar bone resorption, which is mediated through the local production and action of PGS. Inhibitors of PG synthesis eg; NSAIDs, inhibit the appearance of osteoclasts and reduce the rate of tooth movement. Dietary n-3 fatty acids have actions similar to those of NSAIDs, and the intake of dietary lipids affect bone remodelling and subsequent orthodontic tooth movement. The number of osteoclasts and the degree of bone resorption on the pressure side during tooth movement.

Vitamin C

Lack of vitamin C halts osteogenesis and periodontal ligament organization [8,9]. It has been shown that vitamin C deficiency during orthodontic treatment reduces the tooth movement because of its effect on tissue healing. Its main effect is on the periodontal ligament (PDL).

Vitamin D

Vitamin-D and its most active metabolite, vitamin-D3, together with parathyroid hormone and Calcitonin, regulate the amount of calcium and phosphorus in the human organism. It promotes intestinal Ca+2 and PO4-3 absorption. Vitamin- D3 increases bone mass and thus reduce fractures in osteoporosis patients. Considering its beneficial effects on bone tissue, it may be assumed that **it inhibits tooth movement.**

Role of Hormones in Orthodontics

Most of the functions of nervous system are executed by hormonal substances, and endocrine functions are controlled by nervous system.

Growth Hormone (GH)

GH is a protein hormone, secreted by the acidophil of the anterior pituitary gland. Apparently, it has no direct action upon bones, acting through a substance called somatomedin. GH stimulates the liver to secrete.

Effects of Growth Hormone on Craniofacial Growth:

With Idiopathic Growth Hormone Deficiency, the length and depth of the face are inappropriately small for the child's age, with the face maintaining childlike convexity**. Many studies have reported mandibular total length (Gnathion-Condylion) is reduced, primarily as a result of the small ramus height (Condylion- Gonion). In addition, the maxilla is significantly reduced, and there may be a comparable degree of reduction in the mandible. The maxilla is often retrognathic but is affected less than the mandible.** Concerning cranial base size, many studies have reported that the posterior cranial base length is smaller than the anterior cranial base (N-S) length. By contrast, facial convexity decreases with GH replacement therapy, and its main effect seems to be on condylar growth. Another study reported that **growth in the Gnathion-Condylion and lower facial height (ANS-Me) are accelerated, whereas the cranial base length changes minimally.** Cantu et al found that catch-up growth with GH therapy affects the anterior facial height, posterior facial height, and posterior cranial base.

Effect on Dental Development

Dental delay is always less pronounced than height or bone delay**. Dentition seems to be harmoniously delayed,** so that all studied components of dental development (primary root resorption, secondary tooth formation and eruptive movement) display the same degree of retardation.GH influence on growth starts after 9 months of age, so that the effect on the growth of primary teeth is very little known.

Prostaglandins (Pgs).

Prostaglandins and orthodontics

Yamasaki and associates conducted experiments on rats to investigate whether the synthesis of prostaglandins is induced by orthodontic force, and whether exogenous prostaglandins can produce bone resorption similar to orthodontic force. They reported that the application of orthodontic force did indeed cause increased synthesis of prostaglandins, which in turn stimulates osteoclastic bone resorption. **prostaglandins are important mediators of mechanical stress.**

Clinical and animal studies by various authors have identified the role of prostaglandins (PGE1 and PGE2) in stimulating bone resorption. They have reported a direct action of prostaglandins on osteoclasts in increasing their numbers and their capacity to form a ruffled border and effect bone resorption. Studies have also identified other agents such as growth factors (platelet-derived growth factors), hormones (parathormone [PTH]), and interleukins or other cytokines that induce PGE2 production, to effect bone remodelling and tooth movement.

Lee et al also reported that systemic intravenous administration of PGE1 was more effective and produced more bone resorption than local injection. Clinically and an increase in the number of osteoclasts and resorption lacunae, microscopically. The two major Drawbacks associated with the use of prostaglandins as reported by them are: pain reaction and the need for frequent administration because of rapid metabolism of PGE2 in the lung.

Corticosteroids

Corticosteroids are immunosuppressive and anti-inflammatory agents, widely used to treat pathological processes in medical and dental practice, in such a way that patients under orthodontic treatment may present variations in normal bone remodeling due to the use of these drugs. In the process of tooth eruption, however, cortisone has a special effect. Eruption rate is accelerated.

Effects on bone and tooth movement

Evidence indicates that the main effect of corticosteroids on bone tissue is direct inhibition of osteoblastic function and thus the decrease of total bone formation. Corticosteroids increase the rate of tooth movement, and since new bone formation can be difficult in treated patients, they decrease the stability of tooth movement and stability of orthodontic treatment in general.

Estrogens

Estrogen is considered to be the most important hormone to affect bone metabolism in women. It controls bone remodeling during reproductive life, and maintenance of maximum bone mass after menarche Estrogens do not have any anabolic effects on bone tissue; they directly stimulate the bone forming activity of osteoblasts.

