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ORIGINAL RESEARCH ARTICLE

PREVALENCE OF NOMOPHOBIA AMONG STUDENTS, INTERNS AND FACULTY IN A DENTAL COLLEGE IN KERALA

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ABSTRACT

Background: Nomophobia is defined as the fear of being out of mobile phone contact and is considered a modern age phobia. With the advent and prolific use of smart phones by people, the addiction levels have also concomitantly increased. This study was conducted with the objective of assessing the prevalence of Nomophobia among dental students, house surgeons and teaching faculty of a dental college in Kerala.

Methodology: The study was a cross-sectional questionnaire based survey. The target population was the clinical dental students, house surgeons and teaching faculty of a dental college Kerala. The Nomophobia Questionnaire developed at The Iowa State University, herein after referred to as NMP-Q validated by Caglar Yaldirim and Ana- Paula Correia was used in the study. Results were expressed as a number and percentage of respondents for each question. Chi-square test was performed to compare the response in relation to year of study and designation.

Results: Among the 153 respondents, 84 were students, 45 interns and the rest 24 were faculty members. About 96% (n=147) were smart phone users. About 95% of the respondents had an access to internet on their mobile phone. The present study showed that – 2% had no nomophobia, 39.2% had mild nomophobia, 56.2% had moderate nomophobia and 12.4% had severe nomophobia. There was a statistically significant difference in nomophobia levels among teaching faculty, house surgeons and students (p=0.042), where teaching faculty and house surgeons had significantly greater levels of nomophobia compared to the students.

Conclusion: Present study revealed that nomophobia was highly prevalent among the respondents. The most common reasons for smart phone use were calling of family members and friends.

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INTRODUCTION

Nomophobia is defined as the fear of being out of mobile phone contact and isconsidered a modern age phobia introduced to our lives as a by-product of the interaction between people and mobile information and communication technologies, especially smartphones.[1] In olden times, individuals were dependent on phone just for communication purpose but now they have a thirst for it due to countless benefit it provides.[2] In recent times there seems to have been a transformation of the cell phone from a status symbol to a necessity because of the countless perks that a mobile phone provides like personal diary, email dispatcher, calculator, video game player, camera and music player.[3]

As per Telecom Regulatory Authority of India (TRAI), there are about 980.81 million mobile phone subscribers in India making it the world's second largest mobile phone user country in 2015 (TRAI, 2015) [2.] Mobile phones are very fascinating for younger generations, as it gives them a feeling of autonomy, identity and credibility. Besides being just a entertainment object, it helps to keep them in constant contact with their family and friends. Smartphone is popular device capable of processing more information than other mobile phones, making it possible to perform variety of tasks like voice calling, texting people, surfing the Internet, social networking, gaming, for entertainment, etc. Access to the internet is increasingly easy due to improvements in mobile technology and the prevalence of smartphones use. [4]

With the advent and prolific use of smartphones by people the addiction levels have also concomitantly increased. A mental impairment resulting from modern technology has come to the attention of sociologists, psychologists, and scholars of education on mobile addiction. Mobile phone addiction and withdrawal from mobile network may increase anger, tension, depression, irritability, and restlessness which may alter the physiological behaviour and reduce work efficacy. [5]

However the phobia caused due to being out of contact of mobile phone (esp. smart phone), termed as Nomophobia is a relatively new concept. Hence this study was conducted with the objective of assessing the prevalence of nomophobia among dental students, interns and faculty of a dental college in Kerala.

METHODOLOGY

The study was a cross-sectional questionnaire based survey. The target population was the clinical dental students (Third year and Final Year undergraduate students), House surgeons and teaching faculty of Indira Gandhi Institute of Dental Sciences, Nellikuzhy, Kothamangalam, Kerala. The Nomophobia Questionnaire developed at The Iowa State University, herein after referred to as NMP-Q validated by Caglar Yaldirim and Ana-Paula Correia was used in the study.

The questionnaire was divided into two parts. The first part consisted of questions on professional data designation and grade, and information regarding usage of smart phones, duration, frequency and time of usage of smart phones. The second part contained 20 closed ended questions. NMP-Q, used for assessing Nomophobia, consists of self-reported scores (from 1 to 7) ranging from strongly disagree (1) to strongly agree (7), for all 20 questions. The final score per person is interpreted as: 20 (Nomophobia absent), 21-60 (Mild Nomophobia), 61-100 (Moderate Nomophobia) and 101-140 (Severe nomophobia).

The questionnaires were distributed by the house surgeons posted in the Department of Public Health Dentistry. The respondents filled the questionnaire on their own and were asked to return the questionnaire immediately.

Necessary ethical clearance for the study was obtained from the Institutional Ethical Committee. The respondents were briefed about the study and informed consent was obtained from all the participants prior to the administration of questionnaire. The final study sample was 153.

Statistical analysis

All returned questionnaires were coded and analysed. Results were expressed as a number and percentage of respondents for each question and were analysed using the SPSS Version 17 software. Chi-square test was performed to compare the response in relation to year of study and designation; and the level of significance was set at $p = 0.05$.

