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Editorial Office

Office of the SLDA,
Professional Centre,
275/75, Bauddhaloka Mawatha,
Colombo - 7.

Correspondence regarding editorial matters, articles, reviews and news items should be addressed to the Editor, SLDJ, Dr. Upul B. Dissanayake, Dept. of Oral Pathology , Faculty of Dental Sciences, University of Peradeniya, Sri Lanka.
Tele: 0812385821/0812387500/0812392068
0777393318

Correspondence regarding advertisements and financial matters should be addressed to Dr. Malcolm Stanislaus , 50, Hekitta Cross Road, Hendala, Wattala.
Tele: 011 - 2930368

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EDITORIAL

Sydney William Garne and his contribution:

The leading article of this journal has been written on Sydney William Garne, the founder President of the then Ceylon Dental Association. He is one of the few British qualified dentists who migrated and started to practice dentistry in Sri Lanka. He has made a priceless contribution and a yeoman service to the dental profession of Sri Lanka in the mid part of 19th century. It is the duty and the responsibility of the professionals who currently give the leadership to the dental profession to make aware of our young members, so that the memories of this unique character of Sydney William Garne will be passed down to the future generations.

Sydney William Garne's migration to Sri Lanka took place in 1909. With the indomitable courage and continuous agitation by him and his colleagues the Dental profession was legally established in Sri Lanka by passing the Dental Registration Ordinance by the State Council in 1915. Garne was the first dentist to register under the Dental Registration Ordinance without a medical qualification and this was on 16th of June 1916. He established the Ceylon Dental Association on 6th of December 1932. The Ceylon Dental Association from its inception started lobbying the government to commence the formal dental education in Sri Lanka. As a consequence of this continuous pressure, a dental school was started as a unit affiliated to the Ceylon Medical College of the University of Ceylon in 1943.

Stanly Gelbier, is the person who wrote about the life of Sydney William Garne. Stanly is an

honorary senior research associate at the Welcome Centre for history of medicine at ULC and editor of Dental Historian. While dedicating this editorial of the Sri Lanka Dental Journal as a tribute to Sydney William Garne, as the editor of the Journal I would like to express my sincere thanks to Stanly Gelbier who enlightened the Sri Lankan Dental Profession on Sydney William Garne and his valuable contribution to the profession.

Upul B. Dissanayake

Leading Article

Sri Lanka Dental Journal 2011; 41(01-02) 03-08

Sydney William Garne, LDSRCS, FRGS (1875 - 1946), Founding President of the Ceylon Dental Association

Stanley Gelbier

Abstract

This paper follows the life of Sydney William Garne: from qualifying as a Dentist in London, through a short visit to South Africa, to a lifetime of professional service in Ceylon. There, he was the first non-medically qualified dentist to enrol on the Dentists Register. He then became the Founder-President of the Ceylon Dental Association which he ensured was based on the British Association. The responsibilities of that post remained on his shoulders for ten years. All the time, he ran a thriving practice and had a happy family life, including a stepson of whom he was proud.

Introduction

I (the author) was encouraged to think about Sydney William Garne by Henry Carr Green, LDS Manch, DDPH, a much later member of the same family and a retired UK General Practitioner and Community Dentist. He had gathered some information about his relatives, especially on Sydney Garne and his adopted son Spencer. Although qualified in England, Sydney was immensely important in the world of Ceylonese dentistry. He asked me to take forward this research project.

When Green's elderly aunt died in 1985 he was the executor of her will. Amongst her papers Green found a notebook written around 1870 by his great-great, grandfather William Louis Garne for his son Henry. It helped him to trace back a number of generations of his family. Green had retired in July 1984. After dealing with his aunt's affairs, he decided to investigate the family's history. As a fellow dentist he developed a special interest in Sydney William Garne.

Amongst Green's contacts was Richard (Dick) Garne who had been researching on the family since World War II. He told Green that he had been sent a cutting from a 1902 South African newspaper publication with information on Sydney.

Sydney William Garne

Sydney William Garne was born on 24th December 1875 in 'Berkshire, Surrey'. His parents were Henry Frederick Garne (birth: 1849 in Newbury; death: 1898) and Caroline S R Bowker (birth: 1846 in Stoneleigh, Egham). They married in Windsor in 1874. She was previously married to a Mr. Goodman by whom she had a daughter named Kate (who married Sydney James of Egham). In addition to this half-sister, Sydney had a sister, who died in infancy.

Sydney's grandfather was Henry Jenner Garne (1811 – 1877), who in 1842 married Helen Smith of Newbury, Berks. Henry was a younger brother of Henry Green's great, great, great, grandfather, William Louis Garne, author of the diary.

Prof. S. Gelbier (Correspondence) FFPH, MA, PhD, FDSRCS, DHMSA, DDPH Wellcome Trust Centre for the History of Medicine at UCL, 183 Euston Road, London, NW1 2BE; E-mail: s.gelbier@ucl.ac.lk

Looking for a reason as to why Sydney chose dentistry as a career, Green wondered if his father was a dentist, or perhaps a physician or surgeon. However, there was no record of H F Garne in relevant medical or dental directories or registers or the Royal College of Surgeons of England's lists of members. That is not surprising. In fact, Frederick was the proprietor of a 'fancy repository' in Egham in 1882 and 1890. In 1895 he is listed as a private resident in Runneymede Street, Egham. He was no longer noted in the 1899 local Directory but is on the Electors list for 1899 as owner of 3 and 4 Tennyson Villas, Denham Road, Egham. Henry Frederick Garne died in 1898.

Education and training

Sydney William Garne was educated at Kent College, Canterbury and then the Coopers' Company School in Egham. The latter was possibly the Strode's School which was associated with the Worshipful Company of Coopers, a City of London livery company. Between January and December 1891 he attended the King's College School in London's Strand. At the time Sydney went there it occupied the same building as King's London, for which it was largely a feeder school. Students of the school and college mixed together a great deal. In 1897 the school moved to Wimbledon. By then Sydney had left.

Having made up his mind to become a dentist, Sydney Garne obtained a formal education and training, rather than undergo an apprenticeship as was common at that time. The licence in Dental Surgery of the Royal College of Surgeons of England had been introduced in 1860 and the Dentists Register established in 1878, so the dental trade was becoming professionalized. Garne gained a place to study at the Dental Hospital of London which had been founded in 1858 at 32 Soho Square. In the following year, the London School of Dental Surgery was established in the same building to prepare students for the LDS, according to a prospectus issued in August 1858.

' The Dental Hospital of London is founded for the purpose of affording to the poor, generally the means of obtaining gratuitous relief and advice, in such cases as are included in the special practice of Dental Surgery, and also for affording an opportunity of instruction to those who enter the Dental Profession.'

By 1874 both institutes moved to 40 Leicester Square, in 'a disreputable area of London', a ' notorious hunting ground of the debased and profligate'. In 1901 the hospital and school moved to number 32.[i] King Edward VII agreed to become patron, bestowing the title of ' Royal' Dental Hospital on 15th October. Garne would have studied at the second building. He also attended Charing Cross Hospital for medical aspects of the course necessary as the dental hospital was not part of a general hospital. In 1898 Garne passed the examinations for the LDSRCS. His son Spencer said Sydney wanted to study medicine and had possibly started the course when his own father died in that same year. So it didn't happen.

Practice in the United Kingdom

At some stage he was a house officer at Charing Cross Hospital. Evethough he qualified in 1898, Garne did not register his name on the list of dentists held by the Dental Board of the United Kingdom, a sub-committee of the General Medical Council, until 15th May 1901. Before 1921, when dentistry became a closed profession, it was not essential for practitioners to register with the Board. Thus he could have worked without doing so, but it probably would have been unusual if not rare, for qualified dentists not to register. So either he was not working or had gone abroad.

