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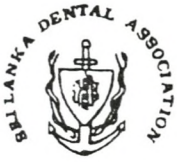

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Volume 36 **Number 01** **April 2006**

EDITORIAL

EVIDENCE BASED DENTISTRY (EBD)

The status quo between the patient and the clinician is changing rapidly. Through the dissemination of knowledge via the media, internet and other resources, patients educate themselves and stand at a position of judgment, holding the doctor responsible for any breach of duty of care or lesser standard of expertise delivered. As such, rather than adhering to conventional scenarios, health professionals will have to change their clinical practices in accordance with the best available scientific evidence for the maximum benefit of the patient. This calls for an extremely high standard of care, professionalism, and expertise that we dentist should aspire to. Thus evidence based practice of dentistry comes into focus. Through this, it is envisage that better quality care can be delivered to the patient today.

Currently, there is a global trend towards the practice of evidence based dentistry. Although practicing evidence based dentistry is not difficult, it is time consuming and exacting. There are a number of barriers in common to overcome. Knowledge and the attitude of the practitioner, assimilating information from a large corpus of literature, inaccessibility to the relevant and most appropriate information, the gulf between the clinician and the researcher and findings of the research, practice based on authority rather than evidence are some of them. There are many successful strategies to overcome these barriers. If we address only the issue of information for

example: currently over 2 million biomedical articles are published every year in some 20,000 journals worldwide and out of which about 500 journals are related to dentistry. Are we to read all? Data bases and a methods of extensive review of the literature and summarizing information gathered from multiple publications (eg: meta analyses) should be developed for our clinicians. Furthermore there needs to be a closer link between researcher and the clinician so that researchers can tailor their research programs in relevance to the practitioners' need.

If we are to advocate the evidence based approach in our country, undergraduates and postgraduate students are the best target of choice at the initial stage. However, the ultimate goal would be that all dental surgeons in Sri Lanka, practice dentistry based on best available scientific evidence. Today is a better time to initiate the change than tomorrow. If we initiate the move right now, *dentistry tomorrow* will be better than today.

Dr. Upul B. Dissanayake
Editor - SLDJ

Leading Article

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Corruption in the health sector

A. N. I. Ekanayaka

Introduction

Corruption is an evil that is as old as human civilisation itself. It is a form of moral turpitude with global dimensions transcending all cultures, communities, and professions extending its tentacles to every sector of human activity. In the modern world corruption is one of the paramount issues of our time. The scale and complexity of organisational structures, the size and power of governments, the pervasiveness of the "market" amidst the existence of a network of economic interrelationships and the sheer volume of financial flows, have today created an environment in which the capacity for corruption to monumentally enrich some and savagely deprive others is unparalleled.

Transparency International (TI) defines itself as the "leading international non-governmental organisation devoted to combating corruption."[1] It carries out this function through its International Secretariat in Berlin and 99 independent National Chapters around the world. TI releases an annual "Global Corruption Report" which each year is devoted to a separate sector of the economy.[1]

The 2006 Global Corruption Report[1] focuses entirely on corruption in the health sector a subject that ought to be of burning interest to all doctors and dentists in Sri Lanka. The report has been hailed by WHO's Director of the Department of Medicine, Policy and Standards as " the reference book for corruption and health for the next coming years."[2] However, it is unlikely that many health professionals would access this 350 pages publication which includes many references and numerous country reports, still less have the time to peruse and digest such a large report.

Consequently the present article is intended to summarize some of the concepts, facts and conclusions in this unique report at a time when Sri Lanka is widely regarded as one of the most corrupt countries in the world. In 2005 Sri Lanka was ranked in 78th position obtaining a bad Corruption Index Score of 3.2 (on a 0-10 scale) as estimated by business people and country analysts [3]. Other than where specifically indicated all the information furnished in this article is culled from the fascinating and highly topical 2006 Global Corruption Report of Transparency International.[1]

Definition

Corruption may be defined as "the abuse of entrusted power for private gain". This definition conveys an understanding of corruption in health care which goes beyond the usual notion that corruption is basically a public sector malady involving a mixture of petty bribery by government health workers and commissions and kick backs received by crooked health ministry officials. On the contrary depending on the type of health care system "the abuse of entrusted power for private gain" to the detriment of patients could be rampant and take many different forms even in the private sector. Moreover the concept of "private gain" can

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be interpreted, to include all forms of collective action by health professionals that are driven by the selfish vested interests of the health sector trade unions and professional organizations. Therefore even strikes by health workers and the use of trade union muscle to thwart progressive health policies thereby placing the vested interests of health workers above the public interest of patients must also be regarded as a form of corruption in the health sector.

The extent of the problem

In the modern world a colossal amount of money is spent on health care resulting in massive flow of funds between the various actors in the health care system. Therefore health care systems are a fertile field for corrupt practice.

The world spends a staggering US $ 3.1 trillion annually on health services of which the US alone spends US $ 1.6 trillion. The British National Health Service employs 1.2 million staff and has an annual budget of US $ 125 billion. In many Third World countries private out of pocket spending on health care exceeds government investment in the health sector. Nevertheless the proportion of total government revenues allocated to health care is considerable – ranging from under 5% in some poor countries to more than 15% in USA, Germany, Ireland and Costa Rica. Clearly there is a lot of money to be targeted by those who would abuse and rob the system.

Like all corruption that which occurs in the health sector is frequently insidious and invisible. Estimates of corruption within health services although obviously underestimates are nevertheless mind boggling. The Global Corruption Report[1] estimates that it runs into tens of billions of dollars. The illegal pickings from counterfeit drugs alone may exceed US $ 30 billion. In the USA Attorney General Janet Reno declared in 1993 that health care fraud was America's "number two crime problem – second only to violent crime!"[4] In the British National Health Service (NHS) losses due to patient related fraud amounted to US $ 305 million in 1999. With the establishment of the Counter Fraud Service (CFS) in 1998 such losses were reduced by 54% by 2002, while losses caused by fraud by medical professionals were estimated to have dropped by 43-54% over the same period. It has been calculated that the total savings to the NHS from the activities of the counter fraud service since 1999 amounted to US $ 1.2 billion (sufficient to construct 10 new hospitals!)[5]. Such figures illustrate both the enormous potential for corruption even in the best of health services and also the fact that it can be contained if effective measures are put in place.

Examples of health service corruption and scandals exposed in other countries across the world abound. In Cambodia where maternal mortality is the highest in the region with infant mortality running at 10% it is widely assumed that 5 to 10% of the health budget disappears even before it is, paid out by the Ministry of Finance. Jobs in the health sector are coveted for the opportunities they offer for personal enrichment, so much so that the job of a low level health official may command a bribe of US $ 3000 and that of a provincial director US $ 100,000, which is many times the salary of poorly paid health workers in that country. Individual corruption scandals involving millions of dollars have been exposed in Costa Rica and Mexico while in Thailand the Public Health Minister was sentenced to 15 year's imprisonment in 2004 for accepting bribes from drug companies.

Public Expenditure Tracking Surveys (PETS) conducted by the World Bank and other organisations in many developing countries have attempted to find out to what extent funds budgeted for health fail to reach target populations due to the irregular diversion of resources at source or poor implementation of programmes. Such surveys have revealed 70-80% "leakage" of budgeted recurrent expenditures, drugs and supplies, and nutritional supplements intended for the poor in various countries. A multi country study

has revealed that absenteeism in the health sector ranges from 23–40 %. In Eastern Europe and the former Soviet Union which are struggling with economic transition 51–89% of respondents surveyed felt that the "majority if not almost all" health workers were corrupt.

Such findings are but the tip of the iceberg provoking the Global Corruption Report to conclude that "growing evidence from around the world indicates that corruption, fraud and abuse are resulting in significant losses of public money and denial of good quality health services to millions of people".

Why is the health sector so liable to corruption?

There are four reasons why health sector corruption is so common and why it is so hard to detect and eliminate. Firstly health services are extremely complex organisations involving many actors. They include patients who demand health care, a vast range of providers who deliver institutional and community care in both the private and public sectors, payers through whom costs are met either directly or indirectly whether by means of social security or health insurance, and finally the government which acts as a regulator. These parties in turn interact constantly with innumerable suppliers of drugs, equipment, and consumables. The transactions between these various parties are complex, many faceted and impenetrable to the scrutiny of civil society. Consequently they are corruptible at many points where self interest, profit and power are the imperatives that drive policy formulation and practice decisions within health care systems.

Secondly, there is the inherent uncertainty of the health care market. The future prevalence of disease cannot be predicted with certainty like the demand for some other consumer product. The need for treatment may vary depending on the diagnostic technology used and the diagnostic criteria applied by physicians. Demand is an additional variable affected by a wide range of socio-behavioural imponderables. Given the range of treatment options available the effectiveness and outcome of treatment is often variable. Consequently the planning management and monitoring of health care, the setting of priorities and the allocation of funds frequently take place under conditions of unpredictability and constant flux opening the door to many abuses.

Thirdly, health care systems function under conditions of information asymmetry and opacity where some are in the know while others are in the dark. Doctors know far more than patients about their sickness. In turn doctors are dependent on pharmaceutical companies for information about the drugs they prescribe. The decisions of health administrators that impact on the day to day work of doctors may frequently lack transparency. Where such groups interacting with each other have unequal access to information there is greater scope for fraud, deceit and mutual manipulation resulting in market failure.

Fourth, the power and mystique of the medical profession, and the total dependence of the public on medical personnel for deliverance from sickness and death, place health workers in a position of unique advantage over both the patients they treat and the governments they serve. Especially in Third World countries where the health sector is held in such awe by society, its abuses frequently pass unchallenged.

Forms of corruption in the health sector

The type of corruption that takes place is largely determined by the way in which health care is funded. There are broadly two systems. In the first the public sector is responsible for both financing and providing services. Alternatively there can be a separation between financing and provision involving third party payment systems. In this modality the providers of services (eg: doctors and dentists) are independent practitioners who are contracted to provide services that are financed by government or employer based compulsory social insurance of

some kind. The latter system would also include the purchase of services by patients through direct out-of-pocket payments to providers or through third party personal health insurance policies. Whether common to both systems of funding or peculiar to one or the other, the following are the main types of corruption observed in the health sector.

1. Large scale, illegitimate diversion of funds allocated for health for other purposes. This may take place at the level of Ministers or senior officials or lower down the system when funds are distributed to decentralized authorities. This even includes in some cases the siphoning off funds earmarked for salaries resulting in the delay or non payment of salaries, for example Nigeria where it was found that 42% of staff were victims of salary delays despite adequate budget allocations. In Ghana it was found that 80% of non salary funds had not reached health facilities.

2. Commissions, kickbacks and graft in the procurement of equipment and supplies by health officials especially in the public sector resulting in soaring costs and the procurement of unnecessary, inappropriate, defective or low quality supplies.

3. Theft and misappropriation of consumables and equipment by health personnel

4. Absenteeism of health workers, their location in stations different to those assigned, and the creation of fictitious ("ghost") cadre positions the salaries earmarked for which can then be conveniently diverted to others.

5. The self referral and diversion of patients from State institutions to the private sector

6. Bribery of health officials as a way of by-passing stringent government regulations, monitoring, approval, and licensing procedures that may have been put in place to ensure the protection of patients and the maintenance of minimum standards. Ironically the greater the extent government regulation of health services with more officials (susceptible to bribery) empowered to clear more approvals at more bottlenecks in the system, the greater the scope for bribery.

7. Health workers in the State sector who are on a fixed salary lack the incentive to work hard. Consequently the services they provide may be characterized by low productivity and compromised quality of care especially in under developed countries where professional regulation is lax and it is rare for dissatisfied patients to sue their doctors.

8. The "supervised neglect" of patients who are on "capitation" systems of payment where a fixed payment is made in lieu of each patient registered in a practice whether or not and irrespective of how much treatment is needed. While in theory "capitation" payments are intended to provide an inducement for preventive care, corrupt and devious ways of defrauding the system so as to rake in capitation payments while maintaining only a deceptive veneer of patient care is a well known weakness of this system.

9. Defrauding of third party organizations (whether government or insurance companies) that make fee for service payments for services contracted out to independent practitioners. This may involve false or exaggerated claims based on fraudulent medical records as well as claims for deliberately excess and unnecessary treatment. According to the British Audit Commission[6] 59% of adults in England had had a scaling the last time they attended for dental treatment. The Audit Commission concluded that " Even on a crude estimate that half of this activity might be

unnecessary–and the evidence suggests that it may be more – about £ 65 million could be saved". Fraud involving such payment mechanisms is the dominant form of corruption within the US health care system with government Medicaid and Medicare programmes showing losses ranging from 5-15% due to over billing. Unfortunately the highly automated systems in place to process and adjudicate claims only facilitate dishonest practice because they only verify whether the claims are in conformity with the required format, rules and operational procedures of the payment system and do not check the accuracy and authenticity of the information itself.

10. One of the most universal forms of corruption occurring at all levels of the system is the system of "informal payments" where it is expected that patients would make some voluntary payment for services that are supposed to be free. Whether through tips, gifts, petty bribes, "gratitude payments", or "under the table" or "brown envelope" gratifications to health workers and officials, informal payments constitute a pervasive form of corruption all over the world. The culture of informal payments compel people in desperate need to purchase as a concession a service to which they are entitled as a right.

    Given the hidden nature of such "unofficial" transactions and the reluctance of patients to "talk" for fear of victimization they are hard to detect or quantify. In the transitional economies of some Eastern European countries informal payments comprise 30-84% of total health expenditure. Informal payments frequently augment the low pay of health workers in many cultures, nearly doubling physician's official salaries in Poland. In Bulgaria where doctors are paid a monthly salary of about US $ 100, informal payments may augment their income by ten fold. Whether as an effective salary subsidy to a physician, a tip to an attendant or labourer in return for an OPD number or some concession in the ward, the burden of informal payments weighs hardest on the poor who are least able to pay. Consequently informal payments affects access to care, fosters discrimination in the provision of care, and increases health inequalities. The ultimate scandal of such an iniquitous system is that it effectively causes the "privatization" of what is nominally a free health care system.

11. Interactions with the pharmaceutical industry constitute one of the most prominent domains of health sector corruption. In under developed countries where sickness is a major reason for the economic ruin of families, personal expenditure on drugs may constitute 50-90% of total individual household expenditure. Even in Sri Lanka with its strong tradition of free health care, of the per capita monthly expenditure of Rs.135.00 on health care Rs.77.41 is spent on medicines.[7] Consequently the increased cost of pharmaceuticals caused by corruption, market failure, and government incompetence places an intolerable burden on the most vulnerable groups in society resulting in a "drug gap" where WHO estimates that one third of people worldwide do not have regular access to basic medicines.[8] Manifold forms of corruption occur at five decision points in the pharmaceutical chain. They include the registration of drugs, the selection of drugs to be ordered, the procurement of selected drugs and their distribution, and finally the use and prescription of drugs by doctors in day to day practice.

    In the registration of drugs the reliance on dubious clinical trials lavishly funded by drug companies, pressure by various authorities to short circuit and rush through approvals, increasing government industry partnerships and even industry subsidized salaries for

officials of regulating agencies may induce leniency and the compromise of standards. In the selection of drugs by organisations, commissions and pay-offs by drug companies may induce relevant authorities to deviate from the WHO model list of essential medicines and order unnecessary and sub standard drugs. Yet other kick backs and commissions may corrupt the procurement of selected drugs under conditions where tenders are not selected on the basis of open competitive bidding and where deliberate delays that lengthen the procurement cycle may be contrived precipitating acute shortages that can then be used as an excuse to deviate from normal procedures and justify purchases from private suppliers as an emergency measure. In the distribution cycle supplies may be lost, damaged or go out of date due to outright theft, bad storage, inappropriate transport, and poor coordination between the centre and periphery where health administration is decentralized.