Table 1: Response to Nomophobia Questionnaire (response in %)

		Strongly Disagree						Strongly Agree
		1	**2**	**3**	**4**	**5**	**6**	**7**
1	I would feel uncomfortable without constant access to information through my smartphone.	22.2	14.8	8.9	25.9	15.6	6.9	6.7
2	I would be annoyed if I could not look information up on my smartphone when I wanted to do so.	14.2	11.2	10.4	14.2	20.9	11.9	17.2
3	Being unable to get the news (e.g., happenings, weather, etc.) on my smartphone would make me nervous	36.6	17.9	13.4	9.0	11.2	6.0	6.0
4	I would be annoyed if I could not use my smartphone and/or its capabilities when I wanted to do so	19.0	8.7	7.9	14.3	23.8	12.7	13.5
5	Running out of battery in my smartphone would scare me.	19.2	20.0	16.9	14.6	8.5	10.8	10.0
6	If I were to run out of credits or hit my monthly data limit, I would panic.	37.1	12.9	15.9	12.9	9.8	3.0	8.3
7	If I did not have a data signal or could not connect to Wi-Fi, then I would constantly check to see if I had a signal or could find one	16.3	13.3	8.1	16.3	15.6	15.6	14.8
8	If I could not use my smartphone, I would be afraid of getting stranded Somewhere	33.8	13.5	12.8	15.8	11.3	6.8	6.0
9	If I could not check my smartphone for a while, I would feel a desire to check it.	17.2	9.7	7.5	19.4	16.4	13.4	16.4
	If I did not have my smartphone with me,							
10	I would feel anxious because I could not instantly communicate with my family and/or friends.	12.8	9.8	11.3	18.0	17.3	13.5	17.3
11	I would be worried because my family and/or friends could not reach me.	9.2	9.9	5.3	20.6	22.1	17.6	15.3
12	I would feel nervous because I would not be able to receive text messages and calls.	16.9	12.3	7.7	20.0	16.9	11.5	14.6
13	I would be anxious because I could not keep in touch with my family and/or friends.	11.4	9.1	9.8	15.9	22.7	11.4	19.7
14	I would be nervous because I could not know if someone had tried to get a hold of me.	20.9	10.9	14.7	16.3	18.6	8.5	10.1
15	I would feel anxious because my constant connection to my family and friends would be broken.	19.1	13.7	11.5	19.1	14.5	11.5	10.7
16	I would be nervous because I would be disconnected from my online identity.	40.2	14.4	16.7	11.4	5.3	4.5	7.6
17	I would be uncomfortable because I could not stay up-to-date with social media and online networks.	30.3	11.7	12.1	14.4	6.8	11.4	8.3
18	I would feel awkward because I could not check my notifications for updates from my connections and online networks.	30.3	19.7	13.6	9.8	9.8	8.3	6.1
19	I would feel anxious because I could not check my email messages.	39.2	15.0	18.0	8.3	4.5	6.0	9.0
20	I would feel weird because I would not know what to do.	42.7	6.9	11.5	16.0	9.9	1.5	11.5

RESULTS

Among the 153 respondents, 84 were students, 45 interns and the rest 24 were faculty members. About 96% (n=147) were smart phone users. About 95% of the respondents had an access to internet on their mobile phone.

Table 1 shows the summary of the response to the Nomophobia Questionnaire scale. Only about 12% of the respondents strongly agreed that they were uncomfortable without constant access to their smartphone. However 50% strongly felt annoyed if they could not look up any information on their smart phone whenever they wanted to. Over half the number of respondents disagreed that being unable to get news (happenings, weather etc.) would make them nervous. About 20% strongly agreed that running out of battery would scare them. More than 50% agreed that they constantly checked their data signal /Wi-Fi signal when their signals were lost. Only about 35% felt that they wouldn't have a desire to check their smart phone if not checked for a while.

When asked if the respondents did not have smart phones with them, about 47% strongly agreed that they felt anxious that they could not instantly communicate with family/friends, and about 55% strongly agreed that they would be worried as family/friends could not reach them. Majority of the respondents disagreed that absence of smart phones would make them nervous as they could not know if someone tried to get hold of them and also that their online identity would be lost. About half the number felt that the felt uncomfortable as they could not stay up to date with social media and online networks. Only about 18% felt that they would feel anxious as they couldn't check their e mails on phone.

NOMOPHOBIA SCORE AND INTERPRETATION

Score	Interpretation	(n)	Percentage
20	Absent	3	2%
21-60	Mild Nomophobia	60	39.2%
61-100	Moderate Nomophobia	71	46.4%
101-140	Severe Nomophobia	19	12.4%
TOTAL		**153**	**100%**

There was a statistically significant difference in nomophobia levels among teaching faculty, house surgeons and students (p=0.042), where teaching faculty and house surgeons had significantly greater levels of nomophobia compared to the students.

DISCUSSION

Mobile phones have become integral part of our life. It is a well-known fact that the number of subscribers for mobile phones have been drastically increasing past few years. The number of internet users in India is expected to reach 500 million by June 2018, according to the Internet and Mobile Association of India (IAMAI).[6] Eighty-eight per cent of households in India today have a mobile phone, according to the 'Household Survey on India's Citizen Environment & Consumer Economy' (ICE 360° survey) conducted in 2016.[7]

Concomitantly, during the last few years, newer disorders have been discovered associated with mobile phone usage, nomophobia being one of them. Nomophobia is affected by various factors and their assessment would definitely help in predicting its outcome. Increase in mobile phone dependence, can result in increased internet addiction and vice versa.

It is a matter of fact that technology can convey information to people without any difficulty. However, over usage of mobile phones may cause psychological illness such as dry eyes, computer vision syndrome, weakness of thumb and wrist,

neck pain and rigidity, increased frequency of De Quervain's tenosynovitis, tactile hallucinations, nomophobia, insecurity, delusions, auditory sleep disturbances, insomnia, hallucinations, lower self-confidence, and mobile phone addiction disorders.[5] More importantly, studies conducted by Baghianimoghadam MH et al., in 2013, Krithika M et al., in 2013, Aman T et al., in 2015 and Chen YF in 2006 show that excess use of the mobile phones can have negative results on academic performance among students.[8-11] Hence, this study was undertaken to assess the nomophobia levels among students and staff in a dental teaching institution in Kerala.

