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Original Research Article

"Impacted Teeth a Review on Gentic Background"

Dr. Teena Maria Wilson[1*], Dr. Sandhya Jain[2]

[1]Postgraduate student Dept of Orthodontics and Dentofacial Orthopedics Govt. College of Dentistry City: Indore Madhya Pradesh India
[2]Head of Department Dept of Orthodontics and Dentofacial Orthopedics Govt. College of Dentistry, Indore Madhya Pradesh India

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***Corresponding author:** Dr. Teena Maria Wilson

Abstract

Impaction is defined by Kuftinec and Shapira as a condition with embedded teeth in socket so that its eruption is prevented and will be locked in that position by either bone or adjacent teeth. Unlike isolated impactions multiple impactions may be due to the underlying syndromes or disorders. In cases where confusion exists in differential diagnosis additional examinations might become necessary to exclude systemic and metabolic disorders.

Keywords: Multiple impacted, syndromes, genetics, dentistry, differential diagnosis.

INTRODUCTION

Eruption is a process of axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane.[1] those teeth failed to emerge to oral cavity due to various reasons like insufficient space, early loss of primary teeth with eventual closure of space, crowding of arches rotation of tooth buds, excessive fibrous tissue over an erupting tooth etc. are termed as impacted teeth. Of all the reasons eruption cysts being relatively rare cause of impaction [1]. Local biomechanical impediments, and secondarily from childhood maxillofacial or dentoalveolar trauma, reconstructive surgery of the facial skeleton, malposition of an adjacent tooth, thick osseous or mucosal tissues above teeth, insufficiently developed jaw bone or the difference in the rate of development and maturation of jaw bones and teeth respectively, disturbances in eruption and direct or indirect effects of cysts or neoplasm of jaws are always in list of reasons for impaction [2].

Literature has a good collection of multiple impacted permanent teeth [3-7]. Bayar *et al.*[8] have reported three cases with multiple impacted teeth involving both jaws in which no syndrome or systemic conditions have been detected. Multiple impacted teeth may be related to syndromes like Cdysostosis[9], Gardner syndrome [10], Yunis – Varon syndrome [11]. Down syndrome, Aarskog syndrome, Zimmerman-Laband syndrome and Noonan's syndrome [12]. Also metabolic disorders like mucopolysaccharidoses[13]. And hormonal disorders like hypothyroidism, hypothyroidism, hypopituitarism, and Vitamin D-resistant rickets. The most common syndrome with impaction is Cleidocranial dysplasia [14].

In molecular level analysis, numerous eruption-regulating molecules having similar and overlapping functions EGF, EGF-R, CSF-1, CSF-1R, IL-1, IL-1R, c-Fos, NFB, MCP-1, TGF-β1, PTHrP, Cbfa-l (now called Runx2), OPG, RANK/RANKL are there. But even the absence of a single factor does not interrupt the event of eruption. Defect in some genes can also be responsible for this condition. Stellate reticulum and dental follicle are the sites for these molecules[15.] This review tries to light some region of genetics which is related to syndromes with multiple impacted teeth as their manifestation. Table 1 shows some syndromes and disorders which can cause tooth impaction and the etiologic gene behind them. A flow chart (Flow chart no. 1) is added for differential diagnosis of syndromes associated with impactions.

Table-1: Disorders causing tooth impaction and their etiologic gene

DISORDERS, DISEASES AND SYNDROMES CAUSING TO TOOTH IMPACTION	EITIOLOGIC GENE
Cleidocranial dysplasia	RUNX2 gene in chromosome 6
Gardner syndrome	ATP6V1B2 gene in Chromosome no 5
Down syndrome	Trisomy 21
Aarskog syndrome	FGD1 Gene
Zimmerman-Laband syndrome	KCNH1 Gene in Chromosome no 1
Noonan's syndrome	PTPN11 Gene
GAPO syndrome	ANTXR1 Gene in Chr2
Osteoglophonic dysplasia	FGFR1 Gene
Osteopathia striata	WTX Gene
Osteopetrosis	CLCN7 Gene
Progeria	LMNA Gene
Singleton-Merten syndrome	DDX58 and IFIH1Genes
Yunis-Varon syndrome	FIG4 Gene
Mucopolysaccharidoses	IDUA Gene

Cleft lip and palate

Most common congenital craniofacial deformity occurs due to the incomplete fusion of facial buds, primary and secondary palate. The prevalence of the isolated cleft lip and isolated cleft palate is approximately 1/1000 and 1/2000 respectively. The incidence was highest reported in Afghans and lowest in negroid[16]. Cleft lip with or without cleft palate is listed as a feature of more than 200 specific genetic syndromes. Isolated cleft palate is recorded as a component of more than 400 described syndromes. The proportion of orofacial clefts associated with specific syndromes is between 5% and 7%. The major features of cleft patients are missing or malformed lateral incisor, superneumeries, breached palate and lips, constricted maxilla with mid face deficiency. Multiple impactions near the cleft are common due to space deficiency, absence of bone and scarring after surgery acting as a barrier. Usage latest diagnostic methods especially CBCT and a craniofacial team for diagnosis and treatment can help in getting a great outcome of result.

