The Dental Needs and Treatment of Patients with Down Syndrome

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KEYWORDS

• Down syndrome • Oral health • Dental management • Systemic considerations

KEY POINTS

- Down syndrome is associated with various systemic findings, oral findings, and diseases.
- Oral health can be maintained through proper knowledge, regular visits, and proper intervention.
- Dentists need to take a holistic approach including behavioral, oral, and systemic issues.
- This review of the literature focuses on oral anomalies, systemic interaction, management, and recommendations.

INTRODUCTION

Down syndrome (DS) is the most common chromosomal disorder and cause of mental retardation. First described by Esquirol in 1838, and later in 1866, this disorder was described by John Langdon Down and named mongolism.[1–3] It is caused by an extra copy of chromosome 21, giving a chromosome count of 47.[1] Trisomy 21 results from nondisjunction of homologous chromosomes 21 during gametogenesis or early after fertilization.[4,5] It occurs most frequently (90%) during meiosis in women. The remaining percentage arises from the father's side or from nondisjunction of chromosomes during a postzygotic mitosis. Maternal age plays a role in the nondisjunction of chromosome 21.[6,7]

Robertsonian translocation to another acrocentric chromosome or isochromosome, usually chromosome 14t(14q;21q) or t(21q;21q). The affected child has 3 copies of the long arm of chromosome 21 instead of 2. In 25% of the cases, one of the parents is trait carrier.[6,7]

Mosaicism is a form of DS in which an individual has 2 or more genetically distinct cell lines, some with the normal number of chromosomes, and some that are aneuploid. It is caused by a nondisjunction during postzygotic mitosis.[6,7]

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Regardless of the chromosomal abnormality background, individuals with DS have a noninherited mental retardation with varying range of cognitive dysfunction and characteristic facial features. General features include cardiovascular defects, skeletal abnormalities, ocular defects, protuberant abdomen, hypogonadism, and delayed puberty. Craniofacial features may include brachycephaly, flat occiput, broad and short neck, hypoplasia of the maxilla, upslanting palpebral fissures, short ears, and small chin. Some of the oral manifestations are periodontal disease, delayed eruption, malocclusion, thickening of lips, macroglossia, and fissured and protruding tongue.

DENTAL CARIES IN INDIVIDUALS WITH DOWN SYNDROME

It is generally agreed that caries prevalence and incidence in both institutionalized and noninstitutionalized individuals with DS is lower than in normal and other mentally retarded individuals.[8–12] A meta-analysis revealed that individuals with DS have significantly lower levels of dental caries.[13] Causes for low caries levels are delayed eruption, spacing of teeth, congenital oligodontia, and some salivary characteristics.[14–16] Small teeth and morphologic anomalies may also play a role.[16–18] A few reports showed higher or no difference in caries level in individuals with DS.[9,13,19,20] Differences in results could be attributed to sample characteristics such as sociodemographic and geographic location. Other risk factors are sweets intake, fluoridation, poor oral hygiene, frequency of dental checkups, deficiency of health education, lack of prevention programs, and the awareness of the parents.[10,15,20–24] Extent of disability and intelligence quotient (IQ) level also influenced oral health status.[21]

PERIODONTAL DISEASES

Patients with DS have increased prevalence of early progressive periodontitis and edentulism compared with normal or mentally retarded individuals.[15,24–26] Severe periodontitis usually occurs during teenage years, with more bone loss than other mentally retarded patients.[27] The average rate of bone resorption is 0.03 mm/y, with severe bone loss in 65% by 35 years of age, primarily affecting the lower anterior area.[27] Both local and systemic factors have been suggested to cause periodontal destruction. Local causes may include reduced oral hygiene, calculus deposits, macroglossia, tooth morphology, gingival tissue abnormalities, saliva characteristics.[28,29] Another factor is the differences in subgingival microbiota among patients with DS.[29,30] For example, there is an increased level of *Propionibacterium acnes* (associated with persistent apical periodontal infections), *Treponema socranskii* (linked to tissue destruction), and *Streptococcus constellatus* (refractory periodontitis).[30]

Systemic factors are DS related, such as oxidative burst intensity of granulocytes and monocytes, depressed chemotaxis, impaired oxidative metabolism, and immunity.[28,29,31–35] Another possible cause is decreased CD4+/CD8+ ratio, and thus altered immune regulation and function.[36]

Poor periodontal health and prognosis have been linked to individual age, IQ level, and parental education.[37] However, supervised brushing, good dental care, and prevention measures tend to improve the periodontal status.[32,37–41]

Properties of Saliva

Studies, although not conclusive, have reported variations in flow rate, pH and salivary electrolyte levels (sodium, potassium, chloride, calcium, phosphorous), α-amylases, buffering capacity, and salivary counts of mutans streptococci.[14–16,42–44] The lower