The present study showed the following results – 2% (no nomophobia), 39.2% (mild nomophobia), 56.2% (moderate nomophobia) and 12.4% (severe nomophobia). The results are comparable to similar study conducted among medical students in Wayanad in 2017 by Madhusudan M et al., where the corresponding figures were 3%, 33.3%, 56.2% and 7.5% respectively.[12] The prevalence of nomophobia in a study conducted in a dental college in Thodupuzha by Abdul Saheer et. al. in 2017 revealed a prevalence of 35.4% as nomophobic[13], also a study by Prasad et al. in Modinagar revealed a prevalence of 24.12% with nomophobia and another 41% at risk of nomophobia [2]. The difference could be due to the scale used for assessment of nomophobia where it is interpreted as absence of nomophobia, at risk of nomophobia and nomophobic. Comparison with few other studies show that prevalence reported among interns of medical students in Indore by Sharma N et. al. in 2015 was 73% and college students in Bangalore by Masthi NR was 67%. However, the results are in sharp contrast with the results of a study conducted by Bivin JB in 2013 in Kerala, where the nomophobia prevalence was reported as 23%. The observed difference could be due to the fact that it was conducted about 6 years ago, when internet usage and addiction was not so rampant as today.

Only 3.2% of the respondents admitted of using mobile phone during or in between classes. This is in contrast to study conducted by Prasad et. al. in Modinagar[3], where 24.7% agreed and 6.9% strongly agreed that they frequently checked their cell phoned during class or clinics. The difference might be due to the ban on usage of mobile phone by students during college hours.

It's an important observation that majority of the study samples felt annoyed when they could not get information on their smart phone/use smart phone when they wanted to. However when questions regarding feeling uncomfortable without constant access to information via smartphone, inability to get the news and weather report, panic following running out of data, afraid of getting stranded due to loss of smartphone; were asked many disagreed to having an issue with the situation. The results are similar to Thodupuzha study conducted among dental students[13].

About 27% agreed that running out of battery scared them. This is similar to the results of Thodupuzha study [13,] and in contrast to the Modinagar study where the corresponding figures were about 32% and 60% respectively.

The most common reason for the use of smartphones in our study was calling of family members which were similar to the findings of other studies. [12-14]

Given the ever-increasing amount of time peoplespend using technology, and the potential deleterious effects such increase can have on health, the present study's investigation on mobile phone dependence pattern and the prevalence of Nomophobia is critically important. Mobile phone usage is not only habit forming, it is also addictive; "possibly the biggest non-drug addiction of the 21st century"[15].

The limitations of the study are that it is based on medical students of one particular college only and hence it cannot be used to generalize the prevalence. The results also rely upon the presumption that the students gave real responses to the self-administered questionnaire.

CONCLUSION

Present study revealed that nomophobia was highly prevalent among the respondents. Nomophobia was not found to be significantly higher among teaching faculty and house surgeons compared to students. The most common reasons for smartphone use were calling of family members and friends.

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BIOCERAMICS IN ENDODONTICS
A REVIEW

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ABSTRACT

Bioceramic materials, since their introduction in Endodontics have significantly changed the prognosis of many cases which were once considered impossible. These materials find wide applications, as in direct pulp capping, apexification, apexogenesis, root repair, root canal sealants, etc. They are also widely used as retrograde filling materials and as bone grafts to enhance bone healing. Ever since its introduction, continuous researches have been going on in this field to introduce better products, providing better biocompatibility and handling properties. This review gives an overview on some of the major Bioceramic materials used in Endodontics, their advantages, properties and applications.

Key words- Biomaterial, Bioceramics, Endodontics.

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INTRODUCTION

A Biomaterial is used to make devices to replace a part or a function of the body in a safe, reliable, economic, and physiologically acceptable manner. The Clemson University Advisory Board for Biomaterials has formally defined a biomaterial to be "a systemically and pharmacologically inert substance designed for implantation within or incorporation with living systems" [1].

The ideal requisite of Biomaterials is that it should be Noncarcinogenic, Bacteriostatic, should not discolor tooth, and promote Cementogenesis, Osteogenesis & healing[2].

The field of Biomaterial science has made giant leaps in the last five decades, due to the extensive research that has been going on in the field. The emphasis was always in developing newer and more biocompatible materials that can replace or develop the body tissues.

Bioceramics is a diverse class of biomaterials. The class of ceramics used for repair and replacement of diseased and damaged parts of musculoskeletal systems are termed bioceramics[3]. Bioceramics in Endodontics find wide applications as in direct pulp capping, apexification, apexogenesis, root repair, root canal sealants, etc. In Periapical surgeries, also they are used as retrograde filling materials and as bone grafts to enhance bone healing.

I. PORTLAND CEMENT:

Portland cement is a hydraulic binding material which when mixed with water tends to harden[4]. It is widely used in building industry. However it also finds wide application in dentistry. The main interest of its use in dentistry is as an alternative to MTA, as it is less expensive and widely available. These two materials show high similarity in their composition, except for the bismuth oxide present in MTA which confers Radiopacity.

It was in the year 1824, that Joseph Aspdin patented this product so-called Portland cement (PC) which was actually obtained from the calcination of the mixture of lime stones coming from Portland in England and silicon-argillaceous materials[5]. Portland cement is used in dental procedure such as pulpotomy, pulp capping, repair of root perforations and root end filling. Sakai et al compared the clinical and radiographic effectiveness of MTA and PC as pulp dressing agents in carious primary teeth. No statistically significant difference regarding dentine bridge formation was found between both groups throughout the follow-up period[6].

II. MINERAL TRIOXIDE AGGREGATE

Mineral Trioxide Aggregate (MTA) was introduced in the year 1993 by Mahmoud Torabinejad. It was later patented and marketed in the year 1995 and was given approval for use in endodontics in 1998. MTA is actually believed to have been derived from ordinary Portland cement or in fact a modification of the Portland cement. Modifications have been made in designing this wonderful material, especially with respect to particle size, setting rate, solubility and possibly toxicity (by reducing the heavy metal content).