Most repugnant of all is the systematic moral corruption of medical practitioners and professional organisations and the distortion of their prescribing patterns by the frenetic marketing practices of pharmaceutical companies. It is reported that in the USA the pharmaceutical, device and biotechnology industry spends US $ 16 billion annually on marketing to physicians, $ 2 billion alone on meals, meetings and benefits. The tantalizing array of benefits with which drug firms are known to "buy" the favour of practitioners include the sponsorship of lavish dinners lunches, and scientific meetings, attractive honorary payments for speaking engagements (which in the USA may be as high as US $ 1000–5000) trips abroad including air tickets for spouses, research funding, drug samples, gifts, assisting academic teachers to prepare teaching materials, consultancies, and prestigious membership in drug company advisory boards in addition to alluring medical students with gifts of stethoscopes and textbooks.

While recognising the right of pharmaceutical firms to engage in legitimate marketing of their products and acknowledging the signal contribution of the industry to research and development, the widespread tendency for health professionals to compromise their scientific authenticity in return for material favours received, is a sad reality of our time. The former Editor in Chief of the New England Journal of Medicine contributing to the Transparency International investigation bemoaned the increasing difficulty of finding authors to write review articles and editorials who had no financial links with the firm whose products were listed in the article.[6] Meanwhile it has been shown that 50% of full professors in the USA who carry out life science research have significant financial arrangements with industry. The corruptive influence of drug companies have penetrated deep into the scientific establishment. In 2004 media reports exposed a shocking conflict of interest when a 9 member prestigious group of scientific experts recommended Statins for lowering LDL, 7 of whom were found to have links with companies that manufactured the drug. Similar exposures have even tainted the reputation of the US Food and Drugs Agency where panel members charged with registering a drug were found to have financial links with the companies making the drug.

Then there is the corruption of academic medicine through links with the pharmaceutical industry. Jerome P. Kassirer distinguished Professor at Tufts University School of Medicine and former Editor-in-Chief of the New England Journal of Medicine has lucidly quoted the ways in which this can take place where "huge financial subsidies tend to distract faculty into emphasising profitable research and to

neglect their teaching duties. It replaces openness with secrecy, privatizes knowledge and replaces part of the social commons by commercializing discovery. It has also created a culture in which the design of studies is sometimes jiggered to create positive results, in which unfavourable results are sometimes buried; in which communication of results is sometimes hindered for commercial reasons; and in which bias in publications and educational materials has sometimes gone unchecked."[9]

12. Finally there is the global market in counterfeit and sub-standard drugs aided by corrupt customs officials and government inspectors who are bribed into allowing them to pass through various check points, alongside an ineffective judiciary. It is reported that in 2001 China (where an estimated 192,000 people died in 2004 due to fake drugs) there are 500 illegal drug manufacturers and in Laos about 2100 illegal medicine sellers. In India where the Minister of Health recently likened profiteering in fake drugs to mass murder, the introduction of the death penalty is being considered for this crime.

Controlling health sector corruption

Health sector corruption is one of the worst crimes against society, selfishly profiting as it does on the backs of the helpless, the sick, the suffering, and the dying. Nevertheless it is hard to detect and prevent.

The Transparency International Global Corruption Report[1] proposes several strategies that may contribute to the reduction of health sector corruption. They include active civil society and community representation in health sector management structures and policy formulation at all levels which is one of the most important bulwarks against corruption. This may be augmented by maximum transparency through mandatory posting of information about health policies, tenders, procurement, and expenditure on the internet enabling public scrutiny of matters that are usually buried beneath piles of files to which only health officials have access. Rigorous independent systems of audit and continuous monitoring of payment systems must be put in place as well as the introduction and emphasis of explicit codes of conduct governing the actions of all parties involved in the planning, administration, funding and provision of health care. Protection and immunity for "whistleblowers" who provide information about corrupt practices in their workplace, providing realistic salaries to health workers, and the imposition of drastic punishments for corrupt acts according to the law without fear or favour, are other measures that need to be put in place.

Health sector corruption in Sri Lanka

The present article has endeavored to provide an overview of health sector corruption from a global rather than a national perspective. Any reliable estimate of health sector corruption in Sri Lanka would necessitate an in-depth study of relevant information that is beyond the scope of this article. Moreover whether sufficient information even exists (and can be accessed through available means) on which to base a responsible conclusion is questionable.

However one thing seems to be clear. Judging by persistent media reports there is considerable public anxiety about corruption in Sri Lanka.
A national newspaper referring to a recent United Nations Development Fund (UNDP) Workshop quoted a senior official of the Bribery Commission as stating that 68% of government servants appear to be involved in bribery and corruption.[1] Recent reports by the Auditor General have highlighted corruption, irregularities, and waste in the public sector. Worse outrage has been expressed in some circles about the deplorable reaction of a high Treasury official who has criticized such exposures as being bad for the morale of the public service.[2] It is also common knowledge that the Auditor General has in a report

to parliament made severe strictures regarding the mismanagement, waste and corruption in the use of donations received following the Tsunami disaster[3] – shocking revelations which in a more open society would have compelled a national inquiry. The Auditor General has also reported, large scale irregularities in the health sector involving the purchase of drugs, underutilization of costly assets, losses, and damages.[4]

Against this background and in terms of his/her own personal experience of our health services, it is for the reader to estimate to what extent the different forms of health sector corruption described globally are prevalent in Sri Lanka. In doing so it is important to be guided by the broad definition of corruption namely "the abuse of entrusted power for private gain". Many features of the Sri Lankan health sector fall short by the standards implicit in this definition. The finding that 60% of the time dental surgeons in hospital clinics are either absent or present but unproductive[5] various abuses in the exercise of the right to private practice by government doctors and dentists, wide variations in the price of private medical and dental care, anecdotal reports of excess, unnecessary and poor quality dental treatment and unethical behaviour, the dominance of trade union vested interests over the public interest as reflected in frequent strikes by health workers and in particular the intimidation of the government into suspending the dental therapist training programme, the retrogressive bifurcation of dental from medical administration in the Ministry of Health to the detriment of dentistry by creating the plum position of DDG (Dental), and the controversies surrounding the appointment of a Forensic Odontologist by the Ministry of Health–these are all in one way or the other examples where entrusted power is arguably being abused for private or sectarian gain. Whether such manifestations are but the tip of an iceberg of corruption in the health sector in Sri Lanka is a question that demands the urgent attention of the government, the profession and the public.

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Management of inherited coagulation abnormalities in dentistry.

D. M. Dissanayake and U. B. Dissanayake

Introduction

Patients with bleeding disorders are a high risk group from the dental point of view. Any routine dental surgical procedure can be, and often is, life threatening for them. Bleeding disorders most often present with excessive bleeding after tooth extraction as the initial manifestation of the disease. Bleeding from the sites of local anesthetic injections into the tissue spaces and development of haematomas even extending into neck is another manifestation that one may encounter. Endotracheal intubation in general anesthesia can also lead to development of retro-pharyngeal haematomas. Hence adequate knowledge on bleeding diseases would be extremely helpful to dental surgeons in the management and treatment of patients. This further emphasies the importance of preventive dentistry which would minimize dental surgical intervention and reduce risks in patients with bleeding disorders.

Theoretically, a defect in any of the factors involved in the coagulation mechanism can cause bleeding in an individual. Hereditary deficiency of coagulation factors is one of the commonest causes of bleeding disorders. A careful family history of the patient will indicate the probability of such disorder. However, about 30% of them may not have a clear family history.[1] The cause for non-familial occurrence of the disease has been identified as spontaneous gene mutations. Haemophila A, Haemophila B and von Willebrand's Disease (vWD) are considered to be common bleeding disorders of inherited coagulation factor deficiencies. The commonest among them, is the von Willebrand's Disease, which perhaps presents in a ratio of one in thousand of the general population.[2] Haemophilia A, occurs with an incidence of 1 in 5000 male births. Incidence of Haemophilia B is 1 in 30,000 male births.[3] The coagulation factors that could be commonly deficient due to inherited defects and their respective coagulation disorders and the patterns of inheritance of them are given in Table 1.

Table I. Inherited coagulation disorders, respective coagulation factors in defect and their mode of inheritance

Disease	Coagulatory factor in defect	Mode of inheritance
Haemophilia A	Factor VIII	X link recessive
Haemophilia B	Factor IX	X link recessive
vWD	Von Willebrand's Factor	Autosomal dominant

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Haemophilia A and B

Haemophilia A and haemophilia B are clinically very similar disorders of two different coagulation factor deficiencies. Both disorders follow the X link recessive pattern of inheritance. Depending on the severity of the disease, bleeding into joints and intra muscular haematomas are the common manifestations of the two disorders. Haemophilia A can be differentiated from haemophilia B only by laboratory investigations. However, due to the difference in coagulation factor defect, the two disorders need two different kinds of treatment. Most of the similarities such as clinical manifestations, disease progression, and the pattern of inheritance paved the way to discuss these two disorders together for the sake of better understanding. The percentage of deficient factor levels in the plasma is used to assess the severity of both haemophilia A and B.[4] (Mild: 6-40% of factor VIII or XI, Moderate: 2-5% of factor VIII or XI, Severe: less than 1% of factor VIII or XI).

Diagnosis

Both a history of excessive bleeding after trauma or surgery and the family history of the patient provide direct and valuable information in diagnosing the disease. Attention should be focused on affected uncles, cousins and grand fathers from the maternal side of the patient in obtaining the history. As maximum number of generations as possible should be investigated in the pedigree to avoid false negative findings due to the phenomenon of skip generations. However, there may be occasions where the family history is vague. Virtually, 30% of haemophilia patients will present with a totally negative family history. As such, laboratory investigations should be compulsorily performed as the next step of diagnosis, irrespective of the findings in the history of the patient.

Laboratory investigations

The following laboratory investigations are mandatory in establishing a diagnosis.

1. Full blood count and blood picture.
2. Platelet count
3. Bleeding time
4. Prothrombin time
5. Activated partial thromboplastin time (APTT)

Those patients who have either haemophilia A or B exhibit prolonged APTT in plasma. Platelet count, bleeding time and prothrombin time remain within the range of an unaffected normal individual. Prolonged APTT is indicative of a defect in the intrinsic pathway of the coagulation cascade. Intrinsic pathway consists of four main coagulation factors, namely VIII, IX, XI and XII. Finally, factor correction tests have to be performed to precisely identify the factor in defect. Starting from the commonest factor in defect, correction tests are performed in the descending order of prevalence (factors VIII > XI > XI >XII).

Von Willebrand's disease (vWD)

Von Willebrand's Disease is the most common inherited bleeding disorder and is found in a ratio of 1 : 1000. Deficiency of von Willebrand's factor (vWF) has been identified as the cause of the disease. The disease shows an autosomal dominant mode of inheritance. Acting as a carrier protein for factor VIII and facilitation of the platelet binding onto sub-endothelial collagen in damaged vessels are the two major functions of the vWF. Both these activities would be crippled in situations where the vWF is deficient. As such clinical manifestations of the disease would show features of both a platelet disorder and a coagulation abnormality. Bleeding into the joints, intramuscular haematomas and ecchymotic patches are the common clinical manifestations of the disease. Laboratory investigations also show alterations in the tests pertaining to both a platelet disorder and a coagulation abnormality (prolonged bleeding time and APTT).

Diagnosis

Both clinical manifestations, family history as well as laboratory investigations are equally valuable in diagnosing the disease.

Laboratory investigations

The compulsory laboratory investigations and expected results in vWD are given in Table 2. (Expected alterations in the investigations in patients with haemophilia A or B are also given in the same table for ease of comparison).

Prolonged bleeding time in patients with vWD is a consequence of failure in platelet adherence to the sub-endothelial tissues, due to lack of vWF. Shortening of the half life of factor VIII protein due to lack of vWF is the other reason for the prolonged APTT in patients with the disease. However, mild variations in the results of these two investigations have frequently been observed. Fluctuation in the magnitude of both bleeding time and APTT is associated with different subtypes of the disease.[5] Subtypes and subsequent minor variations in the results will not be dealt with here, as it is beyond the scope of this article.

Management

General measures

In case of performing major oral surgeries on patients with congenital coagulation factor deficiencies, factor replacement therapy prior to the operation is the ideal treatment of choice. Multiple extractions next to each other, third molar impaction surgeries or any surgical procedure under general anesthesia with endotracheal intubations needs 100% deficient factor level in the plasma prior to surgery. Different disease entities and the appropriate form of replacement therapies are given in Table 3.

Table 2. Laboratory investigations and expected results in different inherited coagulation disorders

Laboratory investigation	von Willebrand's Disease	Haemophilia A or B
Full blood count /blood picture	Normal	Normal
Platelet count	Normal	Normal
Bleeding time	**Prolonged**	Normal
Prothrombin time	Normal	Normal
APTT	**Prolonged**	**Prolonged**
Platelet function test	**Altered**	Normal

Table 3. Types of inherited coagulation disorders and respective treatments of choice

Disease entity	Factor replacement therapy
Haemophilia A	Factor VIII concentrate or Cryoprecipitate (CP)
Haemophilia B	Fresh frozen plasma (FFP) or Factor IX concentrate
von Willebrand's disease	Cryoprecipitate (CP)

Cryoprecipitate (CP): *The Cryoprecipitate, which mainly contains Factor VIII, vWF and fibrinogen is prepared by allowing fresh frozen plasma to thaw at 4 ° C for 10 to 24 hours and then centrifuging it to separate the precipitate.*[6]

Fresh frozen plasma (FFP): *Fresh frozen plasma is produced by separating plasma from fresh blood and then immediately freezing to temperatures lower than –20 ° C. Fresh frozen plasma contains all the coagulation factors in smaller quantities.*

The deficient factor level in plasma should be maintained at a range of 30 to 50 % of a normal healthy adult to achieve haemostasis in performing minor oral surgeries such as single extractions, multiple dental extractions at different sites, deep scalings, and removal of small gum polyps etc. Administration of factor VIII concentrate is the most satisfactory and accurate factor correction method for haemophilia A patients. Intravenous administration of a single dose of factor concentrate prior to surgery is adequate to achieve the anticipated factor levels in patients with mild to moderate factor VIII deficiency. However, assessment of pre operative factor level in plasma is necessary to determine the exact dose. Repeating the same dose is recommended, only if oozing occurs from surgical wounds post operatively. Investigations to confirm that the patient is free of antibodies against factor VIII is a prime requisite before factor correction therapy.[7] Treatment of choice for patients with haemophilia B is the transfusion of fresh frozen plasma (FFP) which consists of all the coagulation factors including factor IX. Most of the clinicians are reluctant to use the commercially available factor IX concentrates due to the possibility of developing unwanted reactions such as disseminated intravascular coagulation (DIC), as of partially activated coagulation factors contained therein.

Irrespective of the extent of the surgical procedure, placement of the patient on antifibrinolytic therapy is a compulsory step in the management.[8] Anti fibrinolytic agents will inhibit the natural fibrinolytic pathway whereby formation of stable fibrin clot is indirectly enhanced. Administration of anti fibrinolytic agents 24 hours prior to the surgery and continuation of it for a period of ten days post operatively is the accepted protocol for anti fibrinolytic therapy.[9] Tranexamic acid and EACA (ε amino caproic acid) are the two best currently available anti-fibrinolytic agents in the market. In case of treating with EACA the recommended dosage is 50-100 mg/ kg body weight six hourly with an initial dose of 200 mg/ kg body weight. Dosage for the tranexamic acid is 25-50 mg/ kg body weight at eight hourly intervals.

Non-surgical dental procedures on patients with mild or moderate haemophilia can be performed only under anti fibrinolytic cover. Topical usage of tranexamic acid in combination with other therapeutic agents is always recommended for haemophilia patients after any oral surgical procedure. Recommended regime is rinsing the mouth with 5% tranexamic acid solution for two minutes, four times daily for seven days.

Desmopressin (1-Deamino-8-D-arginine vasopressin) is a synthetic analog of the antidiuretic hormone. The compound triggers the plasma levels of factor VIII and vWF after administration by releasing factor VIII contained in endothelial cells. Scaling and some equally minor surgical procedures on mild haemophilia A patients (factor V111 >5%) can be carried out under the cover of desmopressin therapy.

Parents of the children with the diseases must know that the bleeding may also occur when primary teeth are exfoliating. First aid measures such as application of pressure and usage of ice should be practised as a first attempt to control bleeding in such cases. If this is ineffective antifibrinolytic drugs may be used. However, in

rare cases, hospitalization and factor replacement therapy may be needed.

