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Contents

Scientific Editorial - Conscious Sedation In Dentistry

Dr.Syed Nabeel | Editor in Chief Dental Follicle - The E Journal of Dentistry | Founder and CEO DentistryUnited.com | Director: Smile Maker Clinics Pvt. Ltd INDIA

Abstract:

Procedural sedation and analgesia, previously referred to as conscious sedation, is defined as "a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardiorespiratory function Conscious sedation has taken a major role in pain relief during dental treatment in many countries. In this short review of the same , a brief discussion on the history, technique , pros and cons have been summarized.

Keywords: Conscious Sedation, sedation dentistry

Introduction:

Conscious “A technique in which the use of a drug or drugs produces a state of depression of the central nervous system enabling treatment to be carried out, but during which verbal contact with the patient is maintained throughout the period of sedation. The drugs and techniques used to provide conscious sedation for dental treatment should carry a margin of safety wide enough to render loss of consciousness unlikely”1 Unlike General Anesthesia where a patient is completely unconscious, asleep, and unable to respond, patients under Conscious Sedation, are able to respond to commands and breath on their own.

There are many ways in which sedation is given but primarily three ways are used normally in the Dental Office :

1. Intravenous Sedation also known as Deep Conscious Sedation. Amnesia is one of the major issues faced in this type.

2. Enteral Conscious Sedation "Orally Administered Sedation", sometimes called "Sedation Dentistry". The patient will often fall asleep. Some degree of amnesia is common. The disadvantage with this method of sedation, is that the level of sedation for each person is not predictable. Benzodiazapines are the most commonly used drugs

3. Inhalation Conscious Sedation, Nitrous Oxide/Oxygen (laughing gas) is the common technique used sedation method used in dentistry. Nitrous oxide gas is no stranger to either anaesthesia or dentistry and its use links both sciences in history. The technique in which low concentrations of nitrous oxide gas is titrated with oxygen has been used for years (as early as 1889) in many countries (USA, Great Britain, Australia, and Scandinavia) and is recognised as clinically successful and cost effective compared to General Anaesthesia.The European Academy of Paediatric Dentistry, the American Academy of Paediatric Dentistry and the British Society of Paediatric

Dentistry all recommend a "titration" technique that involves increasing the dose of N2O in oxygen by 5 to 10% increments in the oxygen mix every 1 minute or so and according to the patient's response until the desired sedative effect is achieved.

1) Inability to communicate; 2) inability to nose breath; 3) severe psychiatric or Behavioural/personality disorders; 4) B12 or folate deficiency/disorders; 5) chronic obstructive pulmonary disease (COPD); 6) neuromuscular disorders, e.g. multiple sclerosis; 7) cancer undergoing chemotherapy with Bleomycin drugs; and 8) in patients during the first trimester of pregnancy.

Advantages:

- Patient relaxation with increased comfort
- Patient co-operation and control of gaga reflex
- Little or no memory of treatment
- Time saving - Fewer appointments needed

Conclusion :

In conclusion Sedation dentistry and the coming of the new breed of sedationists or sedation dentists could help in better treatment deliver across private practices . But the cost of equipment as well as trained assistance is a major concern.

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Hereditary ectodermal dysplasia: A case report

Dr.Vinuta S |Department of Prosthodontics |Maharishi Markendeshwar College of dental sciences and Research |Mullana |Ambala
Dr.Manjunath S M |Department of Oral Pathology Maharishi Markendeshwar College of dental sciences and Research |Mullana |Ambala
Dr.Venkateswar Rao Chava |Dept of Oral & Maxillofacial Surgery | Rama Dental College & Research |Kanpur

Abstract:

Ectodermal dysplasia is an extremely rare genetic disorder, in which abnormal development of ectodermal structures occurs. It is inherited as X–linked recessive trait, in which gene is carried by females and manifested in males. Different combination of defects may give rise to variable phenotypes of this disorder. The tissues in which the primary defects occur are the skin, hair, nails, teeth and exocrine glands. Hypotrichosis, hypohydrosis and hypodontia are the prominent clinical features of this syndrome. Case report of patient suffering from ectodermal dysplasia, who presented with hypodontia and other clinical features, is presented in this article.

Key-words: Ectodermal dysplasia, Hypodontia, Hypotrichosis, Hypohydrosis, Saddle nose.

Introduction:

CASE REPORT

Ectodermal dysplasia is a hereditary disease characterized by deformity of atleast two or more of the ectodermal structures like hair, teeth, nails and sweat glands. More than 170 distinctive syndromes have been described with all possible modes of inheritance. It is a relatively rare disorder with the variation in frequency of 1: 10,000 up to 1: 100,000 of those born alive[1, 2].