MTA is called as hydraulic cement, mainly because it primarily depends on hydration reactions for its setting reaction, compared to other cements which depends on acid base reactions[7]. MTA constitutes the following chemical composition or phases, tricalcium silicate, dicalcium silicate, tricalcium aluminate, tetra calcium aluminoferrite, calcium sulfate dehydrate, bismuth oxide, alkali metal oxides and sulfates[8]. MTA is available in two forms as grey and white MTA. Main difference in white MTA was the elimination of alumino ferrite that is responsible for grey color, which in fact affected the esthetic part.

MTA find wide application in dentistry as in, Direct pulp capping, Pulpotomy, Repair of root perforation, Retrograde root canal filling, Apexogenesis, Apexification, Repair of root resorption and Regenerative procedures. The high alkalinity induced by MTA initially causes necrosis of pulp followed by dentin deposition[9]. It was also found that MTA caused reparative dentin formation in a very short period of time compared to calcium hydroxide. A 100% success rate was obtained in the case of teeth with open apices when MTA was used. Bacteriostatic action provided by calcium hydroxide and MTA was also found to be the same in studies. MTA is also used widely as a retrograde filling material due to the high biocompatibility provided by the material. MTA gives better results when tested for leakage and biocompatibility than IRM and Super EBA, and has the ability of induction of hard tissue[10]. MTA has been widely used as the material of choice in regenerative/ revascularization procedures owing to the high pH and biocompatibility provided by the material. MTA modulates cytokine production and encourages differentiation and migration of hard tissue producing cells whereby hydroxyapatite is formed on the MTA surface, and a biologic seal is created[10]. Latest studies have shown that MTA-enriched nanocomposite TiO2-polymeric

powder coatings support human mesenchymal cell attachment and growth[11].

III. CALCIUM ENRICHED MIXTURE OR NEW ENDODONTIC CEMENT

Calcium Enriched Mixture (CEM) is a newly introduced material, which is chemically similar to Portland cement and Mineral Trioxide Aggregate. The material was developed by Saeed Asgary.

In vitro studies conducted to compare the compositions of mineral trioxide aggregates (MTAs), Portland cements (PCs), and a new endodontic cement (NEC) revealed that NEC differs chemically from MTAs and PC. Phosphorous is the major component of NEC, whereas in MTAs and PCs this element is close to the detection limit. It was also seen that in contrast with MTA, NEC showed surface composition similar to surrounding dentin[12]. CEM just like MTA and Portland cement is found to produce an alkaline environment with a pH ranging from 9-11.5. Clinical uses of NEC are similar to MTA. It has good handling characteristics and forms an effective seal when used as root-end–filling material. The studies conducted showed that Mineral trioxide aggregate and CEM cement were associated with a similar favorable biological response to pulpotomy treatment and demonstrated a more effective induction of dentinal bridge formation compared to Calcium Hydroxide[9].

IV. BIODENTINE

Biodentin is a new bioactive calcium silicate-based cement that has been recently launched in the dental market as a 'dentin substitute'. Introduced by Septodont, this material claims to make use of a proprietary 'Active Biosilicate technology'[13]. Biodentin has been formulated using MTA based cement technology and hence; claims improvements of some of the properties such as physical qualities and handling, including its other wide range of applications like endodontic repair and pulp capping in restorative dentistry.

The product file of Biodentine states that the powder component of the material consists of tricalcium silicate, dicalciumsilicate, calciumcarbonate and oxide filler, iron oxide shade, and zirconium oxide. Tricalcium silicate and dicalcium silicate are indicated as main and second core materials, respectively, whereas zirconium oxide serves as a radiopacifier. The liquid contains calcium chloride as an accelerator and a hydrosoluble polymer that serves as a water reducing agent. The hydrosoluble polymer reduces the viscosity of the cement and improves handling[14].

The setting time of the material is around 9-12minutes, which is much faster compared to MTA which takes around 165 minutes for initial setting. During the setting of Biodentine, the compressive strength increases 100 MPa in the first hour and 200 MPa at 24th hour and it continues to improve with time over several days until reaching 300 MPa after one month which is comparable to the compressive strength of natural dentine i.e. 297 MPa. Relatively easier manipulation and faster setting is the major advantages of this material when compared to MTA. Like MTA Biodentine find wide application in dentistry like direct and indirect pulp capping, as a dentine substitute under composite, as an endodontic repair material, retrograde root filling, and apexification.

V. BIO AGGREGATE

BioAggregate (Verio Dental Co. Ltd., Vancouver, Canada) is composed of nano particle sized Tricalcium silicate, tantalum oxide, calcium phosphate, silicon dioxide and presents improved performance compared with MTA. Tricalcium silicate is the main component phase, tantalum oxide is added as a radiopacifier and it is free of aluminum[15].

The manufacturers claim this material to have an Aluminum-Free Composition so that health problems due to aluminium toxicity are avoided. It also claims to have excellent biocompatibility with the vital peri radicular tissue. BioAggregate mixture is easy to manipulate and apply. The material complements the natural color of teeth and all the ingredients are pure white in color. Tantalum Pentoxide has been substituted for Bismuth oxide (used in MTA) that provides better radio opacity.

The cytotoxic evaluation of mineral trioxide aggregate and BioAggregate were compared by placing them in the subcutaneous connective tissue of rats and it was found that BioAggregate had better inflammatory and foreign body reaction than the MTA group[16]. Studies have shown that the material is non toxic to osteoblasts and has the ability to induce mineralization-associated gene expression in osteoblasts. Presently BioAggregate is manufactured by two companies Veriodental and Diadent. The trade names are respectively IBC BioAggregate and Dia Root BioAggregate. BioAggregate is more biocompatible, has better sealing ability, higher fracture and acidic resistance than MTA 50.

VI. BC SEALER

BC sealer is a revolutionary premixed and injectable root canal sealer utilizing new bioceramic

nanotechnology. The material is introduced and marketed by Brassler USA.