The 1902, 1903, 1904 and 1905 Dentists Registers show Garne was registered at 15 Clarence Street in Staines, but it does not necessarily mean he was working there. The 1901 Census indicates he was living in Staines in that year. The dental list in the Calendar of the Royal College of

Surgeons of England shows that in 1904 Garne was living in Johannesburg, Transvaal. He had emigrated to Krugersdorp in South Africa and was certainly still there in 1905. The 1905 and 1906 calendars suggest Garne did not return the form in those years to update his personal information, so he was dropped in 1907 and 1908. He did not remain in Africa. By 1908 Garne was listed in the Dentists Register at ' Clovelly' in Egham, Surrey, but again, that does not confirm he had returned to England. By then he may have travelled to Ceylon.

The 1909 College Calendar lists Garne as at Clovelly and at the Bristol Hotel in Colombo, Ceylon. Before air travel, a journey by sea from Britain to Ceylon would have taken about a month, so he was unlikely to be practising in both countries. As often happened with ex-pats he no doubt used Clovelly as a UK point of contact.

Garne remained on the British register until he died in 1946. Until 1923 he was registered at Clovelly, from 1924 at the Grand Oriental Hotel, Colombo, and from 1933 at 45 Galle Face Court, also in Colombia.

According to his son, Sydney Garne was the first Dental Lecturer to ' the Royal Naval Dental Corps at Portsmouth'. It has not been possible to trace any association. The Royal Naval Dental Service was not formed until April 1920. However, Harry Green learned that Naval Medical Officers were appointed to Haslar Hospital and the RN Barracks at Devonport in 1892 for ' dental duties.' He wondered if they couldn't cope and called in specialist help to lecture the naval officers about teeth.

South African interlude

Sydney decided to go to South Africa. According to a South African publication, Garne migrated in 1893, but it is probably a printing error. His move came after 'practising successfully at Queen's Gate, London, and Staines, Middlesex', so he could not have gone unless he practised dentistry before taking the LDS examination in 1901. The article suggests he chose Krugersdorp as it offered 'considerable possibilities for the exercise of his science', and the fact he 'remained there proves he has succeeded to his and his clients' satisfaction'. The publication said Garne was a foremost supporter of sport in the district, was a member of the West land and the Krugersdorp Clubs, and of the Pony and Galloway Club.

Spencer also said his father was a dental examiner in South Africa, but again there is no confirmatory evidence. Garne did not stay there. He returned to the UK for a while, then emigrated to Ceylon.

The marriage of Sydney William Garne

In 1910 Sydney married Agnes Bessie (known as Betty) Clapp. She was born on 7th June 1878 as Bessie Gould to a family living in Shanghai, China, where she married Harry Clapp. A son, Spencer, was born in Japan on 16th October 1899. The marriage broke down and Betty went to San Francisco, USA for a divorce. She then travelled to Singapore and married Sydney. It is not clear where they met. Sydney legally adopted her son Spencer but there were no children born to them both.

Practice in Ceylon

The British arrived on the island of Ceylon in the late 18th and early 19th centuries. They introduced extensive crops of tea and coffee. By 1909 Garne had moved there, after a sea voyage of three to four weeks. He settled in Colombo in the South West of the country. Garne initially practised at the Bristol Hotel in Colombo, which was occupied by a succession of dentists until the late 1970s. Ceylon became Sri Lanka in 1974.

From 1924 he was listed in the Dentists Registers at another address in Colombo; the Grand Oriental Hotel (known locally as GOH). Although later renamed as the Hotel Taprobane, by 1994 it reverted to the old name.

Garne's long-standing practice was at 45 Galle Face Court, which he was in the process of selling at the time of death. Although, it was not his first home, for many years Garne lived and worked at apartment number 45. This interesting building with its shallow dome stands near the Galle Face Hotel, at the far end of Galle Face Green in Colombo. It was built in two phases: 1923, first section built as Galle Face Court 1, the first multi-storied block of flats in Ceylon; 1926, a large domed addition, Court 2, with an observatory in the dome for the residents and their guests. The flats were initially let out to Europeans. It later also housed the HQ of Macan Markar, the family of jewellers who built it.
Garne changed his address for registration with the GDC in 1923 and remained at Galle Face Court until his death.

This address saw a succession of dental practitioners. Following Garne came William Hugh Burndred. He was associated with Dr. Horace Norman Barnes, both of whom were attached to the British navy in the war. They were admitted as members of the dental association on 14th December 1946. Both left Ceylon in December 1950. Next came Dr. G.P.D. Rajasooriya (who died in harness) and then Dr. Pakstun for a very brief period. The practice probably closed in the mid – 1970s.

Dentistry in Ceylon and origins of the dental association

From 1915 the practice of dentistry was governed by the Dental Registration Ordinance. The first qualified dentist to register and work in Ceylon was Sperling Christoffelsz, LRCP,MRCS Eng, LDSEdin. Garne was the first dentist to register without a medical qualification, entering the dental list on 16th June 1916. Strangely, it suggested his LDS was from Edinburgh rather than the English College of Surgeons.

According to Hilarian (Hilary) Cooray, a Past President of the Sri Lankan Dental Association and a noted historian of dentistry in that country. 'The name of William Garne is of great importance in the history of dentistry in this country [ie in Ceylon, later Sri Lanka]'. On 6th December, 1932 Garne and eleven colleagues formed the Ceylon Dental Association, with him as Founder President. By then 25 dentists worked in Government Hospitals and the Private Sector. According to a foreword written by Garne to the Constitution of the association, the original rules and regulations were based on those of the British Dental Association. The BDA was thanked by Garne and Gomes, the honorary Secretary, for much encouragement and support and for 'readily accepting our application for affiliation to the Parent Institution'.

Garne remained President of the CDA for 10 years, after which he retired because of ill health. In wartime, the activities of the association were disrupted and election of its office bearers was delayed in 1942. On 12 November Garne said that with much regret, owing to ill health he was not willing to be re-elected President, ' which office he filled just 10 years from the day of the founding of the Association'. Because of his ill health, a few meetings were held at his home. Garne was replaced by J S R Goonewardena (1942-45).

In 1943 a Dental School was established as the Department of Dental Surgery in the Faculty of Medicine of the University Ceylon, Colombo. As an acknowledgement of his outstanding contribution to dentistry in Ceylon Dr. W. Balendra proposed that an enlarged portrait of Garne should be hung at the meeting place of the CDA. A date was fixed for this on the 15th anniversary of the founding of the association. The CDA sought permission from the Director of Medical and Sanitary Services, Dr. Briercliffe, to hang Garne's portrait on the walls of the school. Permission was not granted as it was against policy to hang private portraits in Government buildings and Garne was not a Government employee. On 20th July 1946 the issue was taken up with the new Director of the

School, Balendra himself. He agreed to allow portraits of Garne and Gomes to be mounted in the hall where the association held its meetings.

The Royal Geographical Society

Garne was elected as a Fellow of the Royal Geographic Society on 15th November 1909. On the form he described himself as a Medical Dental Officer of Colombo, so he certainly was in Ceylon at that time. His proposers were Sir Ernest E Shackleton and John Scot Keltie (Secretary of the Society). He had met the famous explorer when Shackleton visited Ceylon en route to one of his expeditions. On 9th April 1940 Garne wrote to the society about the expense of the annual subscriptions: "I trust you did not take any exception to my letter regarding the fees payable by foreign fellows, but of course, I know you appreciate the difficulties we are all going through at present." He ceased membership in the following December.

Death of Garne

Sydney William Garne died aged 72 years at the Joseph Frazer Nursing Home, on 25th October 1946. The funeral was that same evening at the General Cemetery in Kanatte.

The Rev. Proctor held a short service in the chapel and at the graveside. The Times of Ceylon wrote, "Dr. Garne was one of the oldest and best known dental surgeons in the Island." On 4th December the dental association passed a motion of condolence on his death.

Sydney William Garne's Will

Garne appointed his wife, Agnes Bessie (Betty), and a friend, Arthur Nesbitt Strong MA, Barrister-at-Law as Executors for his Will. In it he asked Betty to give some friends a few personal belongings. The rest of the estate was left to her. In the event of her death before him or at the same time, the estate was to be divided among.

