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PREMATURE AGEING DISORDERS IN CHILDREN: A REVIEW

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ABSTRACT

Aging is a natural and inevitable phenomenon. In humans, aging is caused by a complicated interplay between hereditary and environmental factors. Several rare conditions exist in man that exhibit certain phenotypic characteristics associated with aging. [1, 2] Premature-ageing syndromes are a heterogeneous group of rare genetic disorders having features resembling that of accelerated ageing.[3,4] So far, more than 100 syndromes exhibiting early aging symptoms have been identified, with an approximate global prevalence of 1:50.000. Depending on the onset of symptoms, they can be divided into congenital, infantile, juvenile/adult, and adult forms.[4] Progeroid syndromes are a wide range of rare systemic diseases, which share significant anomalies in the orofacial region.[1]

INTRODUCTION

Greek words meaning "prematurely old" are the source of the term "progeria." The majority of these syndromes are known as segmental progeroid syndromes (SPS) because only certain organs and tissues exhibit signs of accelerated aging, such as hair loss or graying, lipodystrophy, scleroderma-like skin lesions, hearing loss, osteoporosis, type 2 diabetes mellitus, and early-stage malignancies. [3-5] While Hastings coined the word progeria, Hutchinson characterized the first patient in 1886 as having a "congenital absence of hair and its appendages". [6]

Progeria is defined by a phenotype that resembles a mix of reduced facial height and early aging. Patients with this uncommon illness require extensive dental care due to their skeletal and dental abnormalities. [7]

Premature-aging syndromes can be classified into three categories based on the underlying molecular defect: diseases resulting from altered nuclear lamina architecture, diseases caused by gene mutations necessary for DNA repair and genome stability maintenance, and diseases resulting from various pathophysiological processes, including mitochondrial impairment and alterations in connective tissue, among others. [8]

Progeroid syndromes include clinically and genetically heterogeneous diseases such as, Rothmund-Thomson syndrome, Cockayne syndrome, Hutchinson-Gilford syndrome, Werner syndrome, Dyskeratosis congenita, Trichothiodystrophy or Tay's syndrome, Petty-Laxova Progeroid Syndrome and Wiedemann-Rautenstrauch syndrome. [9]

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Hutchinson gilford progeria syndrome

Hutchinson Gilford progeria syndrome (HGPS) is a rare disorder marked by premature ageing involving the bones, skin, and blood vessels. Growth retardation, low birth weight, and aging characteristics such atheroma formation leading to myocardial infarction and stroke, large superficial veins, unusual facial features, and extremities that resemble those of an old person, are its hallmarks. Because of myocardial infarction or cerebrovascular accident, resulting from atheroma formation, patients often die at a very young age. [10]

Etiology

It is most likely an autosomal dominant condition that manifests sporadically. Autosomal recessive inheritance is the pattern of inheritance for non-classical HGPS. [11] Mostly, a de novo point mutation in the DNA causes HGPS.[12]

The mutant LMNA gene and/or aberrant post- translational processing (ZMPSTE24 gene mutations) are the two recognized molecular lesions of HGPS. Both mutations lead to progerin, an improperly produced lamin A. HGPS is caused by de novo point mutations in the lamin A/C gene, LMNA, which alternatively splices to produce the lamin A and lamin C proteins. [13]

Clinical features

The diagnosis is primarily clinical, and the symptoms start to show up in the first year of life. At birth, these children appear normal and healthy, but

because of development failure, they gain relatively little weight over time (usually within a year). When they reach the ages of 1.5 to 2 years, their faces are small, their jaws are abnormally large compared to their heads, and they have a high-pitched voice, irregular teeth, a pinched nose, large, wide-open eyes, undersized dystrophic clavicles and absence of sexual maturation. There is a gradual loss of body fat and eyelashes, along with thinning and falling hair that eventually results in total baldness. Veins are visible through the skin's translucency, fragility, and thinness. Complaints of angina, high blood pressure, stiffness of joints and hip dislocation are also common.[14]