This material claims to bond with both dentin and the treated gutta percha (which is provided by the manufacturer). The material claims to provide excellent sealing through the monobloc concept by bonding to both dentin and gutta percha[18]. According to the manufacturer the material contains Zirconium oxide, calcium silicates, calcium phosphate monobasic, calcium hydroxide, filler and thickening agents.

CONCLUSION

Bioceramics has evolved to become an integral and vital segment of our modern health-care delivery system. The beginning of the era of Bioceramics started off with the discovery of Bioglass In 1969 by Larry Hench. Since then the field of Bioceramics has been undergoing constant research and refinement to provide with newer materials that exhibits better biocompatibility and bone forming properties. The idea was always to improve the composition, microstructure, and molecular surface chemistry of various types of bioceramics to match the specific biological and metabolic requirements of tissues or disease state. The biological activity of Bioceramics has to be understood thoroughly through various in vitro and in vivo studies to improve their biocompatibility, and decrease toxicity and the knowledge on mechanical feature will add up to play a key role for the choice of the bioceramics in their broad implication as implants.

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ISSUE OF DEATH CERTIFICATE- WHAT A DENTIST SHOULD KNOW

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ABSTRACT

Death means the permanent disappearance of all evidence of life at any time after live-birth has taken place. Cause of Death Certification is the official recording of a person's death. It records the cause of death of a person as stated by a doctor or another responsible health worker. The death certificate is a standard proforma (form No. 4), devised by world Health Organization (WHO) and is implemented by Govt of India. This form is devised for the sake of uniformity and comparability of data. Filling up of the form requires utmost care and knowledge of morbid events leading to the death of a person. Revised dentist code of ethics allows dentist to issue death certificate. The awareness among dentists regarding this is less and more focus should be done to increase awareness. This article aims to increase the awareness among dentists regarding death certification.

Keywords: Death; Certification; dentist code of ethics; proforma; awareness.

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INTRODUCTION

Birth and Death are the two most important events in the life of any individual as the person's existence starts at the moment of birth and ceases at the moment of death. A person has legal existence between there corded timings of birth and death[1]. The physician's concept of death is total stoppage of circulation of blood with consequent cessation of vital functions. All these definitions require revision from cessation to permanent cessation of respiration and circulation. Therefore a more appropriate definition would be "death is a permanent and irreversible cessation of functions of three interlinked vital systems of body namely the nervous, circulatory and respiratory systems, the so called tripod of life[2].

In India, with the passing of the Act - Registration of Births and Deaths Act - in 1969, registration of these events is mandatory. Registrar General of India is the highest official who compiles the information about deaths and births received from Registrars & Sub-registrars of Births and Deaths at district level, Municipal Corporations and Municipalities of towns and Cantonment Boards of Military Cantonments.

Examination for somatic death

Auscultate for heart sounds (2min).Auscultate for respiratory sounds (3min). Palpate carotid pulse. Occulo-cephalic test, moving head on both sides. Vestibulo-ocular test using cold water. Apnoea test, Magnus test : placing a ligature round the base of a finger. Diaphanous test : looking through the web of the fingers at a bright light. ECG tracing is confirmatory for death.

Role of medical officer

When confronted with 'death' of a person, the medical officer has two tasks at hand. The first is to diagnose the occurrence of death and declare the person dead. The second is to decide the cause of death and certify the same. Having decided that the person is dead, he proceeds to fill up "Death Report" in the capacity of informant. Both the formats of death report viz Legal Information and Statistical Information, are to be filled up for each death. The second task at hand i.e. issuing Medical Certificate of Cause of Death, is done immediately after deciding the person is dead, by the same medical officer who has declared the person dead, provided the medical officer is absolutely certain of the cause of death and if it is a natural death. Deaths due to old age and deaths due to any naturally occurring disease or its complication are Natural Deaths. Correct knowledge of the cause of death is essential as future course of action is different if the death is other than natural or cause of death is not known/doubtful. If the death is not clearly of natural category i.e. other than natural or cause not known/doubtful, the medical officer having carried out first task i.e. declaration of death, Informs the police of occurrence of the death for further course of action. He will not issue a medical certificate of cause of death. Since the bottom portion of the medical certificate of cause of death is required to be produced by the relatives at the cremation ground/the municipality office giving permission for cremation, non-issuance of the same will automatically ensure that the body can not be cremated.

Having taken over the custody of the dead body, the investigating police officer proceeds with inquest and the cause of death is decided after the medico-legalpostmortem is carried out as part of the inquest. This system ensures that the body is not disposed off without necessary investigations by the police into the cause and circumstances of death, when the death is due to other than natural causes.

Certain other points to be kept in mind by the medical officer concerning the issuance of medical certificate of cause of death, are:-

(a) He should not delay, for any reason, issuing the medical certificate of cause of death, once he is sure of the cause of death.

(b) He can not charge any fees for issuing this certificate.

© He should not withhold issuance of medical certificate of cause of death even if his dues have not been cleared by the relatives.

(d) No medical officer should sign medical certifi-

cate of cause of death in advance (i.e. before the individual has died) or without viewing and examining the dead body personally.

Clause 3.10 of revised dentist code of ethics of 2014 allows dentist to issue death certificate. Most dentists are not aware of it. Dentist can certify death of patients under their treatment.

HOW TO FILL THE FORM

Name of the deceased: to be given in full. Do not use the initials. If the deceased is an infant, not yet named, at the time of death, write son of (S/O) or daughter of (D/O), followed by the names of mother and father.

Age: If the deceased is over one year of age, give age in complete years. If the deceased was below one year of age, give age in months and if below one month of age, in completed number of days, and if below one day, in hours.

Cause of death: this part of the form should always be completed by the attending physician personally. The actual certificate is divided in two parts viz. part-I and part-II.

Part-I. It Deals with the immediate cause and the underlying cause of death. The immediate cause or the terminal event is entered here. Part-II. It Deals with other significant condition or disease contributed to the process of death but did not lead to it.