The Will signed in Colombo on 25th October 1944 left nothing to Spencer: perhaps Sydney felt he had enough of his own money. At some time Sydney had given Spencer a ring which he possibly felt was enough as a memento. Spencer said earlier that Sydney had a ring of which he was very proud. It had a crest with a French motif – possibly an 'N' – which Spencer thought was a family crest. May be this was the ring given to Spencer.

Garne's Will lists the companies in which he held shares. On 26th November 1946 an agreement was signed between Betty and William Hugh Burndred, a dental surgeon, who was in the process of purchasing the practice. Garne had agreed before death to sell the practice ("exclusive of the safe and the contents thereof") for £ 3,000, 1,500 of which had already been paid. The sale was completed on 11th November 1946, with the balance to be paid in 20 months. Everything including goodwill was to be included. Letters were to be sent to the patients that the practice had been sold to Burndred.

After Sydney's death Betty and her two sisters settled in a house in Oxfordshire. She made contact with her first husband, Harry Clapp, and visited him in the Norfolk Islands, perhaps en route to Australia to see Spencer. Her brother John had lived out his years in Canada. Betty died on 4th October 1966 in Sunningdale.

Postscript: Spencer Harry John Garne

Spencer Garne was born in Kobe, Japan on 16th October 1899, to Bessie (Betty) Clapp.[xli] Spencer spent his early years in Singapore but his main education was "Berkhamsted [sic], Hertfordshire'. He was adopted by Sydney when the latter married his mother.

In World War I Spencer joined the Royal Flying Corps, on 5th December 1917. At first he was an RFC cadet at Blacksown camp, then he trained as an observer in Hastings, Reading, Uxbridge, Hythe, New Romsey and Winchester. Spencer was then posted to France. His movements were: 21st July 1918, No. 8 HQ Squadron; 8 August,

with tank brigade on the Somme; and 21st August, Arras front. Spencer was wounded on 23rd August 1918 and demobbed on 23rd June 1919.

April 1936 saw the marriage of Spencer to Marion Haynes Padbury (Born 26th May 1903) in Colombo. In World War II Spencer served in the Royal Air Force. He joined up in Ceylon and held a staff appointment in Colombo throughout the war, perhaps engaged in some cipher work. His rank is unknown.

After the war Spencer became a tea planter on the very large and well known Gikiyanakande Estate in Neboda, Western Ceylon. He still retained an English address: c/o Mrs. Atkinson, High Kelton, Berkhamsted, Hertfordshire. It is unclear who owned the estate when Spencer worked there. However the Sunday Times reported in 1988 the former Finance Minister, Ronnie de Mel, has said he owned 50 acres of land to which he was entitled under the Land Reform Law. Under this law his family surrendered 5,000 acres of the highest yielding tea and rubber lands in the country and also some coconut and paddy lands. They included the Gikiyanakande estate. Under this law he applied to keep 50 acres for which he was eligible from his own Glendon estate at Neboda. However, in 1975 the Commission allocated 50 acres from Puttalam Plantations which also belonged to him.

Upon leaving Ceylon in 1959 Spencer and Marion spent some time in the UK then emigrated to Western Australia. Their last address was 86 Tyrell Street, Nedlands in Perth. Spencer and his wife both died in Perth in 1980; Marion (Born on 26th May 1903) on 13th March, Spencer on 14th September.

His Will dated 16th August 1974 named Marion as the main beneficiary, but she pre-deceased him. Having no children his estate of 50,000 Australian dollars was left o Marion's niece, Helene Elizabeth Niquet Wilson, of Sevenoaks, Kent. She was Marion's niece and had also lived in Ceylon for a number of years.

Acknowledgements

Grateful thanks are due to Harry Greene for setting me on this journey and allowing access to his papers. Also those of his relatives who clarified points of information.

Helen Nield, Library Manager and Damyanti Raghvani, Librarian, at the BDA Information Centre gave an enormous amount of help by locating information. Also librarians at the Royal College of Surgeons of England, Welcome Collection and local archives mentioned in the text.

The Royal Geographical Society (with IGS) kindly gave access to Sydney Garne's original application form and some correspondence.

Support, encouragement and information came from Hilary Cooray, the eminent Sri Lankan dental historian.

The original members of the Ceylon Dental Association, 6th December 1932

Sydney Willam Garne	(President)
H Eric Swan	(Vice President)
A Annesley Gomes	(Honorary Secretary)
M A B Brito Muttunayagam	(Treasurer)
J S R Goonawardena	
Sperling Christoffelsz	
S L Cramer	
W Balendra	
V Sinnatamby	
C A R Goowardena	
E P N Abeysundara	
A E Daviot	

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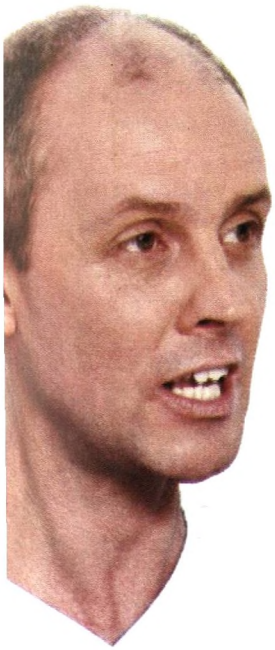

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1. Addy M. Int Dental J 2002; **52** : 367-75

Research Article I

Sri Lanka Dental Journal 2011; 41(01-02) 09-16

Properties of carbonated beverages sold in Sri Lanka: implications for tooth erosion

Nilantha Ratnayake, Lilani Ekanayake and Berty Gangadhara

Abstract:

Objective: To analyse the chemical properties of carbonated beverages commonly available in Sri Lanka in terms of the pH, calcium and fluoride ion concentrations.

Material and methods: Random samples of fifteen brands of carbonated beverages available on the market were analysed for their acidity, calcium and fluoride concentrations.

Results: The pH of the beverages ranged from 2.30 to 3.39. The calcium ion concentrations of beverages were within the range of 0.35 mmol/l and 1.12 mmol/l while the fluoride ion concentrations of all beverages were below 1 ppm and ranged from 0.038 to 0.211 ppm.

Conclusions: The present study revealed that carbonated beverages analysed have low pH values as well as low concentration of calcium and fluoride ions. Therefore these beverages may have a potential to cause dental erosion.

Key words: carbonated beverages, pH, fluoride ions, calcium ions

Introduction

Soft drinks are a common component of the diet in many parts of the world today. Global Soft Drinks report-2008 *indicates* that a total of 552 billion litres of soft drinks were consumed in 2007, which is equivalent to 82.5 litres per person and carbonated soft drinks claimed 36.8% of the soft drink market.[1] According to Naska *et al.* the availability of soft drinks in house-holds in European countries is steadily and significantly increasing particularly among the low socio-economic groups.[2] Similar trends have been observed in developing countries. India is experiencing an increase in the consumption of sugar sweetened carbonated drinks while a recent report indicates that Sri Lankans have consumed 62 million litres of carbonated soft drinks in 2009.[3,4]

It is well documented that the consumption of soft drinks has detrimental effects on oral health. In addition to dental caries, the consumption of carbonated beverages is considered as a risk factor for dental erosion as well.[5,6] The high sugar in soft drinks is responsible for dental caries while the erosive potential of a soft drink is related to its pH, titratable acidity and mineral content.[7]

It may be due to the fact that dental erosion is an emerging oral health problem in many societies and soft drinks is an important factor implicated in the aetiology of dental erosion, several studies have been conducted to determine the erosive

Dr. Nilantha Ratnayake Institute of Oral Health, Maharagama, Sri Lanka.

Prof. Lilani Ekanayake (Correspondence) Department of Community Dental Health Faculty of Dental Science, University of Peradeniya, Peradeniya. Tel: 081-2397320 Fax: 081-2388948 E-mail: lilanie@pdn.ac.lk

Mr. Berty Gangadhara Department of Basic Sciences, Faculty of Dental Science, University of Peradeniya, Peradeniya.

potential of soft drinks by analysing their pH, titratable acid content and ionic composition.[8,9] However, despite the fact that the adverse health implications of soft drink consumption are now well known and the consumption of soft drinks is high in Sri Lanka, there is no information about the properties of soft drinks available on the Sri Lankan market. Knowledge of the properties of soft drinks will be helpful in educating the public about the adverse effects related to their consumption. Therefore the aim of this study was to analyse the chemical properties of carbonated beverages commonly available in Sri Lanka in terms of the pH, calcium and fluoride ion concentrations.