Additionally visible conditions include macrocephaly, reduced sweating, nail dystrophy, onychogryphosis, koilonychia, hypoplastic nipples, keloids, delayed and abnormal dentition can also be seen. Skeletal abnormalities include osteoporosis, osteolysis, necrosis, fracture, coxavalga, dystrophic clavicles, 'horse-riding' stance, thinning of cranial bones, delayed closure of cranial sutures and anterior fontanelle. It is interesting to notice that several signs of aging, such as osteoarthritis, cataracts, presbycusis, and presbyopia, are absent in HGPS patients despite senile degeneration. [15]

The histopathologic changes in the skin include atrophy of the dermis and epidermis. There is progressive hyalinization of dermal collagen and loss of subcutaneous fat. [16, 17]

Treatment

Patients should have 6-12-monthly growth. Assessments for the heart, brain, musculoskeletal system, teeth, ears, and eyes must be done. An annual physical should also include screenings for lipids, electrocardiograms, echocardiograms, carotid duplex scanning, hip X-rays, and bone densitometry scans. Bone fractures can be avoided with protective measures. To prevent atherosclerotic changes, dietary restriction and occasionally medication in the form of low-dose aspirin are advised as preventative measures.

One of the features of the cells in the HGpS children is structural abnormalities of the nucleus (related with build-up of prelamin A), which can be reversed by a class of cancer medications called farnesyltransferase inhibitors (FTIs). These medications, limit the ability of farnesyltransferase to bind farnesyl groups to progerin proteins. [18]

Drugs such as pravastatin, lonafarnib, zolendronate and rapamycin, has shown to reverse the cellular phenotypic deficit in vitro and to improve survival in these patients. [19]

When farnesyl transferase is blocked, prelamin A and its shortened counterpart progerin/LADelta undergo alternate prenylation by geranyl tranferase, as demonstrated by Varela and colleagues. The limited effectiveness of FTIs in enhancing the physical makeup of the progeroid mice models has been attempted to be explained by this study. Additionally, they demonstrated that the combination of statins and amino bisphosphonates significantly improved the aging-related phenotype of Zmpste24 mice and reduced both farnesylation and geranyl geranylation of progerin and prelaminA. Furthermore, these greatly increased the mice's longevity. [20]

According to the results of a two-year clinical treatment trial involving twenty-five progeroid-children, the FTI medication lonafarnib has been shown to effectively promote weight increase and ameliorate skeletal and cardiovascular diseases. [21]

Standardized protocols are not available; every solution is customized based on the needs of the patient and the most recent advancements in prosthetic technology. One of the possibilities for treating dental issues is prosthetic rehabilitation. [22]

Werner syndrome (WS)

Werner syndrome (WS) is a rare PS which shows a similarity to HGPS in its clinical symptoms.

Etiology

It is inherited as an autosomal recessive trait. The mutation affects telomere preservation and subsequent DNA replication in the cell. It is caused by a mutation in the WRN gene, which codes for DNA helicase and is found on chromosome 8.

Clinical features

Individuals with this syndrome develop normally upto about 10 years of age. In the early stages of adolescence, the condition causes cataract development, atherosclerosis, skin shrinkage, osteoporosis, loss of subcutaneous fat, graying, and hair loss. In WS, the average survival age is 54 years.[23, 24]

A high-pitched, hoarse voice, a "pinched" face, narrow limbs, truncal obesity, and flat feet are common characteristics. The two most frequent causes of mortality in these patients are myocardial infarction and malignancy. [25] Additional characteristics include periodontal disease, microstomia, poor oral hygiene, missing teeth, and carious teeth.[26]

Treatment

mTOR inhibitors are used in novel treatment methods. For a wide range of species, the mTOR pathway is a critical regulator of aging and age-related illnesses. The mTOR pathway plays crucial biological roles in both stimulating cell proliferation in nutrient-rich situations (high mTOR activity) and redirecting metabolic resources toward the maintenance of stem cells in nutrient-poor conditions. In WS cells, basal autophagy and mTOR signaling are increased. Long-term rapamycin therapy enhances nuclear morphology, growth rate, and reduces the buildup of DNA damage foci.