Cleido cranial dysplasia

Autosomal dominant nature of inheritance occurs one per million individuals which makes this disorder a rare one. 40% of occurrence has been reported sporadic [17]. Although various phenotypical presentations have been reported some of the clinical manifestations are common. Of the skeletal manifestations missing or hypo-plastic clavicle is the classical one.

Large and wide-open fontanels, delayed primary and permanent teeth eruption, superneumery teeth with crowding, open pubic symphysis, short and broad thumbs, and brachydactyly[18]. Scoliosis, genu valgum, and pes planus are also seen. Other abnormalities of skeleton like obliterated maxillary sinuses, pelvic abnormalities maxillary constriction with dental crowding will also be seen [19, 20]. The patients may show variable degree of neurological symptoms like motor delay. Although more than 48 phenotypes have been identified most of them are associated with multiple teeth impaction. Even though early diagnosis is easier in frank phenotypic expression cases, in mild cases oral findings can be helpful in identification.

Aarskog syndrome

Aarskog syndrome also known as facio-digital-genital syndrome and was tailored to literature by Dagfm Aarskog in 1970. He found this syndrome in 7 male patients of same family [21]. Clinical features include hypertelorisam, ptosis, down slanding palpebral fissures, short nose, wide philtrum and maxillary hypoplasia. Characteristic short 5th finger and short stature helps in diagnosis [22, 23]. They might also show pectus excavatum and prominent umbilicus. Syndromic patients are expected to have normal intelligence and are associated with normal life span.

Ellis-van creveld syndrome

Also known as chondroectodermal dysplasia. It is an autosomal recessive disorder with characteristic four clinical manifestations like chondrodysplasia, polydactyly, ectodermal dysplasia and congenital heart defects. The presence of a variety of oral manifestations makes this syndrome to be noticed first by dental surgeons. The oral and clinical manifestation includes fusion of upper lip to the gingival margin, multiple frenums, microdontia, impactions and congenitally missing teeth. Majority of patients dies due to cardiopulmonary complications during childhood so life expectancy is less. On dental point of view these patients require multidisciplinary treatment under strict

prophylactic coverage due to high risk of infection [24].

Noonan's syndrome

Multiple parts of body are affected by this syndrome and are included with skeletal, cardiac and bleeding disorders. It occurs 1 in 1,000 to 2,500 people. 50-70% of affected patients have short stature. Noonan syndromic patients are characterized with a distinctive facial feature of deep groove between nose and philtrum, hypertelorisam with blue or blue-green colored eyes. Oral features are included with high arched palate, crowed or malaligned arch and micrognathia. They are featured with short webbed neck and low hairline at nape of neck [25]. A vast range of bleeding disorders has been identified along with this syndrome which can cause bruising, epistaxis or prolonged bleeding. So, extreme care is mandatory while treating these patients at clinic.

Gardners syndrome

Autosomal dominant disorder with highly variable expressivity. Affected gene is ATP6V1B2 gene in Chromosome no 5. Major feature is multiple adenomatoid polyposes in intestine along with epdermoid cysts and multiple osteomas. Oral findings are included with multiple impacted permanent and super neumerary teeth in almost 30% of patients [26]. The intestinal polyp makes it a variant of familial adenomatoid polyposis. Some of the extra intestinal features like osteomas and skin and soft tissue tumors make its diagnosis earlier. Asymptomatic osteomas presented usually in mandible aids in differential diagnosis though they are also seen in skull [27]. Skin tumors range from osteomas, lipomas and fibromas.

Osteopetrosis

A genetic disorder causing abnormally increased bone density and there by high incidence of bony fracture. Based on the inheritance mode osteopetrosis are classified as X inked, autosomal recessive and autosomal dominant. The mutation associated with this syndrome affects osteoclasts which causes impaired bone remodeling and thereby causing denser bone formation. The mildest form is autosomal dominant, also known as Albers-Schönberg disease. The major sign and symptom is increased fracture rate. Spine irregularities like scoliosis also will be evident. The dense skull bone causes impingement of cranial nerves and there by cause's visual, auditory and other sensory and motor impairment [28]. Lack of normally functioning bone marrow can lead to anemia, immune deficiency and abnormal bleeding which should be considered during treating a patient affected by osteopetrosis with invasive methods.