Materials and methods

The present study was a component of a broader study on prevalence and risk indicators for tooth wear in 17 year olds in Sri Lanka. The ethical approval for that study was obtained from the Ethical Review committee of the Faculty of Medicine, University of Colombo.

Random samples of fifteen brands of carbonated beverages available on the market were analysed for their acidity, calcium and fluoride concentrations in the Bio Chemistry laboratory of the Department of Basic Sciences, Faculty of Dental Science, University of Peradeniya. Samples were collected randomly from different outlets in three districts of the country and one bottle from each brand was purchased from a district. The samples purchased were in sealed plastic bottles stored in a dry place at room temperature away from direct sunlight and came from different batches. The minimum volume of a bottle was 330 ml.

Determining pH of beverages:

Hanna pHep® pH meter (Hanna Instruments inc., Italy) was used to measure the pH of beverages. The instrument was calibrated using a pH 7.01 buffer solution. Measurements were made at room temperature. The first reading was taken immediately upon opening the bottle and subsequently four other readings were taken within a period of not less than 10 minutes. Since there were three bottles from each brand, 15 readings were recorded for each brand and the mean of the fifteen readings was considered as the pH of the beverage.

Determining calcium and fluoride ion concentrations of beverages:

Orion® Benchtop Ion Selective Electrode meter (Analytical Technology Inc., USA) was used for determining both the calcium and fluoride ion concentrations. Inoplus® calcium electrode (Orion Research Inc., USA) and the Orion® fluoride/ combination fluoride electrode (Orion Research Inc., USA) were used in the analysis of calcium and fluoride ions respectively. Five readings were taken from each sample and the mean was determined for each brand using 15 readings from the three samples.

Results

The ingredients and the manufacturer as listed on the label of the bottles of carbonated beverage analysed are given in Table 1.

Table 2 shows the pH, calcium and fluoride ion concentrations of the carbonated beverages that were analysed for their chemical composition. The pH ranged from 2.30 (Coca Cola®) to 3.39 (Elephant Ginger Beer®/ Seven Up®). The calcium ion concentrations of beverages were within the range of 0.35 mmol/l (My Cream soda® and My Cola®) and 1.12mmol/l (Elephant Orange Crush®). The fluoride ion concentrations of all beverages were below 1 ppm and ranged from 0.038 (My Cream Soda®, Elephant Cream Soda®) to 0.211 ppm (Seven Up®).

Calcium, fluoride ion concentrations and pH values of carbonated beverages reported in some studies conducted in developed countries are compared with the findings of the present study in Table 3. Compared to the pH values of Coca Cola® and Fanta® reported in other studies, the pH values of Coca Cola® and Fanta® samples analysed in the present study were the lowest

Discussion

Fifteen carbonated beverages available on the Sri Lankan market were analysed for their pH value, calcium and fluoride ion concentrations. As the present study was a component of a broader study on tooth wear including tooth erosion, the chemical analysis was limited to assessing the above properties. According to Lussi and Jaeggi pH value, calcium and fluoride ion concentrations are important factors that predict the erosive potential of a beverage as they determine the degree of saturation with respect to tooth mineral, which is the driving force for dissolution of tooth mineral.[7] Moreover the measurement of pH is considered as a simple and practical method to assess the erosive potential of a beverage.[10]

The pH of the carbonated beverages analyzed in this study ranged from 2.30-3.39. It is well established that enamel dissolution occurs below the pH of 5.5. Therefore all carbonated beverages considered in this study have the potential for enamel dissolution as their pH values are well below the critical level of 5.5. The inherent acidity of carbonated beverages is due to the acids that are added during the manufacturing process. Acids such as citric and phosphoric acid are added to improve the organoleptic properties of a carbonated beverage such as taste which are important for their consumption.[9] The differences in pH levels of the carbonated beverages analyzed may be attributed to varying types and amounts of acid present in the beverages. According to the labels on the bottles, the acidulant in Cola drinks analysed in this study was phosphoric acid (330) while in all other beverages the acidulant was citric acid (338). Of all the carbonated beverages Cola Cola® (pH=2.30) had the lowest pH. Similar findings have been reported in other studies as well.[11,12] It has been shown that the pH of a beverage is the strongest determinant of its erosive potential and dissolution of enamel increases logarithmically inversely with the pH of the drink.[9,13] Therefore as Coca Cola® has the lowest pH, it is reasonable to assume that it has the highest erosive potential as well. In fact epidemiological evidence substantiates this point. A study conducted among adolescents in Sri Lanka has found that consumption of Coca Cola® was significantly associated with tooth wear including tooth erosion in adolescents.[14] The pH values of Coca Cola® and Fanta® marketed in Sri Lanka are lower when compared to the pH values of the same brands sold in developed countries (Table 3). Therefore the erosive potential of these beverages may also be greater than those same brands sold in the developed countries.

Calcium, phosphate and fluoride ions could reduce the erosive potential of acidic beverages.[15] A comparison between different beverages has found that even small differences in calcium, phosphate and fluoride are responsible for the distinct differences in erosive potential of beverages.[16] This highlights the importance of these ions in influencing the erosive potential of a beverage. When the beverages were analysed for calcium and fluoride ion concentrations it was found that the concentrations of these ions were very low. Findings from other studies also indicate that calcium and fluoride ion concentrations of many carbonated beverages are low (Table 3). Though not available in Sri Lanka, several calcium enriched carbonated beverages are currently available on the market in some countries. As these beverages have a reduced capacity to demineralize enamel they may offer some benefit to those who are at risk of tooth erosion.[17]

The carbonated beverages were analysed for their properties in only one laboratory and is therefore a limitation of the present study. However, using three samples of each beverage which were purchased from three different districts for analysis and also taking five readings for from each sample may have minimized errors to a certain extent. Therefore in future studies of this nature, it would be an advantage if analysis is carried out in multiple laboratories.

The present study revealed that carbonated beverages analysed have low pH values as well

as low concentration of calcium and fluoride ions. Therefore it could be concluded that they may have a potential to cause tooth erosion.

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Table 1. Ingredients and the manufactures of beverages analysed*

Beverage	Ingredients*	Manufacturer
Coca Cola®	Carbonated water, Sugar, Acidulant (338), Caffeine, Colour (150d), Natural flavours	Coca-Cola Beverages Sri Lanka Ltd
Pepsi®	Carbonated water, Sugar, Caramel (E150d), Acidulant E338, Caffeine, Flavour	Ole Spring Bottlers (Pvt) Ltd
My Cola®	Carbonated water, Sugar, Acidulant 338, Natural flavour, Preservative 211	Sigma Overseas Holdings S&A
Elephant Orange Crush®	Carbonated water, Sugar, Citric acid, Colour (110), Preservative (211), Essence Orange Crush	Ceylon Cold Stores PLC
My Orange®	Carbonated water, Sugar, Acidulant (330), Colour (110), Flavours, Preservative (221)	Sigma Overseas Holdings S&A
Fanta Orange®	Carbonated water, Sugar, Acidulant (330), Colour (110, 112), Artificial flavours, Stabilizer (414, 444, 480), Preservative (211)	Coca-Cola Beverages Sri Lanka Ltd
Mirinda®	Carbonated water, Sugar, Acidulant (E330), Food starch, preservative E211, Colour E110, Emulsifier and Stabilizer (E445), Flavour, Buffering agent (E331)	Ole Spring Bottlers (Pvt) Ltd
My Cream Soda®	Carbonated water, Sugar, Acidulant (330), Colour (102), Natural flavours, Preservative (211)	Sigma Overseas Holdings S&A
Elephant Cream Soda®	Carbonated water, Sugar, Citric acid, Essence Cream Soda, Colour (E102), Preservative (E211)	Ceylon Cold Stores PLC
Ole Cream Soda®	Carbonated Water, Sugar, Flavour E330, E211, E102	Ole Spring Bottlers (Pvt) Ltd
7-Up®	Carbonated Water, Sugar, Acidulant (E330, 296), Buffering agent (E331), Preservative E211, Flavour	Ole Spring Bottlers (Pvt) Ltd
Sprite®	Carbonated water, Sugar, Acidulant 330, Natural flavour, Salt (331) Preservative (211)	Coca-Cola BeveragesSri Lanka Ltd
Elephant Ginger Beer®	Carbonated water, Sugar, Citric acid, Sea foaming stabiliser, Preservative (E211)	Ceylon Cold Stores PLC
My Ginger Beer®	Carbonated water, Sugar, Acidulant 330, Flavour, Preservative (E211), Colour (110)	Sigma Overseas Holdings S&A
Elephant Necto®	Carbonated water, Sugar, Citric acid, Essence Necto, Colour (E124,122,133), Preservative (E211)	Ceylon Cold Stores PLC