Part-I

Only one cause is to be entered in each line. Line-a: Immediate cause of death is entered here, like disease/abnormality/injury/poisoning etc. The immediate cause is defined as the immediate or terminal event leading to death. Mode of death such as respiratory failure, cardiac failure etc is not an appropriate entry. Line-b: Next considers whether the immediate cause is a complication of delayed result of some other cause. If so, enter the antecedent cause in part-1 line- b. The antecedent causes refer to the pathological process or injury responsible for death. Thus it is a disease or injury that has initiated the train of morbid events leading directly to death or it is the circumstances of the accident or violence that produced the fatal injury3.Line-c: Sometimes there will be three stages in the course of events leading to death. If so, line-c will be completed.

Part-II

Other significant conditions contributing to death, but not related to disease condition causing it. Onset: complete the column for interval between onset and death whenever possible, even if very approximate e.g. from birth, or several years etc.

The sequence of events to be followed, in death certification are[4].

The cause of death is known

7.1. Death of a person- the attending physician/registered medical practitioner issues the death certificate, in prescribed proforma. It is as per provision made in the Registration of Birth and Death 1969 (18 of 1969.)[5]

7.2. The certificate is send to Registrar of Birth and Death. (As per above Act)

7.3. Registrar or his representative certifies the extract given in the certificate.

7.4. This certified death certificate is admissible as evidence for the purpose of proving birth/death. (Section 76,of Indian Evidence Act 1872 (1 of 1872).

B) The cause of death is not known/ uncertain.

7.5. The last attending doctor does not issue the death certificate.

7.5.1. The case is informed to the police.

7.5.2. The body is kept in safe custody and handed over to the police for autopsy.

7.5.3. After autopsy the facts of death is communicated to local registrar.

7.5.4. Death certificate is issued by registrar of Birth and Death.

CONCLUSION

Considering the importance of correct certificationof medical cause of death and death registration, theforegoing account of the different aspects of the same,has tried to clarify the difficulties and doubts encounteredby the registered medical practitioner while certifyingdeath.The awareness among dentists regarding the issue of death certificate is less and more focus should be done to increase awareness. This article aims to increase the awareness among dentists regarding death certification.

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FIGURE 1 – FORM 4

FORM –4

MEDICAL CERTIFICATE OF CAUSE OF DEATH

(Hospital in-patients. Not to be used for still-births)
To be sent to Registrar along with From No.2 (Death Report)

Name of the Hospital _____
I hereby certify that the person whose particulars are given below died in the Hospital in Ward _____ on _____ at _____ A.M./P.M. _____
Name of the Deceased _____ for use of Statistical Office

Sex	Age at Death			
	If one year or more, age in years	If less than one years, age in Months	If less than one month, age in Days	If less than one day, age in Hours
1. Male 2. Female				

	CAUSE OF DEATH	Interval between on set & Death approx	
1. Immediate cause State the disease, Injury or complication, which caused death, not the mode of dying such as heart failure, asthenia, etc.	(a) due to)or as a consequences of)		
Antecedent cause Morbid conditions, if any giving rise to the above Cause, stating underlying conditions last.	(b) due to (or as a consequences of)		
	(c)		
II Other significant conditions contributing to the death but not related to the diseases or conditions causing it.			

Manner of Death :
1. Natural 2. Accident 3. Suicide 4. Homicide
5. Pending investigation

How did the injury occur

If deceased was a female, was pregnancy the death associated with 1. Yes 2. No.
If yes, was there a delivery ? 1. Yes 2. No.

Name and signature of the Medical Attendant certifying the cause of death

A BIZARRE DYSPLASIA OF THE GINGIVA - CASE REPORT WITH REVIEW OF LITERATURE

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ABSTRACT

Gingival enlargement is currently used to describe medication-related gingival overgrowth or hyperplasia, a reactionary phenomenon that occurs with the use of several types of therapeutic agents, chiefly antiepileptic drugs. This disorder was recognized since 1939, shortly after the introduction of phenytoin. Along with the review of literature we are also giving a case report of a female patient with gingival enlargement as anparadigm.

Keywords : Drugs, Gingival enlargement, Phenytoin

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INTRODUCTION

Drugs used locally or systematically can induce several changes in the micro and macroscopic tissues.[1] These drugs may be of therapeutic use but may cause adverse effects of other body organs or systems and one such conflicting effect is the overgrowth of the gingival tissues.

"Gingival Enlargement "is a term used to describe medicine - related gingival overgrowth. Gingival hyperplasia is defined as an abnormal growth of the periodontal tissue.[2] But the latter is discontinued as by histopathology this cannot be accepted now as hyperplasia merely means increase in the number of cells but enlargement is caused due to the increase in the extracellular volume of the cells.

Drug induced gingival enlargement due to the chronic use of phenytoin was first described by Kimball in 1939.[2] In 1948 Brandon hypothesized that phynetoin had direct effect on the gums. In 1975, Angelopoulos commented that phenytoin induced degranulation of mast cells lead to increased collagen formation. Larmas in 1976 said that phenytoin caused the proliferation of the basal cell layer which increased the epithelial connective tissue interface and this was later confirmed by Hassel et al. Saito et al in 1999 postulated that there is p53 expression in drug induced gingival overgrowth which proved that this is associated with DNA abnormalities.[3]