Thurman published the first report of a patient with ectodermal dysplasia in 1848 and Weech was the first to call it by the present term in 1929[3]. It is typically inherited as a cross-linked recessive trait so that the frequency and severity of the condition is more pronounced in males than in females[4].

Clinical features include anodontia or hypodontia, defective nails, dry skin, depressed nasal bridge, prominent forehead, sparse, fine blond hair with abnormal texture of the scalp, eyelashes and eyebrows, and protuberance of the lip which will show dryness & cracks[1].

The classic form of ectodermal dysplasia (Christ-Siemens-Touraine syndrome) is thought to be X-linked and involves hypodontia, hypohydrosis, hypotrichosis, and a characteristic facies.

Case report

A 30 year old male, A known case of ectodermal dysplasia was presented to our clinic for evaluation of teeth abnormalities. The patient gave a history of delay in the eruption of permanent teeth, less sweat production and intolerance to heat.

On extra oral examination, the patient had sparse hair on the body and scalp. Hairs present were fine in texture & color. A prominent supraorbital ridge, small and outwardly placed ears and flattened nasal bridge were present. Both upper and lower eyelids showed sparse eyelashes and eyebrows were thin. The skin was warm and dry. Other facial features were frontal bossing, saddle nose, thick everted lips (Figure 1).

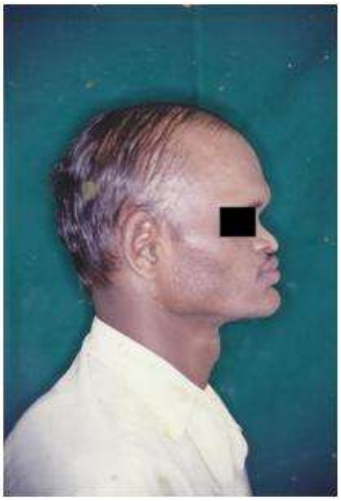

Figure-1Extraoral features showing hypotrichosis, saddle nose, everted lips.

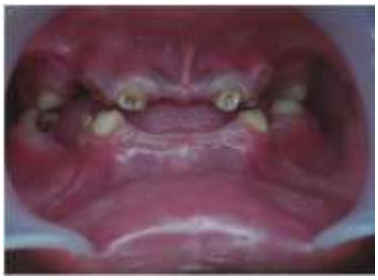

Figure-2 Intraoral features showing hypodontia, conical teeth and underdeveloped maxillary and mandibular ridges

Intra oral examination revealed multiple missing teeth in both the arches. Maxillary arch showed the presence of six teeth, while in the mandibular arch there were five teeth present. Both maxillary and mandibular arches were underdeveloped due to the absence of teeth. Teeth present were cone shaped, smaller in size and showed abnormal central cavity formation on the incisal or occlusal surfaces (Figure 2). Patient was referred to prosthodontist for oral rehabilitation.

Discussion

Ectodermal dysplasia is a hereditary condition characterized by abnormal development of the skin, hair, nails, teeth, and sweat glands, cranial-facial structure, digits and other parts of the body. Individuals with this disease suffer from hypodontia, hypotrichosis & Hypohydrosis.

Ectodermal Dysplasia is not a single disorder, but a group of closely related conditions. More than 1700 different

syndromes have been identified. Multiple genes have been discovered that cause ectodermal dysplasias. Most common mode of inheritance of ectodermal dysplasia is X-linked, whereas both recessive and dominant forms also exist. Autosomal recessive disorders are phenotypically indistinguishable from the X-linked forms. Each person with ectodermal dysplasia may have a different combination of defects. One may have hair and nails affected, while another may show involvement of sweat glands and teeth. Each combination is considered a distinct type of ectodermal dysplasia.

Ectodermal dysplasias are rare diseases with an estimated incidence of seven in 100,000[5]. The pattern of inheritance is different, including Mendelian modes and sporadic cases. Several classifications have been proposed from a clinical point of view, with molecular genetic attributes and based on identified causative genes that most often are involved in processes of intercellular communication and signalling[6].

The two common type of ectodermal dysplasia are hydrotic and hypohydrotic form. Hydrotic ectodermal dysplasia is autosomal dominant in nature and patients present with a scalp hairs which are soft, dawny and darker in color with anodontia or hypodontia. Other features hydrotic types are dystrophic nails, eyebrows that are frequently absent and eyelashes, pubic and auxiliary hairs will be scanty or absent. Lips, sweat gland and nasal bridge are normal in this patient. Whereas, hypohydrotic ectodermal dysplasia is autosomal recessive in nature. Scalp hairs will be fine in texture, fair and short. Other abnormalities presented by patient are anodontia or hypodontia, lip protrusion, reduction or absence of sweat glands, underdeveloped nasal bridge, absence of eyebrows and variably affected eyelashes, pubic and auxiliary hairs. Nails will be normal in this type[7].