* As listed on bottle label
Acidulant 330= citric acid; 338= phosphoric acid

Table 2. pH, calcium and fluoride ion concentrations of carbonated beverages

Beverage	pH *mean (SD)*	Calcium mmol / l *mean (SD)*	Fluoride ppm *mean (SD)*	Fluoride mmol / l *mean (SD)*
Elephant Orange crush®	2.91 (0.14)	1.12 (0.05)	0.069 (0.013)	0.004 (0.000)
My Orange®	3.00 (0.29)	0.52 (0.08)	0.043 (0.010)	0.002 (0.000)
Mirinda®	2.69 (0.21)	0.57 (0.11))	0.198 (0.011)	0.005 (0.000)
Fanta®	2.70 (0.17)	0.55 (0.01)	0.109 (0.016)	0.006 (0.000)
My Cream Soda®	3.10 (0.13)	0.35 (0.06)	0.038 (0.009)	0.002 (0.000)
Ole Cream Soda®	3.29 (0.22)	0.54 (0.04)	0.196 (0.015)	0.010 (0.000)
Elephant Cream Soda®	3.10 (0.21)	0.53 (0.02)	0.038 (0.005)	0.002 (0.000)
Elephant Necto®	2.80 (0.12)	0.69 (0.09)	0.070 (0.022)	0.004 (0.001)
Elephant Ginger Beer®	3.39 (0.19)	0.78 (0.04)	0.061 (0.010)	0.003 (0.000)
My Ginger Beer®	2.99 (0.16)	0.49 (0.03)	0.040 (0.004)	0.002 (0.000)
Sprite®	2.99 (0.27)	0.40 (0.05)	0.098 (0.008)	0.005 (0.000)
Seven – Up®	3.39 (0.26)	0.55 (0.03)	0.211 (0.013)	0.011 (0.001)
Coca Cola®	2.30 (0.11)	0.58 (0.09)	0.122 (0.014)	0.006 (0.000)
My Cola®	2.79 (0.17)	0.35 (0.03)	0.048 (0.011)	0.002 (0.000)
Pepsi®	2.43 (0.19)	0.79 (0.07)	0.142 (0.023)	0.007 (0.001)

Table 3. Comparison of pH, calcium and fluoride ion concentrations of carbonated beverages reported in various studies with the findings of the present study

Beverage	pH	Calcium m mol/ l	Fluoride ppm	Fluoride m mol/ l
Coca Cola®				
Hara and Zero (11)	2.46	0.43		0.011
Jager *et al* (12)	2.47	0.08	-	-
Attin *et al* (15)	2.53	0.94	-	0.011
Lussi *et al* (16)	2.60	0.84	0.13	-
Zero and Lussi (17)	2.60	0.8	0.131	-
Present study	**2.30**	**0.58**	**0.122**	**0.006**
Fanta®				
Jager *et al* (12)	3.03	0.06	-	-
Lussi *et al* (16)	2.90	0.75	0.05	-
Present study	**2.70**	**0.55**	**0.109**	**0.006**
Sprite®				
Attin *et al* (15)	2.69	1.25	-	0.013
Lussi *et al* (16)	2.90	0.26	0.06	-
Present study	**2.99**	**0.40**	**0.098**	**0.005**
Pepsi®				
Lussi *et al* (16)	3.10	0.90	0.04	-
Present study	**2.43**	**0.79**	**0.142**	**0.007**
Seven-up®				
Hara and Zero (11)	3.20	0.07	-	0.003
Present study	**3.39**	**0.55**	**-**	**0.011**

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Research Article II

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Descriptive analysis of sub types of Amelogenesis Imperfecta

E.M.U.C.K. Herath, P.R. Jayasooriya, I.R. Perera

Abstract

Objective: Amelogenesis imperfecta (AI) is defined as clinically and genetically diverse group of conditions that are caused by mutations in genes. The present study aims to describe the different clinical presentations, patterns of inheritance and syndromic forms observed in a group of children with AI in Sri Lanka, in order to create awareness among Dental Surgeons, regarding this complicated group of disorders.

Study sample: The study sample comprised of 12 females and 8 males belonging to sixteen families.

Results: Hypoplastic type (Type I) of AI was the commonest form affecting 45% of the children. Thirty percent of children showed features of hypomaturation type (Type II) while 15% and 10% showed features of hypocalcification (Type III), hypoplastic/hypomaturation (Type IV) form of disease respectively. Sub typing performed with Witkop and Sauk classification as a guidline revealed all sub types except for IE (hypoplastic diffuse smooth type), IIC and D (hypomaturation-snow capped type) and IIIB (hypocalcified-diffuse type). Children who may have Kohlschutter syndrome, Cone rod dystrophy and Tricho-dento-osseous syndrome were identified and are undergoing further investigations to confirm the diagnoses.

Conclusion: By describing the different forms of AI, we expect to improve the awareness of this complicated group of conditions among Sri Lankan Dental Surgeons. In addition, the importance of identifying the syndromic forms of AI is highlighted.

Key words: Amelogenesis imperfect, pattern of inheritance.

Introduction

Amelogenesis Imperfecta (AI) is a developmental disorder of genomic origin, associated with abnormal enamel formation. Although AI is considered as a single disease entity, it actually represents a group of heterogeneous conditions, with diverse structural defects of enamel resulting in a range of clinical phenotypes.[1,2,3]

The structural defects of AI may occur at the time of formation of the organic matrix, mineralization of the matrix or maturation of enamel giv-

Dr. E.M.U.C.K. Herath BDS, MS (Restorative Dentistry), Senior Lecturer and Consultant Paedodontist, Div of Paedodontics, Department of Community Dentistry, Faculty of Dental Sciences, University of Peradeniya, Sri Lanka.

Dr. P.R. Jayasooriya (Correspondence) BDS, PhD (Japan), Senior Lecturer in Oral Pathology Department of Oral Pathology, Faculty of Dental Sciences, University of Peradeniya, Sri Lanka. Tel: +94-81-2397435 Fax: +94-81-2388948 E-mail: primalij@yahoo.com

Dr. I.R. Perera BDS, MSc, MD (Community Dentistry), Dental Public Health Specialist, Community Dental Unit, Dental Institute, Colombo, Sri Lanka.

ing rise respectively to either hypoplastic, hypocalcification or hypomaturation type of AI. However, within each of the afore mentioned categories, sub types with different patterns of inheritance and wide variety of clinical manifestations exists resulting in at least 15 subtypes of AI. Hence it is essential to use a classification to characterize the different sub types of AI. The most widely used classification by Witkop and Sauk (Table 1) is based on phenotype and pedigree analysis.[4] Although an ideal classification should also include genomic analysis to identify the mutations associated with the sub types, it is not used routinely for diagnostic purposes.[2,3]

At present, AI is diagnosed based on the family history, pedigree plotting and clinical observation. Furthermore, the diagnosis involves exclusion of other enamel defects such as fluorosis and chronological hypoplasia, prior to establishment of likely inheritance patterns and clinical phenotype using Witkop and Sauk classification as a guideline.