In addition, selective inhibitors of p38 mitogen-activated protein kinase (MAPK) have been studied as a possible therapeutic intervention for WS and other diseases related to chromosomal instability. Primary fibroblasts undergo premature senescence due to the activation of two tumor suppressor pathways, p53/p21WAF1 and pRb/p16INK4A, which is mediated by the MAPK during stress-signaling.[25]

Human induced pluripotent stem cells, or hiPSCs, are also studied by Cheung et al., 2014; Shimamoto et al., 2014 who demonstrated reversion of senescence-related cellular abnormalities of hiPSC generated from fibroblasts from WS patients. Numerous variations were found in the gene expression profiles of WS and control fibroblasts, the majority of which are thought to be related to cellular aging. [27, 28]

The necessary dental procedures are restorations and prosthetic rehabilitation. Thermoplastic prostheses are flexible, making them simple for insertion and removal from the mouth, which

is helpful in cases of microstomia. [26]

Dyskeratosis congenita (DKC)

DKC is an inheritable bone-marrow failure (BMF) disorder.

Etiology

It is linked to mutations in DKC1, TERC, TERT, NOP10, NHP2, TIN2 or TCAB1 genes16, implicating the physiology of telomeres17.

Clinical features

Includes nail dystrophy, abnormal skin pigmentation, mucosal leukoplakia and pulmonary complications. [29]

Anomalies in structures generated from ectodermal tissue lead to severe periodontal damage. Patients may have bone loss, recession, bleeding, and gingival inflammation, all of which are symptoms of juvenile periodontitis. Additionally, minor taurodontism and a reduced root/crown ratio may be present. Extra-minor characteristics include microcephaly, developmental delay, intrauterine growth retardation, abnormalities of the eyes and hair, excessive perspiration, short stature, hypogonadism, enteropathy, liver disease, esophageal and urethral stenosis, osteoporosis, and avascular necrosis of the shoulders and hips.[30] Additional prevalent conditions include hypodontia, small maxillary lateral incisors, short-blunted roots, delayed eruption, tooth mobility, severe dental caries, and early tooth loss.[31]

Treatment

Since DKC is a multisystem disorder, it is important to monitor many systems of the body. Clinical trials for telomerase-based therapies are now underway. They offer benefits, drawbacks, challenges, and hopes when combined with existing cancer therapies. Research has shown that between 50% and 70% of patients responds to androgens. Sex hormones have the ability to influence telomerase.

Oxymetholone has a 70% response rate in patients. DKC individuals treated with danazol, a synthetic androgen derivative also referred to as 17α-ethinyl testosterone, reported beneficial activity. Its main effects are antigonadotropic and antiestrogenic, with fewer masculinizing side effects.

For patients with DC, allogeneic hematopoietic stem cell transplantation, or allo-HSCT, is the sole cure for BMF. However, underlying organ dysfunction has been linked to severe transplant-related toxicities, especially when myeloablative conditioning regimen dosages of chemotherapy or chemoradiotherapy are used.[30] Oral prophylaxis, fluoride treatment, careful recommendations for oral hygiene, restorations, and extractions are all included in dental therapy. [31]

Trichothiodystrophy (or Tay's syndrome)

Trichothiodystrophy (TTD) or Tay's syndrome is an autosomal recessive disease with depressed RNA synthesis.

Etiology

Patients have abnormal production of transcription factor II H(TFIIH), a general transcription factor active in basal transcription and nucleotide excision repair, as a result of mutations in genes encoding any of the three subunits of TFIIH-ERCC2 (XPD), ERCC3 (XPB), and GTF2H5(TTDA).[32]

Clinical features

It is characterized by brittle hair, ocular problems, mental and general growth retardation, small stature, and other developmental abnormalities such as congenital ichthyosiform erythroderma.[33] The other related traits are gingival hypertrophy, caries, enamel hypoplasia, maxillary hypoplasia, cleft lip, and dental abnormalities.[34]

Treatment

Currently, TTD has no recognized effective treatment. In addition to dermatology, several other medical specialties must be involved in a multidisciplinary approach for the multisystem disorders of TTD to be effectively managed. [33] After UV exposure, it has been shown that DNA repair-deficient fibroblasts can restore their normal p53 response through retro virally mediated correction using the XPD gene. [34] Oral prophylaxis, meticulous oral hygiene instructions, restorations, and gingival surgical treatments are all included in dental therapy.