Local measures:

Whenever surgical intervention is compulsorily indicated precautions should be taken to prevent or minimize unnecessary tissue damage. Apposition of wound margins by suturing is an added advantage in obtaining haemostasis. Application of fibrin glue into the extracted socket will be helpful to achieve a good sealing of the wound. Fibrin glue is a therapeutic blood product available as two separate compounds which develops an artificial clot when mixed at the site of the injury. In relation to oral surgical intervention, it is of paramount importance that there is the least possible interference with the attached gingiva around teeth and the periosteum. Simply shifting the attached gingiva from the underlying tissue or periosteum even in healthy patients causes excessive post-operative bleeding. The best is key hole surgery where there is as little interference as possible with the attached gingiva, to minimize post operative bleeding. Patients should be advised to take a diet of cold liquids and minced solid foods for 5 to 10 days. Being advised to refrain from smoking is equally important. Pain killers such as ASA (Acetyl salicylic acid) or NSAIDs (Non Steroidal Antiinflammatoy Drugs) that could aggravate bleeding should not be prescribed. Paracetamol/ acetaminophen and codeine are the pain killers of choice. Patients should be advised to complain any swelling, difficulty in swallowing, or hoarseness of the voice immediately.

Preventive measures:

Good oral hygiene and restrictive dietary habits are essential to prevent both dental caries and gingival/periodontal diseases in haemophilia patients. Complying with the primary health care measures have been identified as the best method of approach in preventing dental disease in the first instance.[10] The haemophilia patients themselves, together with their immediate family members and friends should be targeted in the process of delivering health education programmes. Implementing dietary restrictive attitudes in patients with haemophilia and guardians of them, applying fissure sealants, administration of fluoride via community water fluoridation, local application of fluoride (gel application) and usage of fluoridated tooth pastes have been recognized as the most effective primary health care measures in preventing dental disease. Patients must be aware of the need of regular dental visits at least once in six months for the early identification of treatment needs. The haemophilia patients, parents and guardians of them as well as the haemophilia community as a whole should be educated to realize the importance of bringing these health care habits into practice early in the life for their own benefit in the long term[10]. Dental appointments for children with bleeding should be started when deciduous dentition begins to appear.

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Review Article

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Aetiology of nursing caries: a review

E.M.U.C.K Herath

Abstract

Nursing caries is an infectious and transmissible disease. Scientific evidence shows those infants whose oral cavities are colonized by mutans streptococci (MS), and those who have feeding habits characterized by frequent and prolonged oral exposure to cariogenic substrates, are likely to have a significant increase in mutans streptococci population in their mouths. Such an increased microflora is associated with a high risk for rampant dental caries. Dental caries is a result of destruction of tooth structure by acid-forming bacteria, such as mutans streptococci and lactobacilli, found in dental plaque, in the presence of sugar.The aetiology of nursing caries is very complex. A number of risk factors have been identified. From the dental surgeon's point of view, it is of paramount importance to identify the children at risk and introduce both prevention and control of the disease at the onset.

Key words:

Nursing caries, early childhood caries

Introduction

Nursing caries is a infectious and transmissible disease that affects infants. It is associated with prolonged and frequent consumption of milk or any kind of cariogenic substrates during bed time and/ or nap time beyond 12 months of age.[1] Mothers in western societies, have got used to filling the feeding bottle of their babies with sweetened milk, fruit juice, or some other sweetened solutions. However, in eastern societies, nursing caries is mainly due to breastfeeding in the night time.[2] Nursing Caries is also defined as a severe type of early childhood caries (ECC).[3,4] It is generally agreed that nursing caries constitutes rampant dental caries in association with continuation of inappropriate bottle or breast feeding even after weaning starts. Terms such as nursing bottle mouth, baby bottle tooth decay, nursing bottle caries, night bottle mouth, and nursing bottle syndrome are used to describe severe forms of early childhood caries.[2] Some of these terms are uniquely specific to the behavior that may lead to development of early childhood caries. However, baby bottle tooth decay has been the term of choice among lay people because it is easily understood.[1] It is a particularly virulent form of dental caries which rapidly destroys teeth within a short period as a result of improper feeding habits.[2] The use of milk or sweetened drinks in baby bottles and/or inappropriate breastfeeding is associated with the condition.[1,5] Nursing caries shows a particular pattern of smooth surface dental caries in which the upper primary incisors and upper first primary molars are usually affected the most. The lower first primary molars are also often involved. The reason for this is the continues bathing of the teeth in the upper anterior segment in breast milk or sweetened liquied in frequent feeding. However, the lower incisors are

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usually spared from caries or only mildly affected as a result of the shielding effect of the tongue during suckling. Teeth in the lower anterior segment are further protected from caries owing to the cleansing effect of saliva from sublingual and submandibular glands.[1]

However, there is a group of children who commonly present with extensive destruction of teeth without typical features of nursing caries.[6] Such children are slightly older (3-4 years) and often have multiple carious teeth with inter dental involvement at the initial presentation. This entity of caries is known as rampant caries. There is however, no clear demarcation between rampant caries and nursing caries and as such the term "early childhood caries" has been suggested as the most appropriate term which encompasses both entities.[6] The purpose of this paper is to review the aetiology and preventive measures of nursing caries.

The American paediatrician Jacobi was the first to describe nursing caries in 1862. However, Milnes cited that it was Fass (1962) who gave the modern description of nursing bottle syndrome.[2] Although nursing caries had been described since 1960s, it has not received adequate attention from health professionals other than paedodontists. The declining caries incidence among children who live in industrialized world is well documented.[4] However, children in developing countries such as Sri Lanka still experience higher rate of tooth decay.[7] Unfortunately, primary teeth are not considered as much important as the permanent teeth in developing countries.[2] An added disadvantage a high rate of early childhood caries in developing nations is that the dental health of pre-school children in whom nursing caries is more common is basically ignored. Both a shortage of health personnel and the lack of awareness among parents are the main reasons for this.[7]

Aetiology of nursing caries

W.D. Millar (1890) explained the chemoparasitic theory of dental caries[2,10] and Black laid the foundation for operative dentistry based on Millar's theory.[10] At present Millar's chemoparasitic theory is commonly known as the acidogenic theory of caries. According to the acidogenic theory the main features of caries process are;

1. Fermentation of dietary sugars to organic acids by microorganisms in plaque on the tooth surface

2. Rapid acid formation, which lowers the pH at the enamel surface below the critical level at which enamel will dissolve

3. When sugar is no longer available for microorganisms, the pH within plaque will increase due to the outward diffusion, metabolism and neutralization of acids in plaque. This leads to remineralization of enamel.

4. Dental caries progresses only when demineralization is greater than remineralization.[2] (Fig. 1)

A better understanding of the scientific background of early childhood caries contributes to the diagnosis and effective management of children with the disease. Four aetiological factors are responsible for caries. These include tooth, bacteria, substrate and duration of exposure to the substrate. Demineralization of teeth (caries) only occurs in the presence of all four factors. However, the effect of these four factors can be altered by a set of modifying factors like fluoride, chlorhexidine, constituents and the rate of salivary flow (Fig.1).

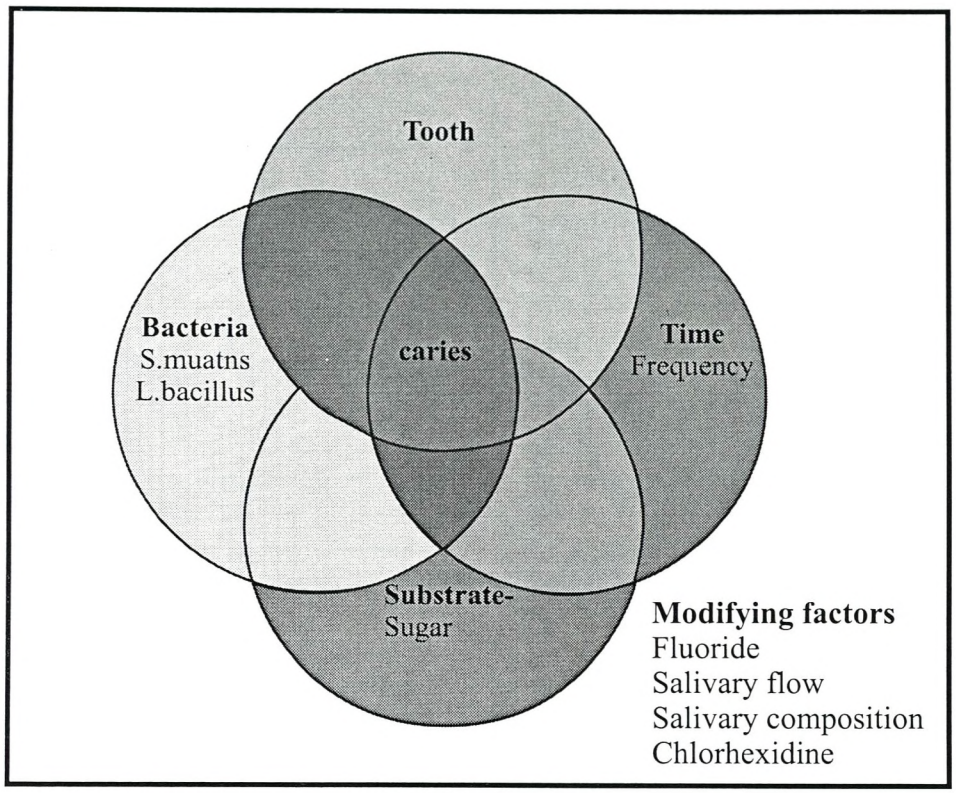

Figure 1. Factors affecting caries development

Role of micro-organisms in nursing caries

Studies on animals have clearly demonstrated that dental caries can be characterized as an infectious and transmissible disease. Infection is quite specific and commonly involves MS. Strains of this species isolated from dental plaque and caries lesions in human, constantly exhibit a high level of pathogenicity when tested on rodent models under optimal conditions. Clinical studies on spread of caries further demonstrate that a significant increase in oral MS levels conversely relate to the onset of dental caries. Based on these findings, several investigators have used salivary levels of MS to predict the caries risk.[12]

The odontopathic potential of these bacteria also relates to their adherence characteristics, higher potential of acidogenicity and aciduric nature. Collectively the available information indicates that MS are the primary aetiological agent in human dental caries. Other potentially cariogenic bacteria are also found in dental plaque. These organisms are weakly competitive and proportionately smaller when compared to the total plaque community. Levels of all cariogenic bacteria become clinically insignificant, with a balanced diet (low sugar) of a child and the process of demineralization and remineralization remains in equilibrium. When demineralization and remineralization remains in equilibrium there is no tooth decay. When the frequency of fermentable carbohydrate intake increases, the plaque pH falls below the critical level (approximately 5.5) and the process of enamel demineralization starts. The process of demineralization distroys the tooth surface.

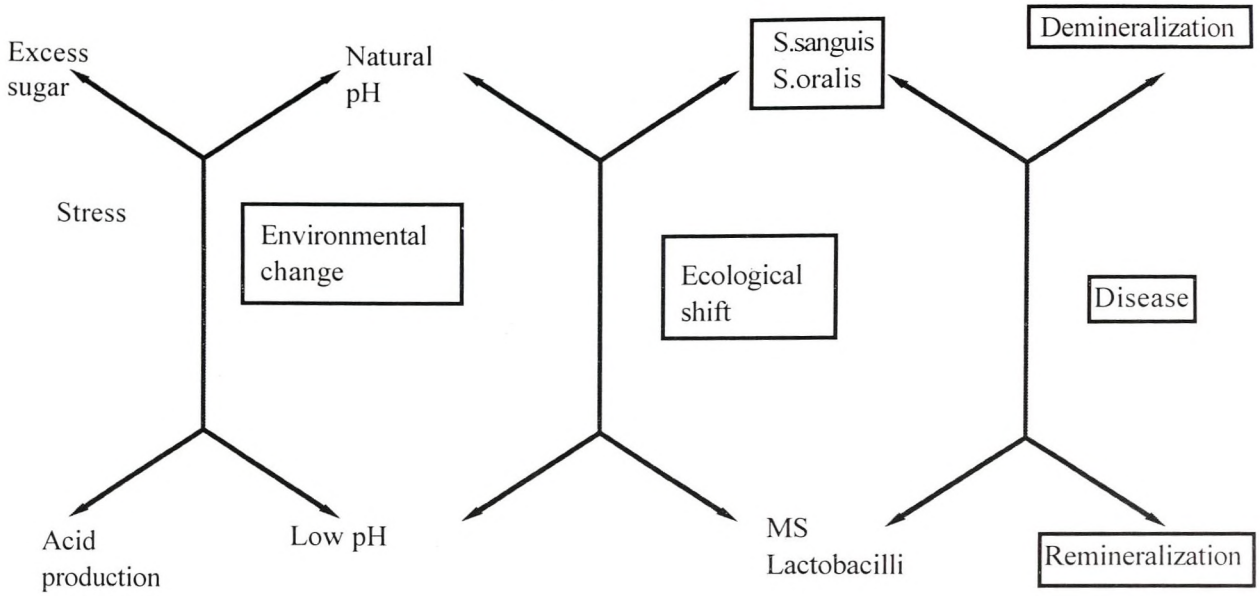

Figure 2. The ecological hypothesis and the etiology of dental caries

A reduced plaque pH (below the critical level) favors doubling of the density of cariogenic microbial flora by encouraging the proliferation of aciduric bacteria such as MS and lactobacilli.[13] Amplified aciduric microbial flora further shift the balance towards demineralization. This kind of change in the natural balance of the oral flora in relation to cariogenic process was described by Marsh (1994) under his ecologic plaque hypothesis.[9] Marsh indicated that caries occurs as a result of a change in the natural balance of the resident plaque microflora which is brought about by an alteration in local environmental conditions. Under the hypothesis, existing dynamic relationship between the oral microbial flora and the host was also taken in to consideration. Impact of alterations in host factors such as salivary flow rate and composition of saliva on plaque composition is also taken into account (Fig. 2).

The fundamentals of the ecological plaque hypothesis have a significant impact on prevention of caries. It shows that the disease can be controlled not only by directly targeting the pathogens but also by interfering the factors that are driving the deleterious shift in the balance of the microflora. Maintenance of the balance in microbial flora and thereby reduction in the development of caries can be achieved by controlling the modifying factors: eg: lowering the acid challenge by reducing frequency of sugar intake, and/or by increasing salivary flow rate.[10] Night feeding, or inappropriate feeding of sugary diets continuously provide a substrate for aciduric bacteria for colonization. The final result would be an ecological shift in the environment of the oral cavity and enhancement of the demineralization process.

van Houte observed a high level of MS density in experimentally developed caries lesions on teeth of hamsters who were fed with a high sucrose diet.[12,13] Experimental studies on human have repeatedly demonstrated that more than 50% of the cultivable oral microbial flora is represented by the MS in children with nursing caries.[1] In contrast, it has been shown that MS represent

only less than 1 % of the plaque flora in children with negligible level of caries. Both experimental and other circumstantial evidence proves that nursing caries is a specific infection caused by MS which is one of the aetiological agents of the disease. It is further highlighted that those infants, in whom the oral cavities are colonized by MS are at risk for nursing caries.[12]

Primary oral infection

Oral cavities of predentate infants are transiently contaminated[12] or free from MS.[10] Experimentally it has not been possible to isolate MS from mouths of normal predentate infants.[13] The lack of hard surfaces for adherence, a feeble capacity of attachment to epithelial surfaces and washout effect of the saliva are the main reasons for this.[15,16] However, colonization by MS has been detected in the oral cavities of predentate infants who used appliances such as cleft-palate obturators. Gradual establishment of MS population has been observed with the eruption of the primary teeth. Collectively, the available information indicates that, although MS can contaminate the mouth transiently in infants, these organisms require a non-shedding hard surface for their persistent colonization. Therefore, they establish in the human mouth only after the eruption of primary teeth.[12] Most of the time babies get infected with cariogenic bacteria at the time of tooth eruption from mothers who have poor oral hygiene status. Hence, it is important to maintain good oral hygiene during and after pregnancy in order to minimize the transmission of *Streptococcus mutans* from mother to child.