The prevalence of AI varies from 1:700 to 1: 14,000 depending on the population studied.[3] Although, AI is not a very common disorder that the Dental Surgeons would encounter in their day-to-day practices, they may see a few patients over the years. As the correct identification is essential to provide the best management, which should not only include restorative treatment, but also genetic counseling as well as reassurance of both parents and patients, thorough knowledge of this complicated group of conditions are essential.

Therefore, the present study aims to describe the different clinical presentations, inheritance patterns and syndromic forms observed in a group of children with AI in Sri Lanka, in order to create awareness among Dental Surgeons, regarding this complicated group of disorders.

Method

Patient selection

Children presenting to the Division of Paedodontics, Faculty of Dental Sciences, University of Peradeniya over a period of two years from 2008 to 2010, with the aim of seeking treatment for discoloured/ sensitive or abnormal appearance of teeth were first screened to identify the patients suffering from AI. AI was diagnosed by evaluating the history, pedigree analysis, clinical and radiological appearance of teeth, and histopathological specimens when available. Sub typing of AI was performed using the findings afore mentioned with Witkop and Sauks classification as a guideline (Table 1).

The study sample thus obtained included twenty children with AI, belonging to 16 families with four families contributing two children with AI each.

Results

The demographic data with the diagnosis including type and sub type of AI achieved for each child is shown in Table 2.

In the present study sample, the mean age of the children affected with AI was 12 years and ranged from 3-17 years. Female predilection (12/20) with male to female ratio of 2:3 was also noted. With reference to racial distribution, majority of the affected children were Sinhalese 65 %(13/20) followed by 15 %(3/20) Tamils and 20 %(4/20) Muslims.

Out of the twenty patients with AI, 45 %(9/20) had hypoplastic (Type I) (Fig 1) form of disease while 30 %(6/20), 15 %(3/20), 10 %(2/20) respectively had hypomaturation (Type II)(Fig 2), hypocalcification (TypeIII) (Fig 3), hypoplastic/ hypomaturation (Type IV) form of disease.

With reference to identification of patterns of inheritance and pedigree plotting, a positive family history could be identified in 50% (8/16) of the families having children with AI. With pedigree

plotting, positive identification of pattern of inheritance as either autosomal dominant or x-linked pattern was possible in 7 families (8 children) and one family (one child) respectively. In addition, in the group of children without positive family history, 8 had parents who were related to each other leading to consanguinity in the family. These 8 children were considered to show autosomal recessive pattern of inheritance, as consanguinity is known to give rise to diseases showing afore mentioned pattern of inheritance. The remaining 3 children without identifiable pattern of inheritance were considered as sporadic cases of AI.
The clinical presentation of AI to some extent depends on the type and in the present study out of the 9 children showing hypoplastic pattern, two children in one family presented with generalized pitted sub type (IA) where teeth had enamel which contrasted well with underlying dentine radiologically. Clinically pits were seen arranged in to horizontal and vertical lines in areas with other areas showing a more haphazard distribution. These pits were more common on the buccal surfaces of teeth compared to other surfaces. As, no other family member was affected; these children were considered to have a sporadic form of AI (Patient no 16 and 17, Table 2). Three children belonging to two families had localized pitted sub types (IB and IC) where the pits were seen on buccal surfaces mainly with incisial edges showing regular enamel, which contrasted well with dentine radiologically. Out of the three children one child (Patient no 9, Table 2) had IB sub type with autosomal dominant type of inheritance, while in the remaining two children both dentitions were affected, in addition the parents of these children were related allowing the condition to be classified as IC with autosomal recessive type of inheritance (Patient no 4 and 5, Table 2). None of the children included in the study had diffuse smooth type (IE); hence it was not possible to demonstrate the lionization effect, which is observed in females. Two children in one family showed diffuse rough pattern (IF), with teeth shaped as crown preparations and containing open contact points, exhibiting a very thin layer of enamel (Patient no 13 and 14, Table 2). Two children had teeth with exposed rough dentine without any enamel and anterior open bite, which was categorized as enamel agenesis type (IG) (Patient no 15 and 20, Table 2).

Four and two children belonging to three and two families respectively showed hypomaturation type diffuse pigmented (IIA) (Patient no 7, 10, 18 and 19, Table 2) and diffuse pigmented (IIB) (Patient no 1 and 11, Table 2) pattern of AI. These children had enamel, which showed a similar radio density to dentine. Clinically the shapes of the teeth were normal while colour ranged from mottled to yellow brown. With reference to patient no 11, both mother and sister of the patient showed less severe form of slight irregularly arranged vertical bands of opaque enamel alternating with normal enamel under transillumination. In our study sample no children had snow capped pattern of hypomaturation type of AI.

Three children presented with hypocalcification type of AI, where the parents said that the children had brown, yellow colored teeth, which were appropriately shaped on eruption. On examination, most of the teeth did not show any enamel on occlusal surfaces, while except for the cervical portion other areas had exposed dentine (Patient no 2, 3 and 6, Table 2). Two children also exhibited rapid calculus deposition. The study sample included children with hypocalcified type of AI showing only autosomal dominant inheritance pattern where the teeth are less severely affected (sub type IIIA). None of the children in the present sample had autosomal recessive inheritance pattern (sub type IIIB) with severely affected teeth.

Two children showed AI of Hypomaturation-Hypoplastic type with Taurodontism (AIHHT) (Type IV) where pits were evident on the buccal surfaces with other areas showing a yellowish brown colour (Patient no 8 and 12, Table 2). Radiological investigations revealed single rooted teeth with large pulp chambers and molars showing varying

degrees of taurodontism. However, features of Tricho-dento-osseous syndrome (TDO) including curly hair and bone sclerosis in addition to AI were only observed in patient number 12.

Discussion

According to earlier definitions AI was considered as a specific enamel defect without the involvement of other structures.[5] However, recent findings have led AI to be considered as a genetic disease that may exist in isolation or associated with other features in syndromes.[3] Syndromes/conditions that have been associated with AI include Kohlschutter syndrome, TDO syndrome, Platyspondyly, Nephrocalcinosis and Cone- rod dystrophy.[3] One child (Patient no 15-Table 2) included in the present study may have features of cone rod dystrophy that has been linked to a micro deletion at 2q11 with autosomal recessive type of inheritance.[6,7] In our patient also, the autosomal recessive pattern of inheritance could be confirmed as she was from a family exhibiting consanguinity. At present the patient is undergoing further investigations to confirm the diagnosis. In addition, another child (Patient no 10-Table 2) coming from a consanguineous family had epilepsy and is undergoing further investigations to determine if he is suffering from less severe form of Kohlschutter syndrome.[8,9] Although, patient no 8 and 12 both have AIHHT, patient number 8 did not show features of TDO syndrome, such as curly hair and skeletal changes including bone sclerosis, while patient no 12 showed features of TDO syndrome. According to a previous study taurodontism of the first mandibular molar has been shown to be useful to distinguish between TDO syndrome and AIHHT.[10] Supporting the above conclusion, patient no 8 did not show taurodontism of mandibular first molar in contrast to patient number 12, who presented with severe form of taurodontism of mandibular first molar. According to literature, Nephrocalcinosis is thought affect children who have hypoplastic type of AI, which is inherited in autosomal recessive trait.[11] Therefore, patients, no 4, 5 and 20 who have fulfilled above criteria will be investigated to identify whether they are suffering from Nephrocalcinosis.

AI is a complicated group of conditions resulting in different clinical phenotypes, which usually correspond to the structural defect. As, diagnosis based purely on the clinical phenotype result in over/under diagnosis of the condition, clinicians recommend the use of both clinical observation and pedigree analysis when diagnosing AI. Following clues were used to identify the inheritance pattern of AI in the present study,

1. Autosomal dominant inheritance: Male to male transmission with approximately half the offspring of an affected individual showing clinical features of disease and affected males and females showing similar clinical features.

2. Autosomal recessive trait: Unaffected parents having affected offspring with parents in consanguineous marriages.

3. X-linked inheritance:
   Recessive trait: No male-to-male transmission
   Dominant trait: Male offspring showing more severe presentation with some females in the family showing lionization.