Genetic studies of more than 300 cases have revealed X linked mode of inheritance with its gene locus being Xq11-21.1, the gene is carried by the female but manifested in the male. Mutations in EDA, EDAR and EDARADD genes are now identified to cause hypohydrotic ectodermal dysplasia. These genes provide instructions for making proteins that work together during embryonic development. Ectoderm-mesoderm interaction is essential for the formation of several structures that arise from the ectoderm including skin, hair, nails, teeth and sweat glands. Mutation in the EDA, EDAR or EDARADD gene prevents the normal interaction between ectoderm and mesoderm and impairs the normal development of hair, sweat glands and teeth[8].

Manifestations of the disorders are not clinically apparent in newborns, as they become prominently evident during infancy or early childhood. Freire Maia and Pinheiro published an exhaustive review and classification system for these disorders using a numeric system of 1 (hair), 2 (teeth), 3 (nail), 4 (sweat glands) for characterization[9].

Severe hypodontia or even anodontia in children are very rare conditions, most often associated with congenital syndromes such as Down syndrome or ectodermal dysplasia. More than 170 clinically distinct hereditary syndromes in which ectodermal

dysplasias are noticed throughout the world [1].

The essential features are reduced or absence of sweating, hypotrichosis and total or partial anodontia. The patients with this disorder have facies suggestive of congenital syphilis. The cheek bone are high and wide, with prominent frontal ridges and chin, saddle nose, sunken cheeks, thick everted lips, large ears and sparse hair. The skin is smooth, soft, dry, wrinkled especially around eyes and appears prematurely aged. Other symptoms should be noticed in these patient are abnormal nails, abnormal or missing teeth, absent or very thin hair, absent or decreased tears, decreased skin pigment, foul-smelling nasal discharge, heat intolerance, large forehead, low nasal bridge, poor hearing, poor temperature regulation, poor vision and thin skin[1,4,10].

The deciduous to permanent teeth may be rarely fully absent or reduced. The conical pointed teeth are key feature of the syndrome and may be the only obvious abnormality. Usually incisors and/ or canines are characteristically affected. The scalp hair is usually sparse, fine and blonde. Alopecia is often the first feature to attract but it is seldom total.

Absent or reduced sweating causes heat intolerance and affected individuals may present with unexplained fever in infancy or childhood. Mental retardation reported in 30 – 50% of cases and is believed to be due to damage from prolonged fever and febrile seizures.

The diagnosis is usually made with the identification of hypotrichosis, characteristic facial features, hypohydrosis and rarely anhydrosis, and teeth abnormalities. Abnormalities in the development of tooth buds result in hypodontia and peg-shaped or pointed teeth. The teeth are smaller than average, and the eruption of teeth is often delayed. The extent of hypodontia may be useful in assessing the severity of the disease[1, 4, 11.]

There is no specific treatment for this disorder. Supportive treatment is indicated to the patient that includes supplementation of denture by Prosthetic treatment in adults for mastication and speech. Importantly, aesthetic dental interventions in patients and malformed teeth and malocclusion helps with the development of a positive self-image12, wig can be suggested to improve appearance, artificial tears to replace normal tearing and prevent drying of the eyes, saline nose spray for nostrils to remove debris and prevent infection. Patients should be advised to live in a cooler climate and take cool water baths or use water sprays to keep a normal body temperature. However, external cooling is less effective in these patients as heat transfer from the core to the skin is reduced, probably due to poor capillary dilation[13].

Conclusion

Having ectodermal dysplasia will not shorten the life span of the patient, but constant attention to temperature regulation and other problems including dental problems associated with this condition is necessary.

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CYTOTOXICITY OF DENTIN BONDING AGENTS- REVIEW

Dr.Rukhsar Amjad Vankani |Mumbai |INDIA

Abstract

Cytotoxicity is the extent of the destructive or killing capacity of an agent. Dentin bonding agents; are applied in close proximity to dentin and pulp, so the effect of the dentin bonding agent on the cells of dentin and pulp are of importance as micro leakage and post-treatment sensitivity have been persistent problems when composite resin is bonded to tooth structure also the direct biological risks of left out monomers are of great importance.

Keywords: Dentin bonding agents, cytotoxicity, biological risks

Introduction

This review concentrates only on the effect of dentin bonding agents on biological tissues and the risks of slightly cured or incompletely cured dentin bonding agents. Since, they are applied in very close proximity to the pulp, they should be very biocompatible; but that is not possible in general situations. There are lot of factors in play after the dentin bonding agent has been applied and set to cure, like the contents of the dentin bonding agent, the salivary enzymes, the moisture in the mouth, the chances of contamination, insufficient or incomplete curing cycles, etc. This review briefly discusses them.