Role of cariogenic substrate and time factor in nursing caries

Both human and laboratory animal investigations have demonstrated that frequent and prolonged oral exposure to cariogenic substrates facilitates the intraoral accumulation of aciduric bacteria and accelerates dental caries activity.[1,4] Further they highlighted that accumulation of microflora to pathogenic levels precedes the onset of clinical disease. These experimental observations support the concept that infants who are colonized by MS and whose feeding habits are characterized by frequent and prolonged consumption of cariogenic substrate are likely to have a drastic increase in their oral MS population. Such a microbial shift is associated with a high risk of nursing caries.[6,10] Development of nursing caries is associated with prolonged breast feeding on demand too. Breast milk which contains lactose around 7% favors the acid production in the presence of aciduric bacteria. However, breastfeeding up to around 1 year during which the primary dentition has not been established is not harmful for the teeth. Experiments on animal models have shown that cow's milk which contain 4% of lactose is not much cariogenic compared to the breast milk. However, some clinical studies have suggested that the inappropriate consumption of cow's milk at night using the feeding bottle might be associated with nursing caries.[6] It is shown that children who go to sleep with the bottle or nipple in their mouth, are more likely to get nursing caries. Lack of muscular activity and reduction in salivary flow during the sleep has been identified as the reasons for this.[1] Long-term use of medications containing sugar, also prvide substrate for the development of caries.

Tooth factor for nursing caries

Hypoplastic teeth or teeth with defects are more susceptible to caries. Hypoplastic teeth are common in children who are suffering from protein energy malnutrition As hypoplastic teeth are more liable to enamel cavitation, tooth decay is common in children with protein energy malnutrition. Presence of fluorides enhances the remineralization process against demineralization (caries) and reduce enamel cavitation. Additionally teeth can be made stronger by introducing fluorides which will change the tooth structure so that it can withstand acid attacks.[10]

Conclusion

Nursing caries continues to be a major public health problem on a global scale. It is important to address the aetiological factors to prevent the

disease. The first step in the aetiology of nursing caries is the primary infection by mutans streptococci; the second step is accumulation of these organisms to pathogenic levels as a consequence of frequent and prolonged exposure to cariogenic substrate; and the final step is the rapid demineralization and cavitation of enamel resulting in rampant dental caries. Therefore, prevention of nursing caries can be achieved by avoiding the primary infection by mutans streptococci and controlling the proliferation of the micro-organism by modifying or stopping inappropriate feeding. Accordingly, the development of a preventive regime, targeting microbial risk factors, dietary factors and to a lesser extent tooth factors (making tooth stronger) would result in a more widespread and successful approach towards prevention.

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Research Article 1

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Intra examiner reliability of two methods of occlusal classification

N. S. S. Jayasooriya and N. Nagarathne

Abstract

Objective: The purpose of the present study was to determine the intra examiner reliability in using Angle's classification and British Standard Institute's Incisor classification and also to determine the preferred system of classification for undergraduate clinical setting.

Material and Methods: Thirty sets of study models which included all classes and subdivisions of Angle's classification and British Incisor classification were selected from records taken between 1994 and 2003 at the Orthodontic Unit, Faculty of Dental Sciences, University of Peradeniya. Four groups of examiners (four postgraduate students, four graduates, four final year and four third year students) were asked to classify occlusions of these models using both systems of classifications on two different occasions. Procedure was repeated two weeks later to assess intra examiner reliability.

Results: Postgraduate students showed the mean highest reliability ratio and percentage agreement in using Angle's classification (24:30 and 79.99%). Correlation coefficient and Kappa values showed a substantial level of agreement. The third year students showed the lowest reliability ratio and percentage agreement (19:30 and 63.33%). Kappa values indicated moderate level of agreement. When British system of classification was considered Reliability ratio, Percentage agreement, Correlation coefficient and Kappa values were greater in all groups than with the Angle's classification.

Conclusion: The findings of this study revealed that the British Standard Institute's Incisor classification system is superior to the Angle's classification system when intra examiner agreement is considered. British Standard Institute's Incisor classification system is the most reliable classification system for use in undergraduate clinical setting.

Key Words: Occlusal classification, Reliability

Introduction

Occlusal and facial pattern vary widely among individuals even in the same community. Therefore, it is important to categorize them into groups. The objective of any system of classification is to gather together the individuals with similar features or with a common aetiology.[1] However, as with any other biological system, in occlusal and facial pattern, there is a spectrum of continuous variation, making designation of borderline cases difficult leading to low intra-examiner and inter-examiner reliability in using the classification systems. Different classification systems are used for different purposes.[2]

The most widely used classification system is that proposed by Edward Angle.[3] Angle's classification is based on the positional relationship between the first permanent molars in maxillary and mandibular

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arches. This system of classification has been widely criticized. Many challenged Angle by developing their own system of classification.[1,4,5,6] Others have criticised Angle's classification and have not proposed a better method of classification.[7,8,9] Angle's classification fails to distinguish between similar antero-posterior discrepancies, which need different treatment modalities. Angle's classification also had been challenged because it only addresses sagittal dental dimension and does not address other two planes of space, which determines the final facial appearance.

In order to improve drawbacks of Angle's classification, incisor classification was developed. The incisor classification was first described by Ballard and Wayman,[10] which is based on the positional relations of the incisor teeth; rather than the first molars. This avoids the problems associated with drifted molars in a crowded arch. It is also mainly descriptive. Later it was adopted as British Standards Institute's Incisor classification of malocclusion.[11] Inter and intra examiner reliability of the British Standard Institute's Incisor classification also had been questioned. Later, Williams and Stephens.[12] have suggested inclusion of class II intermediate category to improve the examiner reliability of incisor classification.

At present, Angle's classification and British Standards Institute's Incisor classification are the classification systems widely used in orthodontic diagnosis especially in undergraduate clinical settings. Both classification systems have their own drawbacks for the use as diagnostic classification systems. It is important to bear in mind all these drawbacks of both systems of classifications, which are encountered when they are being used by undergraduates in orthodontic diagnosis.

The purpose of the present study was to determine the intra examiner reliability in using Angle's classification and the British Standards Institute's Incisor classification for the purpose of diagnosis in clinical orthodontics. This study also attempts to identify the preferred method of occlusal classification system for use in undergraduate clinical setting.

Material and Methods

Thirty study models were selected from a large pool of orthodontic records taken between year 1994 and 2003 by the second author for orthodontic treatment at the Orthodontic Unit, Faculty of Dental Sciences, University of Peradeniya. Models were selected purposely to include cases, which posed difficulties in diagnosing and also to include all classes and subdivisions of both classification systems. Thirty sets of study models were included in the final sample. Examiners included four postgraduate students and four dental graduates. Two groups of dental students who have completed clinical appointments in orthodontics were selected from both final year and third year students.

Each examiner was given a set of written and verbal instructions for the use of both classification systems. Angle's classification system included seven categories and British classification system included five categories. Different sets of study models were used for the training session. Each examiner was given adequate time to familiarise with both systems of classifications. No attempt was made to reach a specific level of proficiency in applying the classification systems. All examiners were asked to rate thirty sets of models using both classification systems and record the findings individually. Rating of the study models were done under standardized conditions. One system of classification was used at a time to exclude the possibility of confusion between ratings of the two systems. The procedure was repeated two weeks later by all examiners on all study models using both systems of classification. Data analysis was done using SPSS version 10, statistical package. Intra examiner reliability was assessed using reliability ratio, percentage agreement, Spearmann's correlation coefficient and Kappa values.

Results

Results of the intra examiner reliability in using both classification systems are shown in table 1, 2, 3, 4 and 5. Tables 1, 2 and 3 show the mean

reliability ratios and the mean percentage agreement whole group and ratings of individual examiners. The reliability ratio is represented as a fraction of the study casts rated consistently at two rating sessions by one examiner out of the total study casts included in the study. Postgraduate students scored the highest mean reliability ratio (24:30), when compared with the other three groups in using Angle's classification. Graduates, final year students and third year students showed mean reliability ratios 20:30, 23:30 and 19:30 respectively in using Angle's classification. Mean percentage agreement was calculated using reliability ratio, which ranged from 63.33% to 79.99%. Postgraduate students had the highest percentage agreement and third year undergraduates showed the lowest percentage agreement in using Angle's classification. When British Standard Institutes Incisor classification was used the mean reliability ratio ranged from 20.25:30 to 26.75:30 and the mean percentage agreement ranged from 67.5% - 89.14%.

Table 4 and 5 show the comparison of ratings of four groups of examiners in using both classification systems. All four postgraduate students showed a satisfactory level of agreement in using Angle's classification (Correlation coefficients 0.725-0.784). Kappa values, which ranged from 0.65 to 0.78 indicated a substantial level of agreement in two rating sessions for postgraduate students group in using Angle's classification. Graduates showed a moderate level of agreement. Coefficient of correlation and Kappa values of final year students showed a comparable result with postgraduate students (Kappa values 0.65-0.748). The third year students also showed a moderate level of agreement. When British Standard Institute's Incisor classification was considered all four groups of examiners showed a higher level of agreement compared to Angle's classification. Postgraduate students showed the highest reliability ratio and percentage agreement. Final year students showed both reliability ratio and percentage agreement which are comparable with postgraduate students. Correlation coefficients and Kappa values indicated very high level of agreement in postgraduate student group except for the second examiner. Among the final year students, second and third examiners showed very high level of agreement (Kappa values 0.946 and 0.902 respectively). The other two showed a substantial level agreement (Kappa value .685 and 0.763). Third year students also showed a substantial level of agreement.

Discussion

Angle's classification describes malocclusions using the antero-posterior positional variations of maxillary and mandibular molars. This divides the malocclusion into three main categories, namely class I class II and class III. Depending on the relationship of the incisor teeth, class II malocclusion is further subdivided into two divisions, division 1 and division 2, which results in four categories namely, class I, class II division 1, class II division 2 and class III. Depending on the molar relationship on both right and left sides they are further divided into subgroups, which result in seven categories. Though, Angle's classification describes features of malocclusion of each category it is grouped into seven classes which, results in a discrete variable.

British incisor classification is based on the relationship of the upper and lower incisors. This divides malocclusions into three classes namely, class I, class II and class III. Class II is subdivided into two divisions, division 1 and division 2. Class II indefinite was added later to overcome the problem of variation of inclination of upper incisors, which resulted in five categories. Though the British classification system is also descriptive in nature, each category is given a number resulting in a discrete variable. As both these classification systems deal with discrete variables it was easy to make direct comparison unlike a classification system, which uses a continuous variables, for example Katz.[13] It is important to note that the models assessed in this study were purposely selected from a large pool of orthodontic records available in the clinic in order to include all categories of malocclusions, which is uncommon to see in day-to-day clinical practice.

Findings of the present study revealed that the intra examiner reliability of Angle's classification is lower than the British Standard Institute's incisor classification. There are many reasons for this. Angle used the relationship of the mesio buccal cusp of the upper first molar with the mesial groove of the lower first molar as the key relationship of the Angle's classification. Angle's class I is defined when mesio buccal cusp of the upper first molar occludes with the mesial groove of the lower first molar. Class II or III is defined when lower molars are at least half a cusp width either anterior or posterior to normal relationship. But in nature the occlusal relationship does not fall into these three discrete categories. The judgement of a case, which has less than half a cusp discrepancy is difficult and subject to examiner variability. This ambiguity leads to a low intra and inter examiner reliability.[7] In Angle's classification both right and left molar and incisor relationships should be examined before determining the final classification. Even a slight error in identifying one aspect may add to the variability. But in the British system of classification only incisor relationship is examined and three categories are identified looking at the occlusal relationship of incisor edges of lower incisors to the cingulum plateau of the upper incisors. Two divisions are identified. by examining the inclination of incisors. As only incisors are examined the chances of making an incorrect judgment is reduced when compared with Angle's classification. Further, Angle's system has seven categories, which can lead to a mathematical disadvantage when compared with British system of classification, which has five categories when reliability ratios are calculated.

Though there are many publications discussing ambiguities of Angle's system of classification only few studies have been published in determining its reliability. Many studies have been carried out using questionnaires investigating opinions of the orthodontists regarding the classification systems. The only study, which is available in published literature is the study carried out by Due *et al.*(1998),[14] They have investigated the intra examiner reliability of Angle's classification, British Incisor classification and Katz classification using study models. Out of the three methods Katz classification was found to be the best classification system with regard to intra examiner reliability. British incisor classification was found to be superior to Angle's classification. In the present study Katz classification was not included due to two main reasons. Katz classification uses a continuous variable, which leads to difficulty in direct comparison unlike the other two systems of classification and only Angle's classification and British incisor classification are used at present in undergraduate clinical setting. Findings of the present study agree with the findings of the study carried out by Due *et al.*(1998), supporting the low intra examiner reliability of Angle's classification.

Conclusion

When findings of all reliability ratios, percentage agreements, correlation coefficients and Kappa values were considered the postgraduate students showed the highest level of intra examiner reliability in using both systems of classifications. Final year students showed moderate to substantial level of agreement in the use of Angle's classification and substantial to very high level of agreement in using British system of classification. British system of classification showed a higher level of intra examiner reliability when compared with the Angle's classification in all four categories of examiners. The results of the study revealed that British Standards Institute's incisor classification system is superior to Angle's classification system when intra examiner reliability is considered. British Standards Institute's Incisor classification is the most suitable classification system for use in undergraduate

clinical setting as it has a higher level of intra examiner reliability.

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Table 1. Mean reliability ratios and percentage agreement in classifying 30 sets of study models using two classification systems

	Angle's Classification		British Incisor Classification	
Examiner	**Reliability Ratio**	**Percentage Agreement**	**Reliability Ratio**	**Percentage Agreement**
Postgraduate	24:30	79.99	26.75:30	89.17
Graduates	20:30	66.66	23:30	76.66
Undergraduates (Final year)	23:30	72.66	26.5:30	88.33
Undergraduates (Third year)	19:30	63.66	20.25:30	67.50

Table 2. Intra examiner reliability in classifying 30 sets of study models using two classification systems (Reliability ratios)

	Angle's classification				British incisor classification			
Examiner	**1st**	**2nd**	**3rd**	**4th**	**1st**	**2nd**	**3rd**	**4th**
Postgraduates	24:30	25:30	22:30	25:30	26:30	26:30	29:30	26:30
Graduates	20:30	19:30	22:30	19:30	23:30	25:30	24:30	20:30
Undergraduates (Final year)	23:30	22:30	24:30	23:30	24:30	29:30	28:30	25:30
Undergraduates (Third year)	20:30	19:30	19:30	18:30	20:30	19:30	22:30	20:30

Table 3. Intra examiner reliability in classifying 30 sets of study models using two classification systems (Percentage agreement)

	Angle's classification				British incisor classification			
Examiner	**1st**	**2nd**	**3rd**	**4th**	**1st**	**2nd**	**3rd**	**4th**
Postgraduates	80.00	83.33	73.33	83.33	86.66	86.66	96.66	86.66
Graduates	66.66	63.33	73.33	63.33	76.66	83.33	80.00	66.66
Undergraduates (Final year)	76.66	73.33	80.00	76.66	80.00	96.66	93.33	83.33
Undergraduates (Third year)	66.66	63.33	63.33	60.00	66.66	63.33	73.33	66.66

Table 4 . Intra examiner reliability in classifying 30 sets of study models using two classification systems (Correlation coefficient)

	Angle's classification				British incisor classification			
Examiner	**1st**	**2nd**	**3rd**	**4th**	**1st**	**2nd**	**3rd**	**4th**
Postgraduates	0.764	0.784	0.725	0.784	0.846	0.846	0.941	0.931
Graduates	0.689	0.687	0.725	0.687	0.799	0.824	0.800	0.587
Undergraduates (Final year)	0.764	0.725	0.8	0.764	0.799	0.960	0.852	0.819
Undergraduates (Third year)	0.689	0.687	0.687	0.478	0.587	0.687	0.725	0.587

Table 5. Intra examiner reliability in classifying 30 sets of study models using two classification systems (Kappa values)

	Angle's classification				British incisor classification			
Examiner	**1st**	**2nd**	**3rd**	**4th**	**1st**	**2nd**	**3rd**	**4th**
Postgraduates	0.730	0.778	0.65	0.778	0.826	0.811	0.948	0.826
Graduates	0.562	0.559	0.65	0.559	0.682	0.768	0.748	0.587
Undergraduates (Final year)	0.682	0.65	0.748	0.682	0.685	0.946	0.902	0.763
Undergraduates (Third year)	0.662	0.559	0.559	0.400	0.55	0.559	0.65	0.562

Research Article 2

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Serum IgG subclass response to RgpA-Kgp complexes of *Porphyromonas gingivalis* in periodontitis patients

P. S. Rajapakse and E. C. Reynolds

Abstract

Objective: The objective of the study is to evaluate the IgG subclass response to gpA-Kgp complexes in a group of patients with chronic periodontitis and age and sex matched subjects with healthy periodontal tissues with no loss of connective tissue attachment.