Effect on tooth movement

Studies have shown that **Estrogen decreases the velocity of tooth movement.** Oral contraceptives taken for long periods of time can influence the rate of tooth movement. **Androgens also inhibit bone resorption and modulate the growth of the muscular system and may affect the length and results of orthodontic treatment.**

Estrogen influences the composition and degradation of collagen fibers in the periodontal ligaments and the remodeling of the alveolar bones. While estrogen influences the deposition and cross-linking of collagen fibers, it also enhances the alkaline phosphatase (ALP) activity and the secretion of osteocalcin (OCN) and osteoprotegerin (OPG) in the periodontal ligament cells (PDLCs) **.**

Rate of tooth movement is tied up with activity of osteoclasts. Estrogen can inhibit activity of osteoclasts in a direct or indirect manner, thereby modulating bone resorption.

Estrogen inhibits tooth movement by increasing the bone mineral content and bone mass and by reducing the bone resorption rate. Several studies have shown that estrogen deficiency accelerated tooth movement.

Estrogen directly stimulates the bone-forming activity of osteoblasts, so it is reasonable to expect a decrease of the velocity of orthodontic tooth movement. Androgens also inhibit bone resorption and modulate the growth of the muscular system. Thus, the excessive use of these drugs by athletes, in an attempt to achieve better athletic scores, may affect the length and the results of orthodontic treatment.

Thyroid Hormones (Th)

Thyroid hormones are recommended for the treatment of hypothyroidism and used after thyroidectomy in substitutive therapy**. Thyroxin administration lead to increased bone remodeling, increased bone resorptive activity, and reduced bone density.**

Thyroid disorders commonly affect craniofacial and dental structures. The dental and craniofacial retardation manifested under prolonged hypothyroid conditions differs from the isolated lack of GH**. The main difference is the cranial vault, which shows growth retardation in hypothyroidism, and reduced facial height in children with prolonged untreated hypothyroidism. Thyroxin administration seems to lead to increased bone remodeling, increased bone resorptive activity and reduced bone density.** Thyroid hormones increase osteoclastic bone resorption in neonatal mouse calvarium by stimulation of prostaglandin synthesis.

Parathyroid hormone (PTH)

Relative studies have confirmed that parathyroid hormone could stimulate both osteoclast-mediated bone resorption and osteoblast-mediated bone formation, therefore accelerating the bone turnover rate. **The ultimate effect of parathyroid hormone on bone remodelling is determined by the administration protocol. Continuous infusion of parathyroid hormone results in a catabolic effect, whereas intermittent injection leads to an anabolic effect. Intermittent low-dose parathyroid hormone analogues have been widely administered in the clinical treatment of osteoporosis.**

Previous studies have investigated the effect of different administration patterns of parathyroid hormone and the results suggested that systemic continuous infusion or local chronic application of parathyroid hormone could accelerate tooth movement through enhancement of alveolar bone resorption, whereas long-term intermittent injection of parathyroid hormone facilitated periodontal repair of bone or root resorption after orthodontic tooth movement through activation of osteoblastic cells.

Salazar et al evaluated the tooth movement with intermittent parathyroid hormone injections mentioned that unlike other osteoporosis treatment drugs such as bisphosphonates, calcitonin, and calcium with vitamin D, which diminish bone resorption. Parathyroid hormones stimulated osteoblast function with no interference in osteoclast activity and facilitated bone remodeling. This could explain the acceleration effect of intermittent parathyroid hormone. Intermittent parathyroid hormone administration, results in an increase in osteoclastic resorptive activity. In turn, the resorptive activity increases the release of osteogenic growth factors from bone matrix and osteoclasts, and it stimulates bone remodelling.

Calcitonin

Calcitonin inhibits bone resorption by direct action on osteoclasts decreasing their ruffled surface which forms contact with resorptive pit. It also stimulates the activity of osteoblasts. Vitamin-D and its most active metabolite, vitamin-D3, together with parathyroid hormone and Calcitonin, regulate the amount of calcium and phosphorus in the human organism. It promotes intestinal Ca+2 and PO4-3 absorption. Vitamin- D3 increases bone mass and thus reduce fractures in osteoporosis patients. Considering its beneficial effects on bone tissue, it may be assumed that it inhibits tooth movement. Calcitonin inhibits bone resorption by direct action on osteoclasts decreasing their ruffled surface which forms contact with resorptive pit. It also stimulates the activity of osteoblasts. Because of **its physiological role**, it is considered to inhibit the tooth movement, consequently delay in orthodontic treatment can be expected.

Conclusion

Corticosteroid, parathyroid and thyroxin hormone have been shown to accelerate orthodontic tooth movement whereas Estrogens inhibit the rate of tooth movement, although no direct clinical evidence is available. However, Vitamin D3 stimulates tooth movement, while dietary calcium inhibit it. Bisphosphonates have strong inhibitory effect on tooth movement.

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