However, in the present study several difficulties/ problems were encountered in the pedigree analysis. With reference to patients exhibiting autosomal dominant type of inheritance, horizontal spread among the siblings of the affected parent's family was not observed except for three families. In addition, the disease was also not observed among cousins (first degree relatives), even when a grandparent was affected. However, it is difficult to determine whether the information related to siblings and cousins are true as the authors were unable to examine all the individuals of the affected families and the information was gathered by speaking to the parents of the affected children only.

It was also not possible to sub classify the type of AI in affected parents as most were wearing prosthesis. However, a single parent (mother) (Patient no 11-Table 2) showed a less severe form than the child. It was correlated to the x-linked type of inheritance observed in the family. The less severe form observed in the mother can be contributed to the occurrence of the lionization (inactivation of one X chromosome, while the other X chromosome is active) phenomenon which gives rise to different clinical appearances in male and females. [4]

In the present study, patients without a positive family history or consanguinity in the family were considered as having sporadic form of AI. However, sporadic form may actually represent examples of new mutations or variable expression with or without incomplete penetrance of a dominant gene. [3] Hence, although patients no 16 and 17 were considered to have the sporadic form, they may actually have autosomal dominant type of inheritance with variable expression in the parent (father). Supporting this fact, father and children, showed peg laterals which is also thought to be transmitted in an autosomal dominant form.[4]

Although, with available information authors attempted to identify the mode of inheritance in the present group of patients, speculative nature of the findings are acknowledged. Hence, in order to confirm the diagnosis and the nature of the mutations genetic analysis is recommended. According to previous studies, X-liked form of AI has been shown to be associated with mutations in AMLEX gene that codes for amelogenin. With reference to autosomal dominant forms of AI, mutations in enamelin coding gene ENAM has been identified to contribute to hypoplastic type, while the molecular aetiology of the hypocalcified type remains unknown to date.[12] TDO syndrome is contributed to DLX3 mutations while molecular aetiology of AIHHT remains unknown.[13] Autosomal recessive form of AI has been shown to be associated with mutations in genes coding proteinases, enamelysin (MMP-20) and kalikrein 4, responsible for processing the extra cellular matrix.[12] However, it was not possible to perform genetic analysis for the present group of patients due to the cost involved.

By describing the different forms of AI, we expect to improve the awareness of this complicated group of conditions among Sri Lankan Dental Surgeons. In addition, the importance of identifying the syndromic forms of AI is highlighted. This baseline data could be used to emphasize the importance of early accurate diagnosis leading to better treatment outcomes contributing to improved quality of life among patients affected by AI.

Table 1. Classification of Amelogenesis Imperfecta

Type	Pattern	Specific features	Inheritance
IA	Hypoplastic	Generalized pitted	Autosomal dominant
IB	Hypoplastic	Localized pitted	Autosomal dominant
IC	Hypoplastic	Localized pitted	Autosomal recessive
ID	Hypoplastic	Diffuse smooth	Autosomal dominant
IE	Hypoplastic	Diffuse smooth	X linked dominant
IF	Hypoplastic	Diffuse rough	Autosomal dominant
IG	Hypoplastic	Enamel agenesis	Autosomal recessive
IIA	Hypomaturation	Diffuse pigmented	Autosomal recessive
IIB	Hypomaturation	Diffuse	X linked recessive
IIC	Hypomaturation	Snow capped	X linked
IID	Hypomaturation	Snow capped	Autosomal dominant?
IIIA	Hypocalcification	Diffuse	Autosomal dominant
IIIB	Hypocalcification	Diffuse	Autosomal recessive
IVA	Hypomaturation-hypoplastic	Taurodontism present	Autosomal dominant
IVB	Hypoplastic-hypomaturation	Taurodontism present	Autosomal dominant

Table 2. Demographic data with the diagnosis of the study population

No	Age/ Sex	Race	Family history	Are the Parents Related?	No of Children with AI	No of Children without AI	Inheritance pattern	Type of AI	Subtype of AI
1	9M	S	No	No	01	02(F)	Sporadic	Hypomaturation (II)	IIB
2	12F	S	Yes Mother	No	01	01(M)	AD	Hypocalcification (III)	IIIA
3	13M	S	Yes Mother	No	02(M)	01(M)	AD	Hypocalcification (III)	IIIA
4*	13F	T	No	Yes	02(F, M)	00	AR	Hypoplastic (I)	IC
5*	15M	T	No	Yes	02(F, M)	00	AR	Hypoplastic (I)	IC
6	8F	M	Yes Mother	No	01	01(M)	AD	Hypocalcification (III)	IIIA
7	6M	S	No	Yes	01	01(M)	AR	Hypomaturation (II)	IIA
8	12F	T	Yes Grandmother	No	02(M, F)	01(M)	AD	Hypoplastic-hypomaturation (IV)	IVA
9	17M	M	Yes Father	No	02(M,F)	00	AD	Hypoplastic (I)	IB
10	15M	M	No	Yes	01	02(M,F)	AR	Hypomaturation(II)	IIA
11	9M	M	Yes Grandfather Mother	No	02(M,F)	01(M)	X-linked	Hypomaturation(II)	IIB
12	13F	S	Yes Grandfather Mother	No	01	02(F)	AD	Hypoplastic-hypomaturation (IV)	IVA (TDO)
13*	13F	S	Yes Grandfather Father	No	02	00	AD	Hypoplastic (I)	IF
14*	16M	S	Yes Grandfather Father	No	02	00	AD	Hypoplastic (I)	IF
15	13F	S	No	Yes	01	01(M)	AR	Hypoplastic (I)	IG
16*	15F	S	No	No	02(F)	01(M)	Sporadic	Hypoplastic (I)	IA
17*	16F	S	No	No	02(F)	01(M)	Sporadic	Hypoplastic (I)	IA
18*	9F	S	No	Yes	02(F)	00	AR	Hypomaturation(II)	IIA
19*	3F	S	No	Yes	02(F)	00	AR	Hypomaturation(II)	IIA
20	12F	S	No	Yes	01	02(M,F)	AR	Hypoplastic (I)	IG