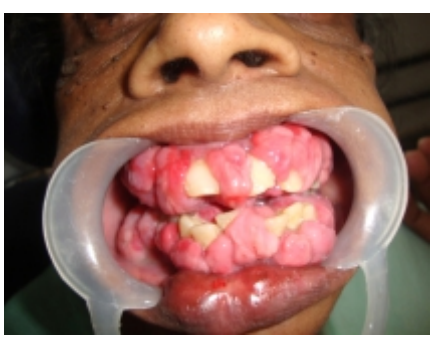
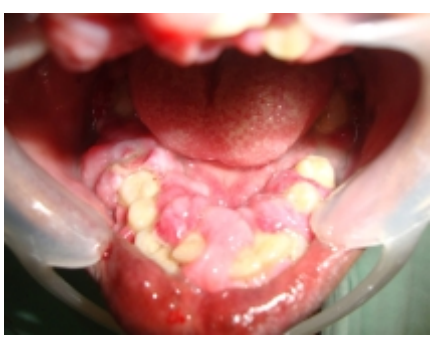
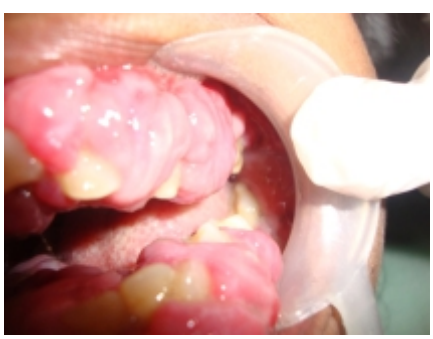
Here we are also affixing a case report of a 60 year old female patient affected with this aesthetically, functionally and psychological disturbance seeded due to prolonged use of anti epileptic drugs.

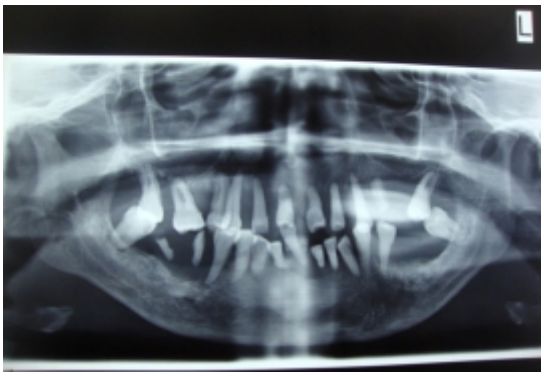
CASE REPORT

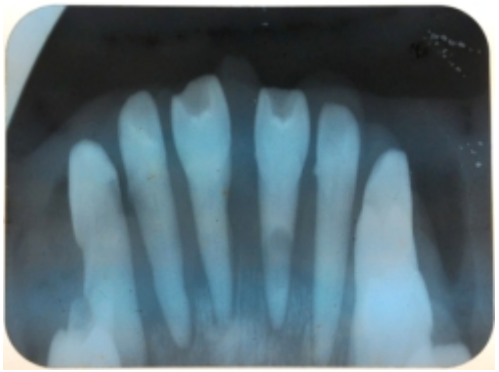
A 60 year old female came to our department with a chief complaint of swollen gums in the upper and lower jaws which she had been noticing for the past 1year. The swelling was initially insignificant which has now increased to the present since the last 2-3 months. She experienced mild pain especially while having food & brushing and also had a unsatisfactory malodor in the last 2 months. She also complained of bleeding while brushing and hence used mouthwashes for cleaning teeth. Since the patient was a psychiatric patient most of the details were given by the bystanders. Patient had a lean built and was malnourished.

She was under psychiatric medication for seizures since the last 35 years. She had been hospitalized several times for the treatment of multinodular goiter, seizure disorder, depression illness, mitral regurgitation and bronchiactasis and was also under various medications. Multiple surgeries were conducted and there was history of hysterectomy, cheiloplasty and surgery for a neck swelling. Drug history revealed that she is allergic to an unknown drug.

Extraoral examination revealed an abnormal face with bimaxillary protrusion and incompetent lips. On intraoral examination there is severe generalized bulbous swelling of the gums which was also edematous. Oblitersation of the buccal, labial and lingual vestibule was seen. Almost 2/3 rd of the teeth were covered resulting in the displacement of the teeth with midline shift. On palpation all inspectory find-





ings were confirmed and there is bleeding present with tenderness. Grade III mobility of almost all teeth was present. Provisional diagnosis was given as leukaemia, granulomatous enlargement and drug induced gingival enlargement.

OPG and Maxillary and mandibular occlusal radiographs revealed severe generalized horizontal bone loss and adequate spacing between the teeth giving a a floating teeth appearance. Haemoglobin levels showed that she was anaemic with a value of 10.8 mg. Blood glucose level was 134mg/dl. Biopsy was advised for this patient where the histopathological analysis came as Drug induced Gingival enlargement with inflammatory changes. Later gingivoplasty was also done.

DISCUSSION

The periodontium comprises of tissues that surround and support the teeth.[1]The gingiva is a pink or pigmented mucous membrane cover that wrap tightly around the teeth and provides anchorage to the dental arches.2 Histologically there is presence of epithelial and connective tissue layer with fibroblasts and the extracellular matrix is predominantly made up of collagen fibres and glycosaminoglycans. The periodontal ligament is a connective tissue layer that surrounds the dental root region and link the cementum to the alveolar bone allocating more functions. The cementum is a hard tissue structure similar to that of the bone. The alveolar bone is a supporting structure that provides anchorage to the teeth due to the attachment of the periodontal fibres.[6]

The etiological factors of gingival enlargement comprises of inflammatory factors which include the poor oral hygiene and periodontal diseases[1], idiopathic, various drug induced neoplasias including antiepileptic drugs, genetic changes, hereditary gingival fibromatosis and also the nutritional and hormonal disturbances. These can cause aesthetic changes in an individual with accompanying symptoms like tenderness, pain, bleeding, abnormal tooth movement, problems in speech and in occlusion which further ushers periodontal disorders.[2] Congenital gingival enlargement includes the fetal valproate syndrome .[5]

But still for gingival enlargement the major causative factor was phynetoin. Currantly, more than 15 drugs have been identified including oral contraceptives, antihypertensives calcium antagonists and immunosupprants chiefly cyclosporins.[2] One basic factor in all these drugs is that it affects the cellular calcium metabolism. Since the formation of the fibroblasts is modulated by calcium these drugs will affect the collagenase activity leading to inactive forms of collagen thus paving way for increased extracellular matrix.