Material and methods: Test group consisted of 33 subjects (16 females and 17 males, mean age 36.4 yrs (SD ± 11.3) with chronic severe periodontitis designated by the presence of at least one probing pocket measurement with 6 mm. Age and sex matched subjects with healthy periodontium (mean age 33.6 SD ± 10.8) served as the control group. Serum Immunoglobulin G (IgG) and IgG subclass response to the RgpA-Kgp Proteinase-adhesin complex of *Porphyromonas gingivalis* W50 was examined using sera from patients with chronic periodontitis. Periodontal pocket depths and number of bleeding sites were recorded.

Results: Significantly higher levels of serum IgG, IgG2 and IgG4 responses against RgpA-Kgp complexes were found in sera of periodontitis patients compared to that of healthy subjects. However, the IgG1 and IgG3 responses were very low and close to back ground levels. Serum IgG2 and IgG4 levels were positively correlated with average probing depth and percentage bleeding sites.

Conclusion: In the study group the differences in antibody profile to tested antigen in diseased and periodontally healthy control subjects are apparent. In the patient group chronic exposure to *P. gingivalis* is evident by higher IgG4 response. Whilst higher IgG2 and IgG4 may not have been helpful in the protection against pathogenic bacteria due to their poor complement activation and Fc binding properties, the failure to mount an adequate level of functionally effective antibody response with IgG1 and IgG3 may have rendered the subjects in the diseased group susceptible to periodontitis.

Key words

RgpA-Kgp complexes, periodontitis, IgG subclasses, *P.gingivalis*

Introduction

Periodontitis is an inflammatory disease of the supporting tissues of the teeth and is a major cause of tooth loss in adults.[1] Emergence of a consortium of specific gram negative organisms has been associated with the onset and progression of

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chronic periodontitis. *Porphyromonas gingivalis* is considered one of the major pathogens in the aetiology of the disease in human.[2] and it is capable of inducing disease in experimental models of periodontitis[3,4,5] Several studies have reported higher antibody titres (immunoglobulin G [IgG], IgM, and IgA) to *P. gingivalis* whole cells and outer membrane preparations in sera from adult periodontitis patients than in sera from healthy subjects.[6,7,8] Furthermore, the severity of periodontitis has been associated with an increased IgG response to *P. gingivalis.*[9,10] Few studies have investigated the antibody response to purified antigens from *P. gingivalis*. Schenk and Michaelsen have reported that sera from patients with periodontitis had elevated IgG titres to purified *P. gingivalis* lipopolysaccharide (LPS) with an IgG isotype distribution of IgG2 >> IgG1 > IgG3 > IgG4. An IgG subclass distribution dominated by IgG2, followed by IgG3 > IgG1 > IgG4, has also been reported; the distribution was determined by using periodontitis patient sera against a *P. gingivalis* whole-cell *sonicate*[11] and against a *P. gingivalis* outer membrane preparation.[12] All these preparations, however, contained significant amounts of LPS, which is known to induce a dominant IgG2 subclass response.[13].

Ogawa *et al.*[14] (1990) have also reported that IgG2 is the dominant subclass response against *P. gingivalis* LPS and that the IgG subclass distribution against a purified fimbrial protein was IgG3 > IgG1 > IgG2 > IgG4. However, in an earlier report by the same group, the fimbria-specific IgG subclass distribution was found to be IgG4 dominant, followed by IgG1 > IgG3 > IgG2.[15] A study carried out in our laboratory with respect to analysis of the IgG subclass responses to the RgpA-Kgp complex revealed that the subclass distribution for both the diseased and control groups was IgG4 > IgG2 > IgG3 = IgG1. The IgG2 response to the complex positively correlated with mean probing depth, whereas the IgG4 response was negatively correlated with this measure of disease severity.[10]

This study investigates the IgG subclass response to native RgpA-Kgp proteinase–adhesin complex of *P. gingivalis* W50 in a group of otherwise healthy patients with moderate to severe chronic periodontitis and age and sex matched subjects with healthy periodontal tissues. The results would be helpful in profiling the patttern of IgG subclass response to RgpA-Kgp complex in this particular study group.

Material and methods

Serum total IgG responses and IgG subclass responses to different *P.gingivalis* antigens were determined using enzyme linked immunosorbant assay(ELISA). Antigens used were formalin killed whole cell RgpA-Kgp complexes of *P.gingivalis* W50. All assays were performed by titration and duplicate measurements were determined for each dilution used.

Preparation of antigens

Preparation of Formalin Killed Whole Cells (FKWC)

Preparation of *P. gingivalis* ATCC 33277 FKWC: Cells were treated overnight with equal volumes of 0.5% (vol/vol) formal saline on a rocking platform. Sterile PG buffer (equivalent to 10 times the volume of treated cells) was then added and the mixture was centrifuged for 10 min at 10,000 x g. The supernatant was removed, and the cell pellet was resuspended gently in PG buffer (20 times the volume of the cell pellet) and centrifuged again for 10 min at 10,000 x g. After the supernatant was discarded, the cells were resuspended in sterile PG buffer to obtain a concentration of 1010 CFU in 0.1 ml. For immunization, the cell suspension was mixed with incomplete Freund's adjuvant (IFA) at a ratio of 1:1 (vol/vol).

Preparation of RgpA-Kgp complexes

The RgpA-Kgp complexes were prepared from a P. gingivalis W50 cell sonicate by anion

exchange, gel filtration, and Arg-Sepharose chromatography as described by Bhogal et al.[16] (1997). The purified RgpA-Kgp adhesin complexes were characterized by sodium dodecyl sulfate (SDS) polyacrylamide gel electrophoresis,[17] and blotting onto a polyvinylidene difluoride membrane, followed by N-terminal sequence analysis as described by Bhogal et al.[16] (1997). Protein concentrations of samples were determined using the Bradford protein assay (Bio-Rad, Richmond, Calif). Proteolytic activity of samples was determined using synthetic chromogenic substrates as described by Bhogal et al. [16] (1997).

Study group

Otherwise healthy subjects were included in this study and with respect to female subjects, those who were pregnant were excluded. Serum samples from a total of 33 subjects were available for study and the age range was 18 years to 53 years with mean age of 36.4 years (SD ± 11.3). There were 16 females and 17 males. Majority (29) of the diseased sample (mean age 37.7 and SD ± 11.6) was collected from amongst patients attending the periodontal clinic and the subjects with healthy periodontium (mean age 33.6 SD ± 10.8) were drawn from amongst the staff and students of the Faculty of Dental Sciences, University of Peradeniya, Sri Lanka.

Criteria for selection

The subjects were characterized as having severe chronic periodontitis or as healthy according to the existing probing pocket depth. A diagnosis of periodontitis was based upon the presence of at least one periodontal probing pocket of 6 mm or more and numerous sites with bleeding on gentle probing. Healthy subjects had no pathological pockets (no gingival probing depths over 3 mm) with minimal number of sites with bleeding and had no connective tissue attachment loss in relation to any of their teeth. None of the subjects had any systemic illness that could affect the status of the periodontium and had not recently used medications that could affect their periodontal status.

Clinical examination and collection of blood sample

All tooth surfaces (six surfaces per tooth) were examined for evidence of periodontal involvement and pocketing and bleeding were recorded in relation to all six sites. Blood samples were drawn from all subjects by antecubital fossa venepuncture. Blood samples taken were kept at 37°C for one hour and then in a refrigerator at 4° C overnight. Serum was separated by centrifugation at 3000g for 10 minutes at 4° C and stored at -70° C in 0.2 mL aliquots until used in enzyme linked immunosorbant assay (ELISA).

ELISA

Serum samples were assayed for the presence of antibodies against formalin-Killed Whole Cells (FKWC) and RgpA-Kgp complexes using an ELISA technique. 1μg/mL solutions in Tris buffered saline (TBS) was used as coating concentration for RgpA-Kgp complexes.

The day prior to the assay the wells of the microtitre plates were coated with antigen (50 μl/well in TBS) using a 12 channel pipette (Titertek/Finn pipette). The microtitre plates were sealed with cling film and incubated overnight at 4°C in a humidified chamber.

The antigen solutions were removed from the wells and the plates were washed three times using TBS and a 12 channel pipette was set to deliver 250 μl per channel. This was the standard washing procedure used throughout the study. The wells were then coated with 200 μl of blocking solution containing 2% w/v gelatine in TBS (gelatine as the blocking agent was decided empirically), sealed and incubated at 37° C for 2 hours. The blocking solution would block any unoccupied binding sites on the plate so that the primary or secondary antibody would not bind non-specifically to those sites. The plates were washed three times as before and the wells

coated with 50 µl of serially diluted patients sera in antibody diluting buffer (TBS with 1% gelatin). Control wells, received antibody diluting buffer only. The plates were sealed and incubated for 90 min at 37° C. Following incubation the plates were washed three times as above and 50 µl secondary antibody solutions were placed in appropriate wells. Antibody diluting buffer only was placed in wells not receiving secondary antibody. The secondary antibodies used were biotyinylated mouse anti human IgG1, IgG2, IgG3 and IgG4 (MAH-IgG1-B, MAH-IgG2-B, MAH-IgG3-B, MAH-IgG4-B). The plates were sealed and incubated at 37° C for 60 minutes. (The secondary antibody dilutions used with respect to IgG1, IgG2, IgG3 and IgG4 were 1: 10000, 1:2500, 1:1000, and 1:500 respectively). Then the plates were washed three times and 50 µl of avidin-peroxidase at a dilution of 1/50,000 was placed into all wells except one control series, which received antibody diluting buffer. The plates were sealed and incubated at 37° C for 30 minutes. Then the plates were washed five times with TBS using a 12-channel pipette. Each well received 100 µl of the colour development solution containing 10 mg/ml TMB dissolved in DMSO, diluted 1/100sodium acetate/citric acid buffer pH 6.0 and 0.004% v/v hydrogen peroxide. The incubation was carried out at room temperature (25° C) in the dark. The enzyme reaction was monitored and allowed to proceed so as to produce minimum background colour and a positive absorbance as close to 1.000 as possible. The reaction was stopped by the addition of 40 µl 2 M H_2SO_4 per well to produce a final volume of 140 µl in each well. The absorbance values for each plate were measured using a 450 nm interference filter in a plate reader (Bio-Rad Microplate Reader Model 450), and recorded as optical density (O.D.) readings

Statistical analysis

Non-parametric comparisons using Mann-Whitney U Wilcoxon rank sum test was carried out in the analysis of serum antibody responses in diseased groups and control groups. Correlation between variables was demonstrated using Spearman's correlation coefficient. SPSS for windows[18] was used in the analysis.

Results

Serum total IgG responses to the FKWC

Sera of all subjects in the test group showed evidence of raised total IgG response to FKWC of *P. gingivalis*. Control sera showed a lower level of serum total IgG response compared to the test sera. However, four subjects in the control group showed evidence for relatively higher total IgG response to FKWC of *P.gingivalis* compared to that of the other subjects in the control group. The differences in the total IgG response to the FKWC in disease subjects (Fig. 1) were significantly higher when compared with those of healthy subjects ($p < 0.005$). These findings lead to further assays to study the serum total IgG response and serum IgG subclass profile for RgpA-Kgp complexes in the study group using ELISA.

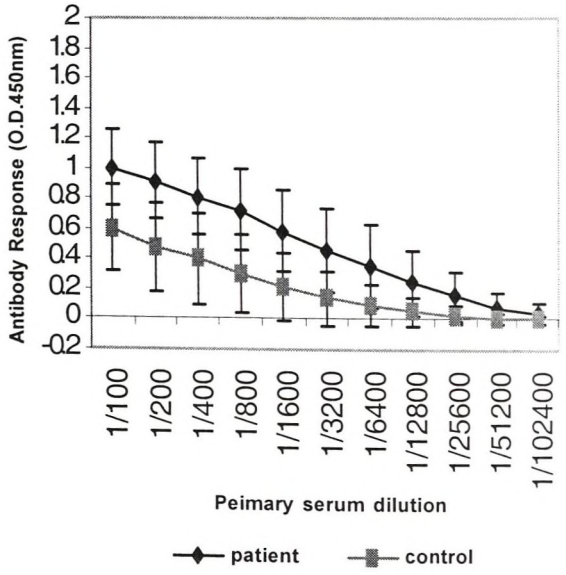


Figure 1. Mean total serum IgG response for FKWC in patients with periodontitis and age and sex matched healthy subbjects

Serum total IgG responses to the RgpA-Kagp complex

All patients and some of the healthy subjects had high antibody titre for FKWC. Therefore, the assays were carried out using the preparation of RgpA-Kgp complexes to detect the total IgG and IgG subclass responses in the sera of subjects in the study group.

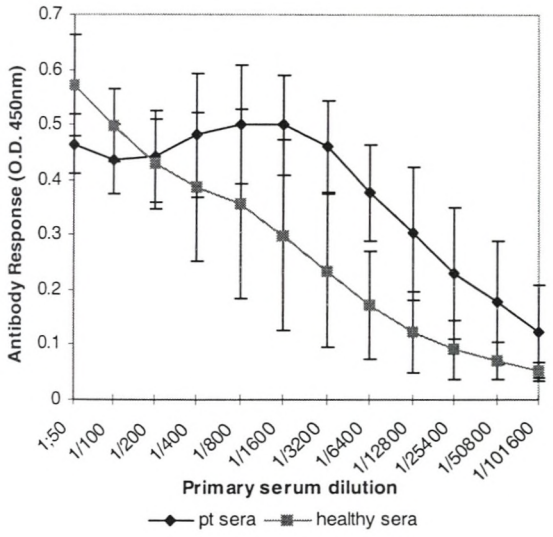


Figure 2. Total serum IgG response for Rgp-Kgp complexes in patients with periodontititis and age and sex matched healthy subjects (Each point represents the mean serum total IgG response for each dilution and standard deviation in each group; OD value at 1/12800 serum dilution was used in data analysis) .

IgG subclass responses to RgpA – Kgp complexes

Serum IgG and IgG3 responses to the Rgp Kgp complexes in diseased and control subjects are shown in figure 3.