Discussion

Biological risks are complex and interactive and still completely defined. The risks are classified as Toxicological properties or Direct risks and Microbiological leakage or Indirect risks. Direct risks are reported to have systemic estrogenic effects and many elicit allergic reactions and might act as carcinogens.

Dentin bonding agents contain monomers similar to composites but mostly all contain Hydroxy Ethyl Methacrylate (HEMA). HEMA is amphoteric and displaces water in dentin but also miscible with most monomers of composites. Dentin bonding agents are biocompatible as they are placed closest to the pulp, but only if they are completely polymerized which is probably just theoretical. Dentin bonding agents are at greater risk to incomplete curing since they are thin an oxygen inhibition of polymerization is significant factor. Even, light cured adhesives require full density curing with the light on very thin increments. Although thin increments of 1-3mm are used, but still complete polymerization never happens, as much as

25-50% of methyl methacrylate monomer double bonds actually remain unreacted in polymer. Any unpolymerized monomer is potential biological liability if it leaches from composite towards pulp of the tooth. Extracellular salivary enzymes may degrade polymerized networks over time marking hydrolyze products available to tissues.

The cytotoxicity of these materials can be tested by, dose response assessment. This assessment is achieved with in vitro cytotoxicity tests, tests for inflammation, tests for immune response, genotoxic (mutagenicity), and, finally, gene expression in odontoblast-like cell lines.

The second step in risk assessment is to determine the doses of the chemicals that will be released by the material. For adhesive resins, a "dentin-barrier test" has been developed to determine the concentrations of components of dental materials that might reach pulpal tissues. The second tier of tests also includes intracutaneous reactivity, skin sensitization, and dental usage tests. Characterizing the risk constitutes the final step of the process. The dose response is compared with the estimated dose exposure: If the dose to cause an adverse response is greater than the estimated exposure by a comfortable safety margin, the likelihood of an adverse event occurring in an exposed population is small, and the material may be deemed to have a low risk of biological problems. Although a few in vivo studies have attempted to document the biological risks of resin-based materials, most information on the hazards posed by the components of resin based restorative materials has been gained from in vitro studies. As early as 1991, Hanks et al. reported the toxic concentrations of 11 components of dental resins on mouse fibroblasts. Later, Ratanasathien et al. (1995) evaluated the effects of simultaneous exposures of cells to several resins. They demonstrated the additive cytotoxic effects produced by HEMA when used as a solvent for BisGMA. The synergism between these 2 molecules has been shown to affect the apparent toxicity of each individual resin component for the cultured cells. These unique experiments established that resins or combinations of resins alter fibroblast mitochondrial activity. Rakich et al. (1999) demonstrated that resin monomers are also a hazard to inflammatory cells that are common in the pulpal tissue, and Noda et al. (2003) have shown that resins alter the secretion of inflammatory mediators from human macrophages. Other studies have shown that HEMA is able to diffuse rapidly through dentin in vitro in sufficient concentrations to cause cytotoxicity (Bouillaguet et al., 1996), and that bonding agents, as used clinically, elute sufficient amounts of monomer through dentin to cause significant cellular toxicity after 1 wk (Bouillaguet et al., 1998). The persistent cytotoxicity observed after 1 wk reinforced the need for evaluation of the long-term effects of the resin monomers on cellular systems. Indeed, long-term studies that used sublethal concentrations of HEMA (Bouillaguet et al., 2000a), TEGDMA, or BisGMA (Lefebvre et al., 1999) for 5-6 wks showed that resins clearly altered cellular mitochondrial activity and total protein content per cell, even at concentrations of 1-10% of those used in short-term experiments. These results confirmed that risk assessment of dentin adhesives must also be considered with a long-term view.

Conclusion

Different generation of Dentin bonding agents have been introduced since the past decade, from separate etchant and primer we have come to the one step bonding agents. Comparative studies have been carried out to test which is more cytotoxic and the results suggest that one step dentin bonding agents are less cytotoxic as their pH is as low as 1.0. Evidence exists with the application of acids on dentin; the permeability increasing effect of dentin etching is dependent on several factors, e.g. dentin thickness of 0.5 mm and higher, no significant effect was measured. Correspondingly, no pulp reactions have been reported after the use of dentinbonding, including acid treatment, if the pulp was covered by an intact dentin layer.However, low pH acids (e.g. phosphoric acid) are applied only for a short time-period (up to 30 s) and are then rinsed away. Acidic monomers, however, remain on the dentin.

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