Cockayne syndrome

Cockayne syndrome is another rare congenital disorder which is characterized by growth failure, atypical photosensitivity and importantly impaired development of the nervous system.

Etiology

This disease is eventually brought on by defects in the DNA repair pathway caused by mutations in any of the ERCC6 and ERCC8 genes.

Clinical features

Growth and development of these individuals become aberrant by the time they are two years old. The aging process is characterized by the unique physical features of cachectic dwarfism, such as sunken eyes, a decrease in skin and hair thickness, and an arched standing posture. These kids have intellectual deficiencies and cognitive impairments, which frequently get worse by the time they are twenty. [35, 36] These patients also exhibit enamel hypoplasia and dental caries.

Treatment

If indicated, treatments such as physical therapy, cochlear implants for hearing, cataract operations, sunscreen for photosensitivity, feeding tubes for malnourishment, dental restorations for caries, etc., may be beneficial. Currently, only symptomatic treatments are available; no treatment that could stop or decrease the disease's course has been found. [37]

Petty-laxova progeroid syndrome (PLWPS)

Petty-Laxova Progeroid Syndrome (PLWPS) is a progeroid syndrome with an unidentified genetic etiology and prenatal age of onset.

Clinical features

Profoundly reduced subcutaneous fat, scoliosis, aplastic and hypoplastic distal phalanges with aplasia and hypoplasia of nails, short stature, large anterior fontanelle, anomalies in tooth position or shape, umbilical hernia at birth, pseudomacrocephaly, wide calvaria, sparse scalp hair and normal cognitive and motor development are common

features. [38,39,40]

Treatment

To support a customized preventative plan, the first dental examination for these individuals should be scheduled as soon as the first deciduous tooth erupts. It is recommended that missing teeth undergo prosthetic rehabilitation.[22]

Wiedemann-rautenstrauch syndrome

Wiedemann-Rautenstrauch syndrome (WRS) is an uncommon progeroid syndrome (PS) with an autosomal recessive pattern of inheritance. WRS, also known as the neonatal progeroid syndrome(NPS), differs from other progerias in the complex of signs and symptoms present at birth. The etiology of WRS remains unknown. [41]

CLINICAL FEATURES

Characteristics include severe intrauterine and postnatal development failure, hydrocephaly, noticeable scalp veins, thin hair on the scalp, eyebrows, and eyelashes, generalized lipoatrophy, increasing neurological degeneration, micrognathia, and natal teeth. Gingival hypertrophy and tooth caries are other oral features. [42]

Treatment

Therapies for WRS are symptomatic. Extractions, restorations and surgical management of gingival enlargement including gingivectomy and flap surgeries, are the treatment options. [43]

Rothmundthomson syndrome

The RothmundThomson syndrome (RTS) is an autosomal recessive disorder that manifests in childhood, usually with early skin lesions in the first year of life.

Etiology

RTS is resulted due to a mutation on the RecQL4 (8q24) gene, which codes for the RecQ DNA helicase protein, which is associated with the mutation of chromosomic stability.

Clinical features

There are several symptoms that can be seen, including alopecia, delayed speech, low weight and height, cholestasis, iron deficiency anemia, carious lesions, delayed tooth eruption, rhomboid glossitis, and hyperkeratotic tongue. These patients also exhibit behavioral issues.

Treatment

It is crucial that these patients with unique requirements receive dental treatment. Dental care comprises tooth extractions, endodontic treatments, composite restorations, and oral hygiene instructions. Non-pharmacologic techniques including tell-show-do, voice control, nonverbal communication, positive reinforcement, and distraction are employed in behavior management. [44]

CONCLUSION

In the planning and execution of dental treatment in these patients, the roles of the dermatologist, prosthodontist, paedodontist and oral diagnosis specialist are vital. The main objectives of dental care are to eliminate the potential and acute infections of the teeth and gingiva. In order to manage and educate the patient about oral hygiene, dental, and occlusal problems, genetic counseling, early detection of the condition, and referral to the dental specialities are also crucial.[45]

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