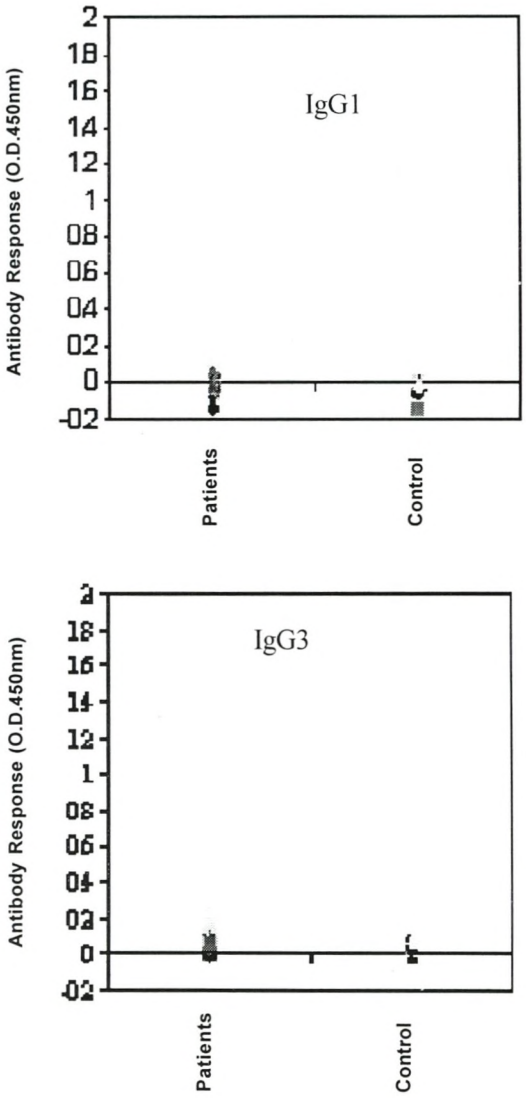
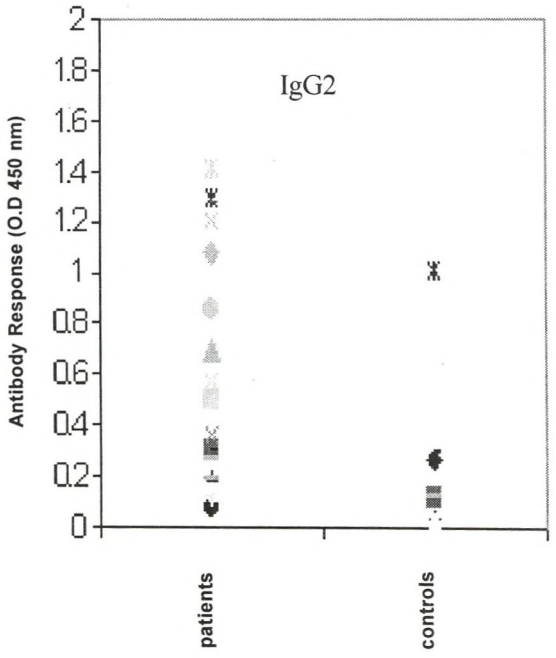


Figure 3. Serum IgG1 and IgG3 binding to RgpA-Kgp Complex in patients with periodontitis and healthy subjects

The serum IgG1 and IgG3 responses in both healthy and diseased subjects were close to background levels (Fig. 3). The statistical analysis using a Mann-Whitney U Wilcoxon rank sum test demonstrated no significant difference between serum IgG1 and IgG3 response to the RgpA–Kgp complex in diseased and control subjects. Therefore, further analysis between these antibody levels and disease parameters were not performed.

Serum IgG2 and IgG4 responses to RgpA–Kgp complexes in diseased and control subjects are presented in figure 4. A Mann-Whitney U Test demonstrated that serum IgG2 and IgG4 responses to RgpA–Kgp complexes in diseased subjects were significantly higher when compared with those of healthy subjects ($p < 0.05$).



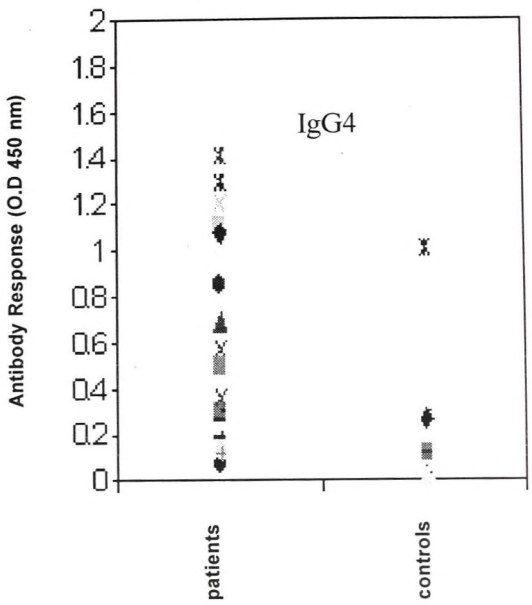


Figure 4. IgG2 and IgG4 binding to RgpA-Kgp complex in patients with periodontitis and age and sex matched healthy subjects

The relationship between serum IgG2 responses against the RgpA-Kgp complex and average probing depth in all subjects was analyzed using Spearman's rank correlation. Spearman's analysis demonstrated that there was a significant ($p<0.01$) positive correlation between IgG2 responses and avarage probing depths (Spearman's correlation coefficient =0.6576)

The relationship between serum IgG2 responses and % sites with bleeding in all subjects was analyzed using Spearman's rank correlation. Spearman's analysis demonstrated that there two variables.

It was also demonstrated according to Spearman's rank corretation that there was a significant positive correlation between IgG4 responses and average probing depth ($p<0.05$) (correlation and coefficient 0.476)

A significant positive correlation was also revealed between IgG4 responses and % bleeding sites also ($p<0.01$) (correlation coefficient 0.493)

Discussion

In this study total IgG responses were determined with respect to FKWC and IgG and IgG subclass responses against RgpA-Kgp proteinase adhesin complexes of *P. gingivalis* W50 were determined in a group of chronic periodontitis patients and age and sex matched subjects with healthy periodontal tissues. In this study it has been demonstrated that there was a significantly higher serum IgG response to FKWC of *P. gingivalis* in periodontitis patients when compared with age and sex matched healthy controls ($p<0.001$). This confirms earlier studies showing similar results.[9,19,20] When the serum total IgG response was determined for RgpA-Kgp complexes the levels were significantly ($p<0.05$) higher in periodontitis patients when compared with that of the age and sex matched healthy subjects.

A similar IgG response pattern to FKWC and RgpA-Kgp complexes in patients and healthy subjects may be attributable to the RgpA-Kgp complexes being the major cell surface antigen on *P. gingivalis*. These findings are in agreement with the results of O' Brien-Simpson *et al.*[10] The very low serum total IgG against FKWC and RgpA-Kgp complexes in the control subjects in the present study may possibly be explained by the intact periodontal tissues in control subjects which may imply that there was little or no exposure to *P. gingivalis* in the past.

Analysis of IgG subclass responses to RgpA-Kgp revealed higher IgG 2 and IgG 4 responses in patients with periodontitis than those of age and sex matched healthy subjects. The relationship between the IgG2 and IgG4 responses in respect to RgpA-Kgp and both the average probing depth and percentage bleeding sites were positively and significantly correlated. A number of reports have also found a dominant IgG4 response to whole cells, cell extracts, purified fimbrial antigens, or RgpA-Kgp complexes from *P. gingivalis*.[10,15,21,22] The dominant IgG4 response in periodontitis patients and the absence of this kind of response in healthy control subjects may reflect the chronic nature of the disease. Chronic infection, where there is persistent antigenic stimulation has been reported to induce a high IgG4 response.[23,24,25]

The other major subclass response to RgpA-Kgp complexes found in this study was that of IgG2. Although IgG2 antibodies are commonly induced by bacterial glycolipids such as LPS,[13] a specific IgG2 response may be induced by the RgpA-Kgp complex as components of the RgpA-Kgp complex have been reported to be glycolipid modified.[26] Further the components of the RgpA-Kgp complex for example, the adhesins, contain repeated peptide sequences.[27] Repeated peptide sequences are known to induce a specific IgG2 response[28] may explain the presence of high IgG2 responses in the present study.

The IgG responses to the RgpA-Kgp complexes of the present study are not consistent with the findings of O'Brien-Simpson *et al*[10] where they found substantial levels of specific IgG antibodies belonging to all four subclasses against RgpA-Kgp complexes in contrast to the high IgG2 and IgG4 and the very low IgG1 and IgG3 levels in the present study. O'Brien-Simpson *et al.*[10] showed that there were significantly higher IgG1, and IgG3 levels against RgpA-Kgp in periodontitis patients than those of the controls. In addition they showed that there were no significant differences in IgG2 and IgG4 levels between periodontitis patients and control subjects. However, in the present study the differences in IgG2 and IgG4 levels between patients and healthy subjects were significantly different. The observed differences may partly be explained by the differences in the composition of the two study samples. In the O'Brien-Simpson's study the control group was defined as subjects with 1- 4 mm pockets and no more than two probing depths of 5 mm and considered to have healthy periodontia, gingivitis or mild periodontitis. Therefore, prior exposure of the control group to *P. gingivails* antigens cannot be excluded as the control group included subjects with gingivitis and mild periodontitis. In contrast, the control group

of the present study had no pockets with more than 3 mm probing depth and was almost free of any clinical sign of gingival inflammation or cumulative effects of periodontitis. Hence there exists the possibility that the majority of the subjects in the control groups of the present study may have had minimal or no prior exposure to periodontal pathogens. The differences in age of subjects in the two study groups may also have accounted for the observed differences in antibody profiles both with respect to total IgG and IgG subclasses in these studies. In the O'Brien-Simpson's study the sample belonged to an older age group (mean age 51.8 ± 9.70 years; age range 36-70 years) than the present study sample (mean age 36.4 years ± 11.3: age range 18-53).

In the present study very low and close to background levels of IgG1 and IgG3 responses to the RgpA-Kgp complexes in the sera of patients and the age and sex matched healthy subjects show that there was a general failure in the study group to mount a substantial level of IgG1 and IgG3 antibody response. Minimal or lack of exposure to periodontal pathogens may explain the low levels of antibodies belonging to all subclasses in control subjects in the present study. Chronic exposure to P.gingivalis over a period of time may be considered as a reasonable explanation to significantly higher levels of IgG2 and IgG4 in the diseased group since *P.gingivalis* is considered as one of the major periodontal pathogens.

It has also been shown that the Fc receptors found on the possible effector cell types (lymphocytes and monocytes) bind IgG1 and IgG3 much better than IgG2 and IgG4.[29] Further, increased efficacy of IgG1 and IgG3 in antibody dependant cell mediated cytotoxicity and complement dependant cell lysis has also been reported.[30] Therefore the failure to mount an adequate level of IgG1 and IgG3 which may play a role in eliminating /limiting the activity of periodontal pathogens may have rendered the subjects in the diseased group in the present study susceptible to periodontitis.

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Research Article 3

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Squamous cell carcinoma invading the mandible

E. A. P. D. Amaratunga

Abstract

Objectives: Mandibular invasion by oral squamous cell carcinoma (Scca) has been recognized as a poor prognostic sign. It is therefore important to identify those tumours with greater potential of bone invasion. The objectives of this study are to find out whether the ability of the Scca to invade the mandibular bone correlates with the histological pattern of tumour growth at the invasive front, degree of osteoclastic activity (density) and the expression of the cell proliferation marker Ki-67.

Material and Methods: Twenty-six hemimandibulectomy specimens resected for primary oral squamous cell carcinomas (Sccas) were classified into 3 groups according to the degree of bone invasion. Six decalcified sections stained with hematoxylin and eosin were prepared from each mandible and used for studying the histological pattern of tumour growth at the invasive front and to determine the density of osteoclasts at the tumour-bone interface. Immunohistochemical staining of Ki-67 was performed on tissues obtained from the tumour to find out the Ki-67 expressivity. The histological pattern of tumour growth, osteoclasts density and Ki-67 expressivity were then compared with the degree of bone invasion.

Results: Results showed that the Sccas with infiltrating growth pattern have a greater ability of bone invasion than those with cohesive pattern of growth at the invasive front. Density of osteoclasts at the tumour bone interface and the Ki-67 exprssivity of the tumour cells have no correlation to the bone invasiveness of the tumour.

Conclusions: This study shows the importance of invasive front grading in histopathology reports.

Key words: squamous cell carcinoma, mandibular bone invasion, osteoclasts, Ki-67

Introduction

Oral squamous cell carcinoma (Scca) is the commonest malignant tumour in Sri Lanka and in other countries of the Indian subcontinent, accounting for about 30-40% of all malignancies.[1] Lingual and buccal sulcuses are two common sites for Scca associated with betel chewing. This is mainly due to the habitual placement of the betel quid in these sulcuses resulting from accumulation of higher concentration of chemical carcinogens released from the betel quid in these areas.[1] The mandibular bone, which lies just beneath the mucoperiosteum of lower gingiva, is often infiltrated by the carcinomas occurring in the lower lingual and buccal sulcuses. Mandibular invasion has been recognized as a poor prognostic sign in cancer of oropharynx and the oral cavity.[2]

Detection of the degree of the mandibular involment and the extent of such invasion are of utmost importance prior to surgery in order to remove all the tumour extensions. However, there is no imaging technique that could show the tumour in the bone with pinpoint accuracy although

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radiographs and MRI scanning are useful.[3] As such, recognition of tumours with greater potential of bone invasion using other parameters would be of significant clinical value. Certain features of invasive mucosal carcinomas such as type, size and grade of the primary carcinoma and the pattern of invasion have been shown to be related to clinical outcome.[4,5,6,7,8,9]

Aims

Aims of this study was to find out whether there is a correlation between the degree of bone invasion by Scca and its histological pattern of growth, its osteoclastic activity at the tumour-bone interface and the expressivity of the cell proliferation marker Ki-67 within tumor cells, so as to determine the predictive values of these parameters with regards to mandibular invasion by Scca.

Material and Methods:

Twenty-six non-irradiated hemimandibulectomy specimens resected for primary oral squamous cell carcinomas were the material used for this study. Specimens were examined carefully and the mucosal lesion (Scca) of each specimen was measured. Only those specimens with mucosal lesions measuring between 1.5-3 cms were taken for the study. Specimens with lesions smaller than 1.5 cm and with lesions larger than 3 cms were excluded in order to prevent undue effects on results by the varying size of the lesions. Examination of specimens also revealed that 18 mandibles were partially dentate and the remaining 8 were totally edentulous.

Each mandible was sectioned into 5 mm blocks in the buccolingual direction with a water-cooled diamond saw, starting from the center of the mucosal lesion. Six such blocks from each mandible were decalcified, sectioned and stained with hematoxylin and eosin.

Immunohistochemical staining for Ki-67 was performed on a further section prepared from the tissues sampled prior to decalcification from the Scca lesion of each mandible.

Specimens were then graded on the basis of the histopathological evidence of the extent of bone invasion by the Scca into three groups namely grade-0, grade-I and grade- II. Grade-0 showed no histological evidence of bone invasion; a clear layer of fibrous connective tissue or periosteum was evident between the spreading front of the tumour and the normal bone surface. Grade- I showed histological evidence of early invasion; only the cortical bone and the marrow spaces immediately beneath the cortical bone had been invaded by tumour. Grade-II exhibited histological evidence of advanced bone invasion causing destruction to deep bony trabeculae and extending close to inferior dental canal.

Hematoxylin and eosin stained sections were used firstly, to reconfirm the previous histopathological diagnosis and then, to assess the histopathological pattern of tumour growth at the invasive front. According to the publication of the Royal College of Pathologists on Standards and Datasets for histopathology reports on Head and Neck Carcinomas,[10] two different histopathological patterns of tumour growth at the invasive front have been described for prognostic purposes. These include: (a) cohesive pattern of tumour growth showing large cohesive tumour islands and strands showing a pushing border at the invasive front with more than 15 cells across each tumour island or strand and (b) infiltrating pattern of tumour growth showing a rather invasive tumour islands arranged in narrow strands, non-cohesive small groups and individual tumour cells. Accordingly, the histopathological pattern of growth was determined for each case.

Haematoxylin and eosin sections were also used to determine the density of osteoclasts in the invasive front. Osteoclasts on the surface of bone trabeculae at the tumour-bone interface were counted manually and the length of the trabeculae was measured using a computerized image analyzing system. The density of osteoclasts was expressed as number of osteoclasts per millimeter length (of bone trabeculae).

Using sections stained immunohistochemically for Ki-67, positively stained cells were counted in

three randomly selected areas of the Scca at the invasive front. One thousand tumour cells were counted in each area and the number of the positively stained cells within the 1000 tumour cells were noted and expressed as number of positive cells per thousand cells (+ve cells/1000). Mean value for each tumour was worked out using the data from the 3 randomly selected areas.