Zimmerman-laband syndrome

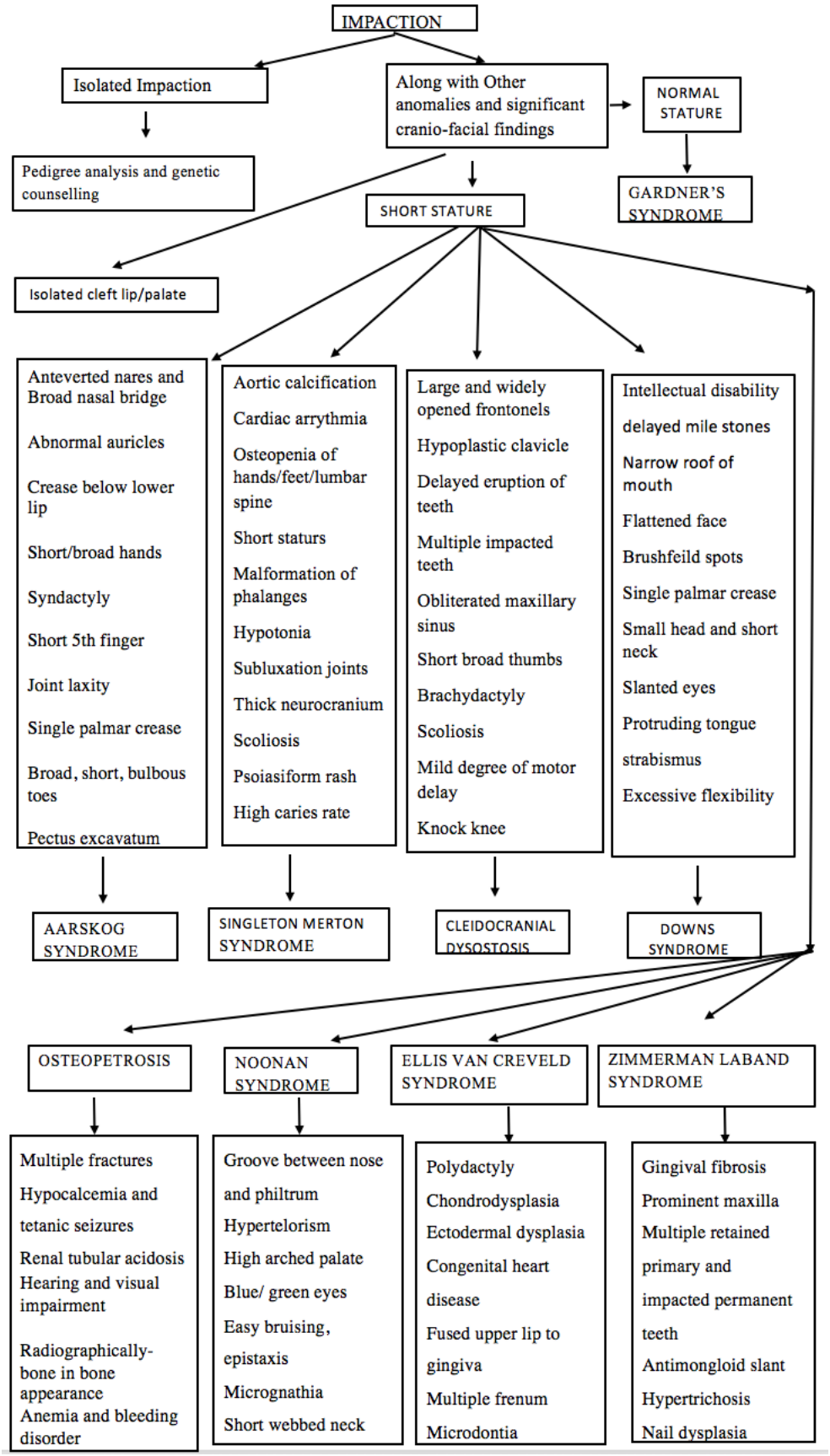
The Zimmermann-Laband syndrome is rare and autosomal dominant in nature with specific characteristics like oral findings of gingival fibrosis and prominent maxilla. Dense gingiva occasionally impairs the normal eruption of dentition and causes impaction. Facial features like thick eyebrows, antimongoloid slant, large mouth and thick lower lip can be seen. Also associated with bulbous nose, floppy ear, hypertrichosis and nail dysplasia [29]. Cases might show an association with cardiac problems. Recently an association with cataract also was found [30]. Radiologically thin cortices and wide medullary canals are seen. On oral side of treatment if the overgrown gingiva affects aesthetics or masticatory efficiency excision of tissues should be carried out.

Singleton-merten syndrome

Identified by Singleton and Merten on 1973 in two unrelated young ladies. They were presented with certain similar findings like severe aortic calcifications and abnormal root morphology of permanent teeth. The syndrome shows cardiac anomalies, aortic calcifications resembling vegetation, osteopenia of lumbar spine, hand and feet. Often the patient will be of short stature. Psoriatic rashes will also be present. Decreased mobility of finger joints along with under developed nails. Facial features like high hairline, smooth philtrum, ptosis and broad forehead can be seen[30]. Patient's OPG will show lack of primary teeth exfoliation which prevents the successor from eruption. Roots of permanent teeth will be short and mandibular molar roots will show resorption. Skull x-rays might show variable thickening also.

CONCLUSION

Multiple impacted teeth have been attracting attention of syndrome specialists and Orthodontists due to their difficulty in handling and treating. Early detection of various syndromes by analysing the pattern and heritability of multiple impactions are possible. It is equally important in giving correct management and informed decisions to syndromic patients as they require early interventions and long-term planning.



Flow chart-1: Differential diagnosis of syndromes associated with multiple impacted teeth

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