In one study it was said that a subtype of fibroblasts called “responders” is affected which is highly sensitive to these calcium biased drugs.[1] This decrease in the calcium causes a decrease in the folic acid

uptake causing folate deficiency leading to altered production of collagenase[2] by the fibroblasts and thus leading to increased over accumulation of collagen causing the increased bulk of the connective tissue.[5]

Phenytoin use is also related to increase in the number of the langerhans cells which can cause a increase in the interleukin 2 and tumor necrosis factor.[7] But the malignant transformation rate has not yet been reported.

The clinical features of the growth may commence during the first three months after the initiation of the therapy especially the thickening of the pappilary and the marginal gingival of the labial surfaces of anteriors developing to lobules and reaches the maximum severity in the twelfth to eighteenth month.[8] The enlargement of the gingival tissue ideally begins in the area of interdental papillae which then spreads laterally to the adjacent areas. If there is adequate plaque control then there will be minimal bleeding with firm gingival consistency and healthy colour. But if the enlargement is associated with bacterial inflammation then the colour will turn reddish to purple.[9] From this can be understood that the bacterial plaque can cause the alterations in the periodontal tissue environment and the systemic factors can enhance the pathological conditions in the underlying connective tissues.[1] The extension of the enlargement depends on the dose, duration of the drug being used. The growth may occur gradually and sometimes in severe cases coverage of the entire tooth is noted with associated displacement of the teeth.[2] The gingival growth along with the deepening of the sulcus can act as an nidus for the bacterial growth which can aggravate the gingival inflammation.[1]The deeping of the sulcus caused due to plaque can also act as a reservoir for the abused drugs.[3]

This usually affects the young individuals between the age group of 8 - 13 years who are on dilantin therapy but people of all age groups can be affected.[3] Both sexes are affected equally but some studies have shown that males are affected three times more when compared to the females.[5] Children born to the affected mothers can also be affected.

This can be a serious problem which can aggrevate the psychological symptoms of the patients as enlarged gingival can affect the esthetic concern of the patient.[5]

Phenytoin (dilantin) was first introduced in 1930s by Merrit and Putnam, and this has acted as the drug of choice as an anticonvulsant in the treatment of grand mal, temporal lobe, and psychomotor seizures.[6]The common adverse effect of this drug was gingival hyperplasia of varying degrees. Long term phynetoin use can also cause enlargement of the lips and thickening of the face and scalp.[7] Incidence rates are found to be 3 - 93 % among the drug users.[8]

Nifedipine a calcium channel blocker commonly used for cardiovascular diseases blocks the influx of calcium into the cardiac muscles.[1] This can cause a decrease in the contractile process of the cardiac muscles thus decreasing the arterial blood pressure. Ramon et al, Heijl et al. and Shaftic et al. reported the prevalence of overgrowth as 0.5 to 83% for nifedipine users. The drug interferes with the calcium metabolism of fibroblast cells and hence reduces the production of the degrading enzyme collagenase. This drug affects the protein synthesis of the fibroblasts.[5] The prenvalence rate is 10%.[9]

Immunosupprants like cyclosporine used for organ transplants also cause gingival enlargement. The prevalence rate is 30%.[9]

Gingival enlargement can be measured by 3 methods.[8] The cast method, photographic method and the clinical measurement method. According to Bokenkamp[8] (1994)

Grade 0 - no signs of clinical inflammation

Grade 1 - Enlargement confined to the interdental papilla

Grade 2 - enlargement involves the papilla and the marginal gingiva

Grade 3 - Enlargement covering three quarters or more of the crown of the teeth

Generally Drug induced gingival overgrowth begins as a bead like enlargement of the interdental papilla

affecting the attached gingival also in the later stages.[8]

The differential diagnosis includes the hereditary gingival enlargement but this is an autosomal dominant trait with a strong family history.[1]Leukaemic infiltration is usually associated with secondary inflammation and so there will be associated bleeding.4Tuberculosis and other granulomatous diseases, including orofacial granulomatosis, Crohn's disease, and sarcoidosis, can also mimic drug-induced gingival enlargement.

The first line in the management is the cessation of the drug but this does not cause a regression of the enlargement. [1] But in long standing cases regression may not take place even on cessation of drug. This had been noticed for 1 year in a study.[5] Somacarrera et al reported that an alternative of the drug can be given to reduce the symptoms.[5] Periodontal treatment which includes both the personal and professional care is advised to control the inflammation supplemented with chlorhexidine mouthwashes and folic acid and applications.[6] Mechanical removal of the plaque, calculus can control the inflammation.[7]A healthy oral environment will help to avert the local microflora eliminating the major focus of infection.[8] For severe cases surgical excision either by gingivectomy, gingivoplastyor periodontal flap approach is done.[9] Laser therapy have also proved to be a boon mainly because of its postoperative haemostasis.

Hassan et al suggested that azithromycin toothpaste is an effective, simple and noninvasive treatment for cyclosporine induced GO. Prasad et al. concluded that systemic folic acid prescribed along with phenytoin delays the onset and reduces the incidence and severity of gingival overgrowth.[1] There was a case of relapse in 3 months of the surgical therapy but with proper home care and chlorhexidine mouthrinses the enlargement can be controlled with a 3 month follow up.[2]

Certain examples of the drugs that cause gingival enlargement.

Anticonvulsants	Phynetoin Mephynetoin Valproic acid Ethosuximide Phensuximide Mesuximide
Calcium channel blockers	Amilodipine Felodipin Nicardipin Nifedipin Nisoldipin Verapamil
Immunosuppresants	Cyclosporin A

CONCLUSION

It is therefore very important that not only neurologists but even the dentists must apprehend the potential conflicting etiology of this aesthetically disfiguring condition and its characteristic features to prevent, diagnose and successfully manage it. Then physicians , general practitioners and dentists must coordinate together for a concise treatment plan that is beneficial to the patients.

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