The association of the degree of bone invasion with the histological pattern of tumour growth was tested with Chi-square analysis. The effect of the osteoclastic activity at the invasive front and the Ki-67 expressivity on the degree of bone invasion was explored with one-way analysis of variance (ANOVA). Statistical significance was accepted when the $p<0.05$. The data were analyzed using the statistical package for social sciences (SPSS) for Windows (SPSS Inc., Chicago, IL, USA).

Results

Table1 shows the number of hemimandibulectomy specimens in each group, classified according to the degree of bone invasion based on histopathological evidence. Out of twenty-four hemimandibulectomy specimens, four showed Grade-0 or no bone involvement. Ten specimens had Grade-I bone invasion while the remaining twelve had Grade-II or advance bone invasion.

Table 2, shows the severity of bone invasion against the two histological patterns of tumour growths. All four specimens (100%) with Grade-0 bone invasion and seven specimens (70%) of Grade-I bone invasion showed a cohesive pattern of tumour whereas only four out of eight (30%) of Grade-II bone invasion showed cohesive tumour growth. In contrast 60% of the specimens with Grade-II bone invasion showed invasive pattern of tumour growth.

The association of the degree of bone invasion with the histological pattern of tumour growth was tested with the Chi-square analysis. Analysis revealed Chi-square = 6.47 and the $p<0.039$. As the p value is less than 0.05, association between the degree of bone invasion and histopathological pattern of tumour growth is statistically significant.

Table 3 shows the density of osteoclasts at the tumour bone interface and the Ki-67 expressivity of tumour cells against the degree of bone invasion.

The effect of the osteoclastic activity at the invasive front and the Ki-67 expressivity on the degree of bone invasion was explored with the ANOVA. revealed $F=0.36$, $P>0.05$ for osteoclastic activity and $F=1.82$, $P>0.05$ for the Ki-67 expressivity of the tumour cells. Results show that the association between the degree of bone invasion and osteoclastic activity and the association between the degree of bone invasion and Ki-67 expressivity are statistically not significant.

Discussion

Out of twenty-six test specimens, four specimens showed no bone invasion whereas ten and twelve specimens showed early and advanced bone invasion respectively. This shows the variable ability of different Sccas to invade bone. Of course, when in close proximity to the bone, any malignant neoplasm would eventually invade bone, as the tumour grows larger with time. In the present study, cases were selected with the primary tumour size within 1.5 cm to 3 cm in order to minimize the effects of varying tumour size on results.

It has been shown that the cohesive pattern of tumour growth is less invasive than the infiltrative pattern of tumour growth and the former has a better prognosis, particularly in relation to soft tissue invasion.[5] Present study shows similar results with regards to the invasion of mandibular bone. According to the results, all the samples (100%) showing grade–0 invasion and 7 out of 10 (70%) with grade-1 had cohesive pattern of tumour growth. In contrast, only 33% of the samples showing grade-2 bone invasion had cohesive histological pattern of tumour growth while 66% of the latter showed infiltrative pattern

of tumour growth. Chi-square analysis revealed that these findings are statistically significant. It should be noted however, there were only 4 samples showing grade-0 or no bone invasion. Statistical analysis would have been more powerful if there were more samples in this group. Hemimandibulectomy is carried out when there is clinical and radiological evidence of mandibular invasion or when such invasion is suspected. As such, the number of samples showing no bone invasion were fewer.

The effect of the osteoclastic activity and the Ki-67 expressivity on the degree of bone invasion was found to be not statistically significant. Wong et al (2000) reported similar findings showing no correlation between Ki-67 or Cyclin D1 staining and invasion pattern or clinical outcome.[11] Although no concrete conclusions are possible, it seems that the extent of the deployment of osteoclasts does not necessarily reflect the severity of the bone resorbing process.

Conclusions

It can be concluded that oral squamous cell carcinomas showing infiltrative pattern of growth at the spreading front have a grater ability to infiltrate into the mandibular bone than the tumours showing cohesive pattern of growth. It is important therefore to comment on the pattern of tumour growth at the invasive front in histopathology reports on oral squamous cell carcinomas. There seems to be no correlation between the severity of bone invasion and the density of osteoclasts or Ki-67 expressivity.

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Table 1. Classification of specimens according to the severity of bone involvement.

Grade of invasion		Number
No bone invasion	(Grade-0)	04
Early bone invasion	(Grade-1)	10
Advanced bone invasion	(Grade-2)	12

Table 2. Degree of bone involvement vs cohesive and infiltrative histological patterns of growth at the invasive front

Grade of invasion		Cohesive pattern	Infiltrating pattern
Grade 0	**(04)**	04 (100%)	-
Grade 1	**(10)**	07 (70%)	03 (30%)
Grade 2	**(12)**	04 (33%)	08 (67%)

Table 3. Degree of bone invasion vs osteoclast cell density and Ki67 positivity

Grade of invasion		Osteoclast density (mean)	Ki-67 positivity (mean)
Grade 0	(04)	01.75	53.25
Grade 1	(10)	02	69.5
Grade 2	(12)	02.25	78.25

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Case Report

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Comprehensive management of a patient with localized aggressive periodontitis

W. N. Kularatne and P. S. Rajapakse

Introduction

The term aggressive periodontitis implies a rapid connective tissue attachment loss around the root of a tooth over a short period of time. This condition affecting the periodontal tissues has been described by a variety of names until it was finally termed aggressive periodontitis at the International Workshop for Classification of Periodontal Disease and Conditions.[1]

The majority of patients with aggressive periodontitis are young although there may be a small percentage of individuals over 35 years. Inconsistent tissue destruction with reference to the level of plaque and calculus deposits, rapid rate of attachment loss in systemically healthy subjects (non-contributory medical history) and familial tendency were considered as the diagnostic criteria.[2] Elevated proportions of *Actinobacillus actinomycetemcomitans* and in some far-east populations, *Porphyromonas gingivalis* have also been implicated in the aetiology of aggressive periodontis.

Several clinical trials suggest that the outcome of periodontal therapy in aggressive periodontitis is correlated to the presence or absence of certain microorganisms, notably *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans*, after therapy.[3,4]

However, a couple of other studies have revealed that aggressive periodontitis is associated with *Porphyromonas gingivalis* but not with *Actinobacillus actinomycetemcomitans.*[5]

Reports from long-term surveys of periodontal therapy document the need for surgical intervention, as the root adherent biofilm left due to inadequate instrumentation in non-surgical treatment is not affected by antibiotics.[6] In aggressive periodontitis, comprehensive mechanical/surgical intervention and antimicrobial therapy constitute an appropriate treatment regimen for long-term stabilization of periodontal health.[7] Continuous follow up and initiation of further treatment will ensure a long lasting functional dentition in recurrent cases.

Case report

A 38 year old male patient presented to the Department of Periodontology, Dental Hospital (Teaching), Peradeniya Sri Lanka, complaining of mobility of the following teeth: 31, 32, 41, 42 and gum recession on 23.

The patient was free from any other relevant medical conditions and his general appearance was healthy. History of the patient showed, no unfavorable social habits such as smoking. He

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brushed his teeth twice a day using a toothpaste containing fluoride. He had already undergone periodontal treatments for one year.

Extra oral examination revealed no abnormalities. Oral mucosa was healthy. Gingiva was inflamed in relation to the lower incisor and molar regions. Plaque score (O'Leary's Plaque Index) was 23% (without the use of disclosing agent) and the bleeding score (Sillness and Loe) was 20%. Bleeding on probing was confined to the incisors and molars. More than 4 mm probing depth was observed on 11, 16, 23, 35, 36, 38, 41, 43, 45, 46 and 47 involving 13.5% of the total tooth surfaces.

Gingival recession was seen (Fig. 1) on 23 (6 mm labially), 41 (2 mm labially and 5 mm lingually), 42 (2 mm labially and 3 mm lingually) and 43 (6 mm labially 5 mm lingually). Grade II mobility was seen on 23, 41, 42, 43, 46 and 47. No attached gingiva was found in relation to the labial aspect of 23 and 43. An unsightly composite filling was present on the root of the 23. Grade I furcation involvement was seen on 36, 46 and 47. Occlusal trauma was detected on 41 and 43. Severe bone loss was observed on orthopantomograph (Fig. 2) and periapical radiographs (Fig. 7 and 8) in relation to distal roots of 36, 46 and mesial surface of the root 43. Moderate bone loss was seen on 11, 23, 41, 42 and 45. The tooth 46 gave a non-vital response to electric pulp tester. However due to the absence of any symptoms or periapical lesion and since there was evidence of calcification of the pulp chamber, endodontic treatment was not considered necessary.

Considering all clinical information, the case was diagnosed as Localized Aggressive Periodontitis and the following initial treatment plan was made:

1 Education and motivation of the patient
2. Oral hygiene instruction
3. Full mouth scaling
4. Root planing on 11, 16, 23, 34, 35, 36, 37, 38, 41, 43, 45, 46, 47
5. Re-examination and definitive treatment planning

The patient was treated according to the treatment plan for a period of about 11 months and the case was re-assessed. Root planing was performed on two visits, which was accompanied by systemic antibiotic therapy for 10 days (amoxicillin 250 mg 8 hourly with metronidazole 200 mg 8 hourly). During this period the patient was well motivated and all the appointments were kept regularly.

Post treatment plaque score (disclosed with erythrosine) after six weeks was 10.1 % and bleeding score (bleeding on probing) was 7%. More than 4 mm probing depth was seen on the distal aspect of 36, 46 and the mesial surface of 43. Grade II mobility was found on 43. Grade I mobility was found on 23, 36 and 46. Grade I furcation involvement (Ramfjord and Ash) was found on 36, 46 and 47. Occlusal trauma was found on 43.

The clinical findings after cause related therapy were used to draw the definitive treatment plan (corrective therapy):

1. Reinforcement of oral hygiene
2. Correction of occlusal trauma on 43 by trimming the cusp
3. Bone grafting in relation to distal roots of 36, 46 and mesial aspect of 43
4. Removal of composite filling on 23, root planing and grafting of subepithelial connective tissue from the palate
5. Review and maintenance therapy

The patient was treated according to the above treatment plan for a period of about 6 months.

Bone grafts were done using conventional method. Chlorhexidine 0.2% mouthwash was given prior to the surgery. A mucoperiosteal flap was raised under local anesthesia and curettage of all granulation tissues was undertaken.

Thereafter the involved root surfaces were root planed thoroughly and the area was cleaned with sterile saline. Freeze-Dried Demineralised Bone Allografts [FDDBA] were then packed into the bone defects (Fig. 3). The flap was replaced and sutured. Post-operatively antibiotics [amoxycillin 250 mg 8 hourly, metronidazole 200 mg 8 hourly] were prescribed for one week with a NSAID [Ibuprofen 200 mg 8 hourly] for three days. Chlorhexidine 0.2% mouthwash was also recommended for two weeks. Suture removal was done after 10 days.

During the root coverage, the composite filling was removed on 23 root and root planing was performed. Then a partial thickness flap was raised in relation to 23. A subepithelial connective tissue graft was taken from the left side of the palate in relation to 24, 25 and 26 region (Fig. 4). The free graft was transformed to the root surface of the 23 (Fig. 5) and the flap was replaced. The donor site was sutured and the sutures were removed after one week. Post-operative management was similar to the above.

The patient was reviewed at one-month intervals. Post treatment findings after three months were as follows:

- Plaque score of 9% and Bleeding Index was 4%.
- 43 had only grade I mobility and 23, 36, 46 showed physiological mobility.
- Gingival recession had reduced to 4 mm on 23 (Fig. 6).
- Probing depth had reduced significantly on treated sites.
- Bone development was evident in relation to the roots of 36, 43 and 46 (Fig. 7 and 8).

The patient was satisfied with the treatment and he agreed to come for review appointments monthly.

Discussion

This patient who was 38 years old was diagnosed as having aggressive periodontitis. Aggressive periodontal disease has been detected in all age groups and ethnic groups.[8] As no more than two teeth other than the incisors and molars were affected (and less than 30% tooth surfaces involved), it was more appropriate to define the diagnosis as localized aggressive periodontitis.[1]

Similar to the management of chronic periodontitis, treatment was started with cause related therapy. Root planing was complemented with antimicrobial therapy as combination is considered to be more effective.[9] Mechanical periodontal treatment alone is adequate to ameliorate or resolve the clinical condition in most periodontal cases. However, adjunctive use of anti microbial agents, delivered either locally or systemically, is indicated in specific situations such as aggressive periodontitis, in patients with systemic diseases affecting the host resistance and in the presence of a poor response to conventional mechanical therapy.[9] Metronidazole affects only the obligate anaerobes including *P. gingivalis*, but not *A. actinomycetemcomitans*, a facultative anaerobe. Synergistic effect against *A.actinomycetemcomitans* has been noted with metronidazole and amoxicillin.[10]

Improvement in plaque score, bleeding on probing score and the reduction of mobility in 23, 41, 42, 43, 46 and 47 confirm the effectiveness of cause related therapy. As the condition was stabilized for a long period it was decided to proceed with the corrective therapy.

Angular bony defects in relation to 36, 43 and 46 were treated by bone grafting with Demineralized Freeze-Dried Bone Allografts (DFDBA). Demineralization in DFDBA enhances the osteogenic potential by exposing the bone morphogenic proteins (BMP), which presumably have the ability to induce host cells to differentiate into osteoblasts.[11]

Though some studies have shown the importance of having a minimum width of attached gingiva for the maintenance of gingival health,[12] others support the conclusion that attached gingiva is not required for the maintenance of the integrity of the periodontium.[13,14] However, the gingival recession in the anterior region is frequently treated by root coverage procedures to improve aesthetics.

As gingival recession in the lower anterior region is not critical in aesthetics, root coverage surgery was carried out only on 23. Because of the insufficient width of the adjacent attached gingiva, it was decided to do a free gingival graft. Because of the involvement of the interproximal areas and the width of the defect, only partial root coverage could be achieved as described by Miller.[15]

When the patient was reviewed after 3 months, only grade I mobility was observed on 23 with physiological mobility on other teeth. There was a marked reduction in plaque score and bleeding score. A 3 mm gaining of attached gingiva was seen on 23. Radiographs showed evidence of bone formation in 36, 43, 46 regions. These results indicate the successful management of this patient confirming the effectiveness of comprehensive mechanical/surgical and antimicrobial therapy for long-term stabilization of periodontal health in aggressive periodontitis.[7] However, continuous review will be needed to assess the long-term success.

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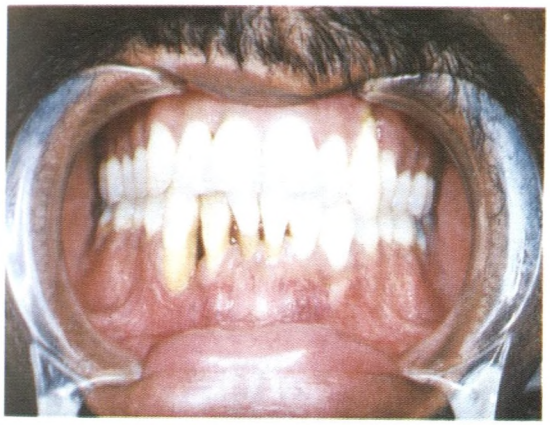


Figure 1. Pre operative appearance.

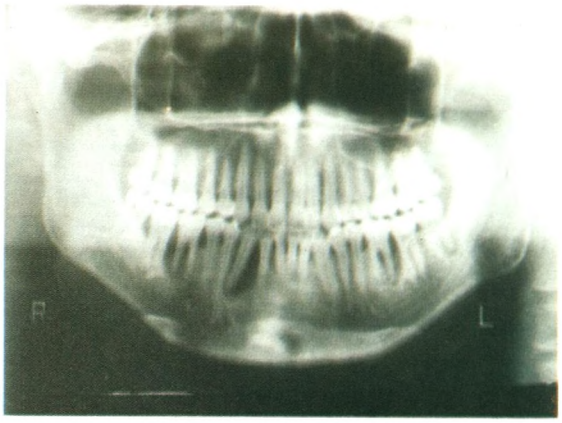


Figure 2. Preoperative othopantomograph

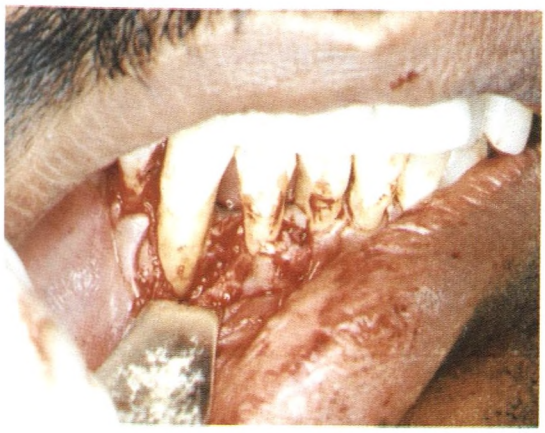


Figure 3. Bone allograft packed into the bone defect in relation to 43.

Figure 4. Harvesting the connective tissue graft

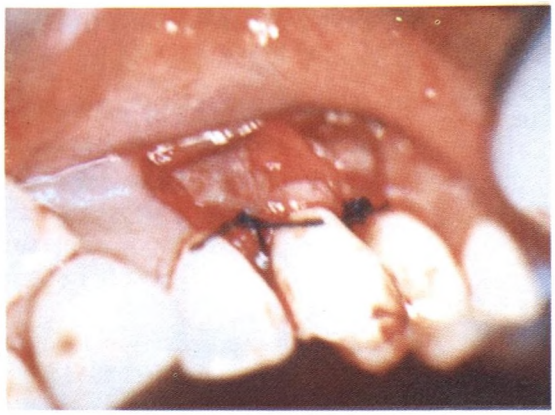


Figure 5. Graft sutured to cover the root of 23.

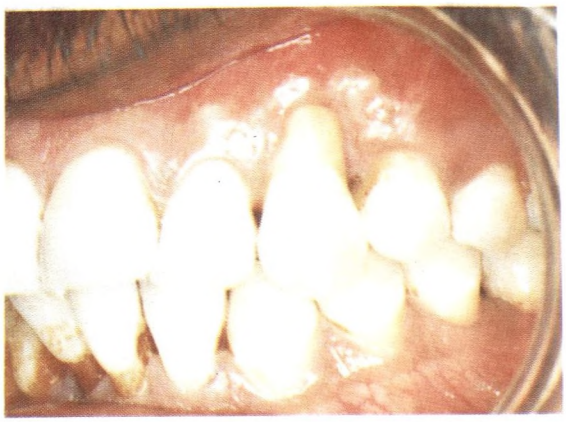
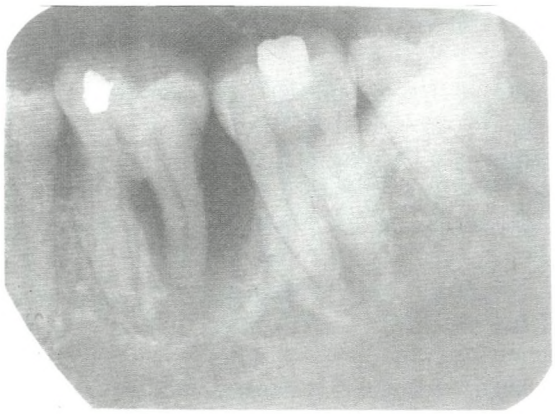


Figure 6. Partial root coverage achieved on 23.



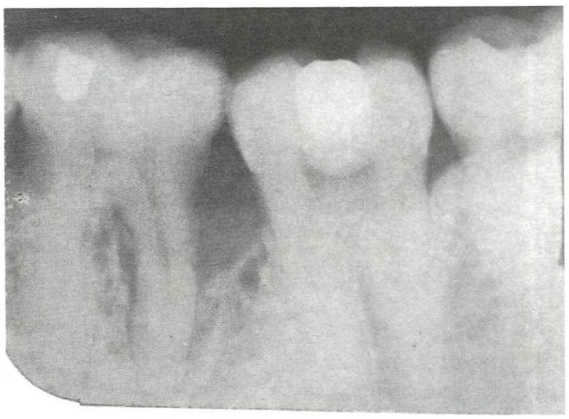
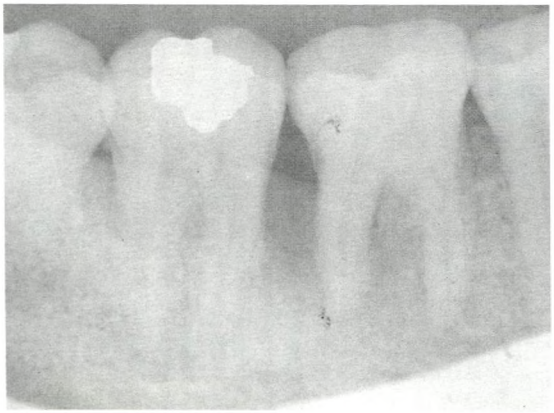


Figure 7. Pre operative and post operative periapical radiographs of 36 region



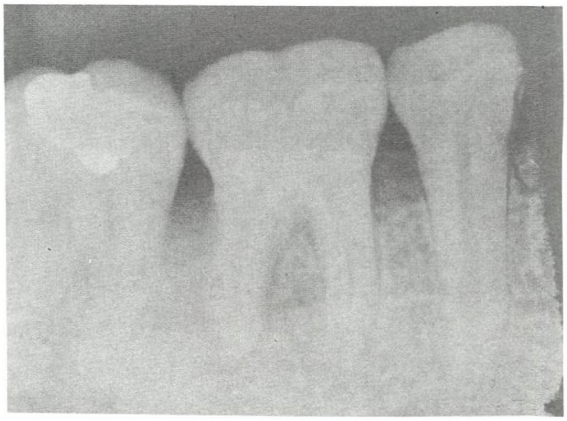


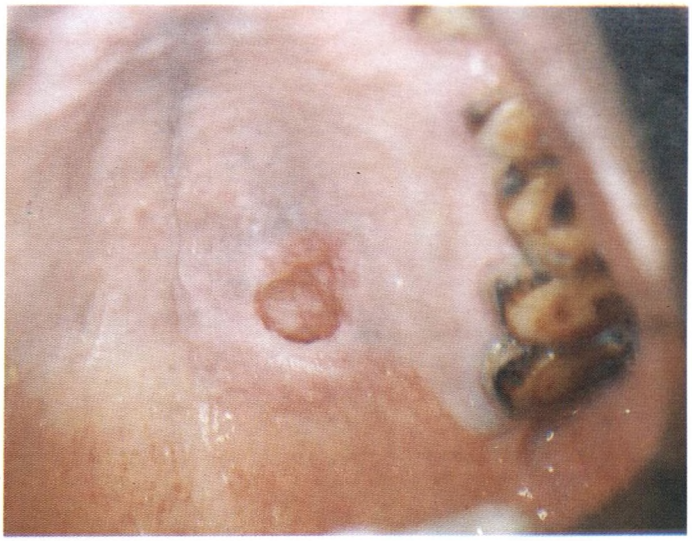
Figure 8. Pre operative and post operative periapical radiographs of 46 region

Sri Lanka Dental Journal 2006; 36(01) 52

Self Assessment / Oral Diagnosis (SAOD)

We hope you enjoy the SAOD section

A 55-year-old man complains of one-week-old ulcer on the hard plate.



1. Which of the following features in the history are important in arriving at the differential diagnosis?
   a. history of smoking habit
   b. history of trauma
   c. history of diabetes mellitus
   d. recent use of drugs
   e. positive family history

2. Which of the following are important differential diagnoses?
   a. erythroplakia
   b. leukokeratosis necotina palati
   c. necrotizing sialometaplasia
   d. pemphigus vulgaris
   e. squamous cell carcinoma

3. What investigation/s are vital to confirm the diagnosis?
   a. incisional biopsy
   b. full blood count
   c. random blood sugar
   d. swab and culture for candida
   e. ESR

Answers

1. a,b
2. a,c,e
3. a

Prepared by: **Dr. A. Ariyawardana B.D.S(SL), MS(Col).**

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The gum protection formula

Sri Lanka Dental Journal 2006; 36(01) 00-00

Appreciation

Dr. M.M.Mukthar

The sudden demise of Dr. M. M. Mukthar on the 20th of December 2005 was a great loss to the Dental profession and the other organizations which he served with dedication. I have personally lost a dear friend and colleague with whom I closely associated for over 35 years.

Dr. Mukthar was an old boy of Zahira College, Colombo where he was a senior prefect and was a brilliant student winning the prestigious special senior prizes for Physics, Botany and English Essay. He had the unique distinction of captaining his house under 16 and under 19 Cricket teams at the same time.

He entered the Medical College, Colombo in 1955 and was elected the first year representative of the Medical students Union and treasurer of the Anatomical Society. After the first year he changed over to Dentistry. During the three years at Peradeniya Dental School he was the treasurer, secretary and at the final year was the President of the Dental Student's Association. Under his presidency the first ever Dental Seminar and the first Dental Journal, "Mirror and Probe" was published long before the Dental profession held seminars and published journals.

After passing out as a Dental Surgeon, he served at the Dental Institute, Colombo for one year. He then started full time private practice and eventually built up a lucrative practice.

He has been an active member of the Sri Lanka Dental Association and rendered unstinted service, having been in the council as the secretary, a record period as the treasurer and finally as the president in 1985.

He was elected treasurer of the General Dental Practitioners Association in its second year and had been its treasurer for a continuous period of 27 years. He was the founder treasurer of the College of General Dental Practitioners and functioned as its treasurer for many years and was the President for two years. He was chiefly instrumental in getting an office for the GPDA at the OPA premises to celebrate it's 25th anniversary. This was indeed a monumental achievement.

As a Council member representing Sri Lanka at the International Association of Dentistry for the handicapped, he attended it's congress in 1986 in Norway and 1988 in Philadelphia and continued to be the only Sri Lankan member of the I.A.D.H. until his demise.

Instructions to Authors

The Sri Lanka Dental Journal publishes the following categories of articles which have relevance to Dentistry and allied sciences.

1. Leading articles - One article per issue. It may be solicited by the Editor. Authors are welcome to submit leading articles on current topics of interest. One's expertise or commentaries on general practice etc. They should be approximately 1500 words in length. References should be 20 or less.

2.Reviews - Reviews are detailed surveys of published research pertinent to dentistry and associated sciences. They should be critical in nature and should not normally exceed 3000 words and 30 references.

3. Research articles - Articles resulting from research work belong to this group. Results from routine clinical examinations or laboratory investigations will not be considered under this category. Subjects may vary from clinical trials to basic science research, historical analysis to dental economics. They should not exceed 3000 words and 30 references. A reasonable number of tables and illustrations will be accepted.

4. Short reports - These include reports on current topics, modified techniques. new materials, practice management etc. Interesting results from routine, clinical work or laboratory il1vestigations also may be accepted.

5. Case reports - Reports such as of rare diseases or conditions. Modifications to accepted treatment procedures, new management methods etc. may be included in this category.

6. Letters to Editors - Subjects unlimited, but may include short critique of published papers in the SLDJ.

7. Miscellaneous topics - Subjects unlimited and the format are free. These may also include details of scientific meetings, conferences, annual sessions, examinations, news and views, visits and obituaries.

8. Proceedings of annual sessions - Abstracts from annualsessions of the SLDA and other colleges will be published under this category.

Submission of manuscripts

Authors submitting a paper do so on the Understanding that no part has been published before. that it is not being considered for publication elsewhere and that it has been read and approved by all the authors.

Manuscripts including Tables and Figures should be sent in triplicate as the work will be reviewed by two or more referees. While papers are subject to editing. The journal does not hold it responsible for statements made by the contributor. The author alone is responsible for the statements made in his paper.

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Manuscript on disk

Once an article has been accepted for publication. the author will be asked to supply a copy of the final manuscript on disk together with two copies of the complete manuscript. Every disk must be clearly labelled with the name of the author, title, software and program version number.

Manuscript style

The manuscripts should be typed, double-spaced: on A4 (2 I 2x297 mm) paper and submitted in correct English: both English and American spelling are acceptable, provided this is consistent throughout the manuscript.. Manuscripts not submitted in proper format or in poor English may be returned without review. The format of a manuscript should be as

follows: Title page. Abstract. Introduction, Material and methods. Results. Discussion. Acknowledgements. References. Figure legends. and Tables, arranged in that order.

He served the community at large having been the Secretary, President and Council member of the Lions Club of Colombo (Host). He was actively involved in social service for over 31 years. He was a Lion of selfless dedication.

Dr. Mukthar was a forum and Exco member and vice president and a life member of the Organization of Professional Associations (OPA). He was the founder treasurer of the benevolent Society of the OPA.

His wife, Nafeela is a retired English graduate teacher having taught at D.S. Senanayake College, Colombo. She has also lectured at the Open University, Aquinas College and the University GELT program. His eldest daughter, Mina is a doctor who is married to a Gastroenterologist and both are working at Apollo Hospital. His Son, Miraz is a Dental Surgeon who is in charge of his father's practice. His youngest daughter, Nyaza is an Attorney at Law with a LLB degree.

I have always believed in honoring a person while he is alive than when he is no more. In this respect, I am pleased that on a proposal made by me to the council of the College of General Dental Practitioners he was felicitated at its annual sessions a few years ago. He was presented a memento and a citation was delivered by Dr. Hillary Cooray.

When I last visited him, he told me, "Wimal my eyesight is failing, but I have no regrets. I have played my innings. All my children are professionals. My son is there to carry my name and profession. What more do I want".

We had many common interests including cricket. We were very good friends and I will miss our occasional chats on the phone.

Dr. Mukthar will be dearly missed by all his fiends and colleagues.

On behalf of the SLDA we express our sincere sympathies and deepest condolences to Mrs. Mukthar and family and in particular to his son Dr. Miraz Mukthar, our Colleague.

Dr. W.G.Wimaladharma.

Title page - The title page should contain the following information in the order given: 1) a concise but informative title; 2) author's full names' (without degrees and titles): 3) author's institutional affiliations: 4) a running title. not exceeding 40 letters and spaces; 5) name, address, telephone, telefax and electronic mail address of the author responsible for correspondence.

Abstract page - Original and review articles must contain an abstract of approximately 250 words with four specified subtitles:

I) Objective: An introductory sentence indicating the objective and purpose of the study.

2) Material and methods: A description of experimental procedure including applicable statistical evaluation.

3) **Results**: A summary of the new. Previous unpublished data and results.

4) Conclusion: A statement of the study's conclusion 3-5 key words according to Index Medicus should be provided.

Introduction - The introduction should carry sufficient background information on the subject of study.

Material and methods - Procedures should be described in such detail as to make it possible to repeat the work. Subheadings may be used to improve clearness. Correct unit abbreviations should be used (e.g.; "h", "min", "s" and "Fm" rather than "hr", "minutes", "sec" and "Fl". respectively).

The authors should consider the ethical aspects of their research and ensure that the work has been approved by an appropriate Ethical Committee. Where applicable, a copy of the ethical clearance certificate should be attached. In human experimentation. informed consent from individuals should be Obtained and this should preferably be stated.

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Tables - The tables should be numbered in the order of appearance in Arabic numerals, Each table should have a brief explanatory title. Each table; should be typed on a separate sheet, with due regard to the proportion of the printed column/page.

Figures - All graphs, drawings, and photographs are considered figures and should be numbered in the order of appearance in Arabic numerals. Each figure should have a brief and specific legend, and all legends should be typed together on a separate sheet of paper. Photographs should be glossy prints and the reverse should give the figure number, title of paper principal author's name and have a mark indicating the top. Colour illustrations may be submitted in instances where their use may contribute significantly to the scientific value of the article. Colour illustrations may be printed free of charge at the Editor's discretion, whereas others may be printed at the author's expense.

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Congress abstracts should not be used as references nor may "unpublished observations" and "personal communications" be placed in the reference list. References cited as "in press" must have been accepted for publication and not merely in preparation or submitted for publication

Examples of correct. forms of references are given below. These are based on the format used in the *Index Medicus.* Abbreviate journal names according to the *List of Journals Indexed,* printed annually in the January issue of *Index Medicus.* List all authors; do not use *et al.* in the reference list.

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Unpublished article

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Books and other monographs

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