* Patient nos 4,5/13,14/16,17/18,19 are siblings

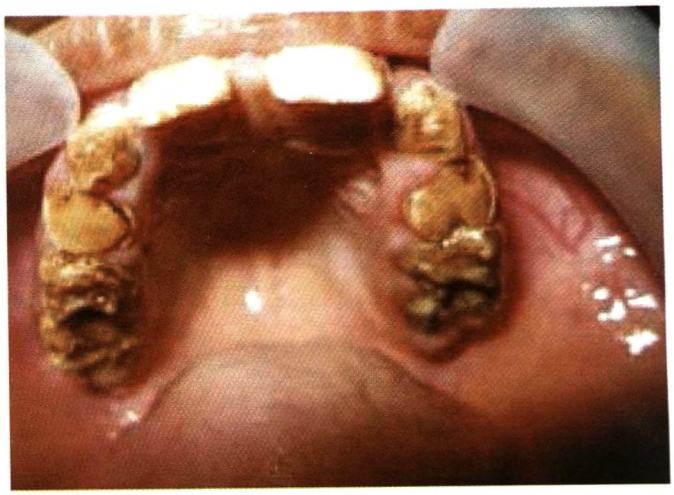

Figure 1. Amelogenesis imperfecta - Hypoplastic type

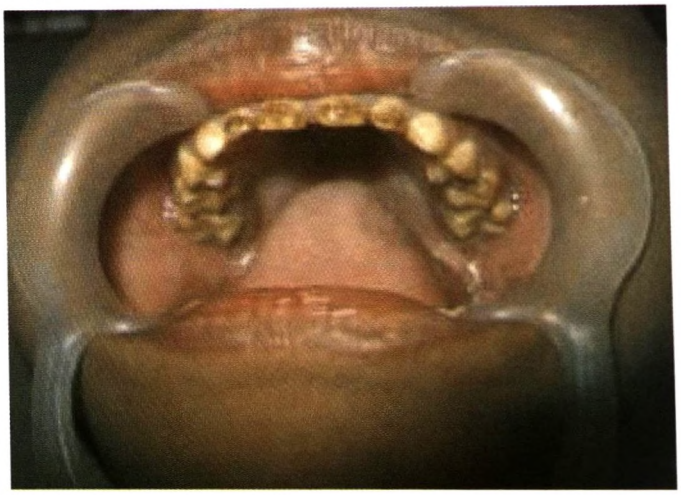

Figure 2. Amelogenesis imperfecta - Hypomaturation type

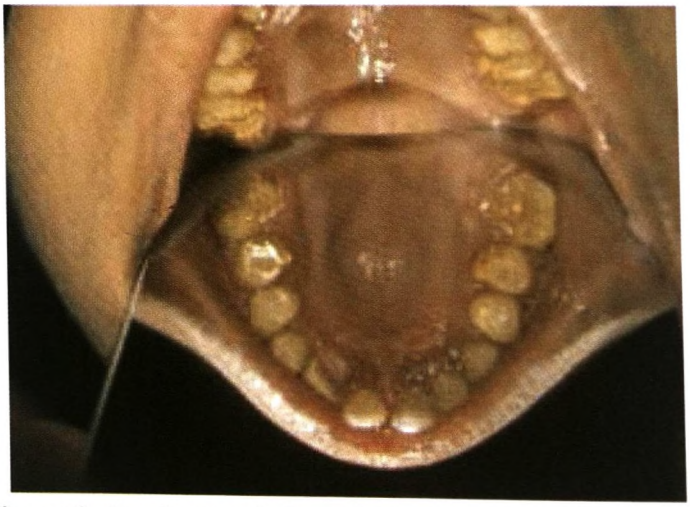

Figure 3. Amelogenesis imperfecta - Hypocalcification type

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Case Report

Sri Lanka Dental Journal 2011; 41(01-02) 26-32

Central Plexiform Schwannoma of Maxilla: A rare case report

Pravin Lambade, Akshay Dhoble, Ashwini Ramakrishna, Deepti Lambade, Alka Dive

Abstract

Plexiform schwannomas are tumors with very low incidence. Plexiform or multinodular growth pattern is reported in 5% of schwannoms. Intraosseous Schwannomas in the maxilla are even rarer. Here we report the first case of central plexiform schwannoma of maxilla in a 33 year old male patient which had grown in a multinodular pattern. The tumor was excised and histologically found to have Antoni type A, type B cells, verocay bodies and plexiform pattern of cellular distribution. The patient was followed up for 18 months with no recurrence.

Key words: Central Schwannoma, Plexiform Schwannoma, intraosseous schwannoma Neurilemmoma, Neurinoma.

Introduction

Schwannoma is a benign tumor that originates from Schwann cells of the peripheral nerves. It can occur at any location where there are nerves with Schwann cells.[1] Clinically tumor affects all ages but peak incidence is between third and sixth decade. Major sites of involvement are the head and neck region and flexor surfaces of the extremities. Sensory cranial and spinal nerves are affected more often than sympathetic nerves and most examples are solitary. Salient gross features are globoid, encapsulated, firm mass that on sectioning is partly hemorrhagic or cystic and is homogeneously tan colored.[2]

According to literature 45 cases of intraosseous schwannoma have been reported in either of the jaws and out of which 41 cases have been reported in the mandible and only 4 cases in the maxilla.[3,4,5]

Five percent of schwannomas grow in a plexiform or multinodular pattern which may or may not be apparent macroscopically. Plexiform schwannomas usually occur in the skin and infrequently in deep sites.[6] Here we report the 1st case of Central Plexiform Schwannoma which grew in a multinodular pattern in the maxilla.

Prof. Pravin Lambade (Correspondence) Professor and Head, Department of Oral and Maxillofacial surgery, Rungta College of Dental Sciences and Research, Khoka kurud road, Bhilai-490024, Chattisgarh, India. Phone: +919822236370, 9893951010. E-mail: drpravinlambade@gmail.com Fax: +07882286481

Dr. Akshay Dhoble Senior lecturer, Department of Oral Pathology and Microbiology, VSPM Dental college, Nagpur 440015, Maharashtra, India.

Dr. Ashwini Ramakrishna Senior lecturer, Department of Oral Pathology and Microbiology, Rungta college of Dental Sciences and Research, Bhilai 491024, CG, India.

Dr. Deepti Lambade Senior lecturer, Department of Prosthodontics, VSPM Dental college, Nagpur 440015, Maharashtra, India.

Prof. Alka Dive Professor and Head, Department of Oral Pathology and Microbiology, VSPM Dental college, Nagpur 440015, Maharashtra, India.

Case report

A 33 year old male patient reported with the main complaint of swelling in right cheek since 8 months and he had undergone an extraction of 17, one year back. No pain or paraesthesia was associated with it.

Extraorally it was a diffuse swelling in the right malar region of approximately 3 cm in diameter, ovoid, normal skin colored, non tender and firm in consistency. Intraorally, single diffuse swelling of approximately 3 cm in diameter extending from the apical region of 16 below the right zygomatic buttress obliterating the right buccal vestibule, extending to the right maxillary tuberosity area.The swelling was non tender, non mobile and firm to hard on palpation and the overlying mucosa appeared to be normal (Fig 1). OPG revealed diffuse radiolucency of approximately 2 cm in diameter, distal to the roots of 16 causing root resorption in the apical region of distal root of 16 (Fig 2 and 3).

Based on the above clinical and radiological features the differential diagnosis of residual cyst, central ossifying fibroma, central giant cell granuloma was made. Haemogram was within normal limits.

Incisional biopsy - H&E stained section revealed well encapsulated lesion, which is composed of spindle shaped cells arranged in palisading pattern with homogenous eosinophilic areas known as Verrocay bodies as seen in Antoni A type. While irregularly arranged Antoni B type tissue was also evident. From these features diagnosis of Schwannoma was made.

Surgical excision under general anesthesia with primary closure of the surgical defect was carried out. Patient was followed up to 18 months with no recurrence.

The gross examination of the excisional biopsy revealed a multi nodular tan colored specimen of approximately 7 cm in diameter, multiple small nodules attached to one another (Fig 4).

Excisional biopsy - H&E stained section revealed well encapsulated lesion forming multiple large and small nodules (Fig 5). Lesional tissue composed of spindle shaped cells arranged in palisading pattern with homogenous eosinophilic areas resembling Verrocay bodies as seen in Antoni A type tissue (Fig 6 and 7). While irregularly arranged Antoni B type tissue was also evident. From these features final histopathological diagnosis of Central plexiform schwannoma of right maxilla was made probably arising from posterior superior alveolar branch of Maxillary division of trigeminal nerve.

Immunohistochemical staining shows positivity to S100 protein revealing neural origin of the tumor (Fig 8).

Discussion

Schwannomas are a group of neoplasms composed of differentiated spindle cells having the ultra structure and immunophenotype of Schwann cells. Three variants are currently appreciated they are classical schwannoma, cellular schwannoma and melanotic schwannoma.[2] Most schwannomas are uninodular masses surrounded by a fibrous capsule consisting of epineurium and residual nerve fibers.[6]

Gabhane *et.al* (2009) reported four morphologic variants of schwannomas namely Usual, Ancient, Cellular, and Plexiform. Out of which Plexiform had the lowest percentage (4.68%).[7]

Plexiform schwannoma is a rare nodular variant, except for its multinodular or plexiform architechture, it has the same histologic features as the usual schwannoma but with marked cellularity dominated by Antoni type A pattern.[7]

"Plexiform" in the setting of peripheral nerves refers to a process involving either multiple nerve

fascicles or nerves. Plexiform schwannomas have been reported to recur locally and sometimes erode bone.[2]

Five percent of schwannomas grow in a plexiform or multinodular pattern which may or may not be apparent macroscopically. Plexiform schwannomas usually occur in the skin and infrequently in deep sites. Like classic schwannoma, they are encapsulated but as a group tend to be more cellular. It is important to be aware of this fact as there is a risk of misinterpreting a lesion as a sarcoma arising in a plexiform neurofibroma. The fact that the lesion does not have a level of atypia commensurate with the mitotic activity, lacks geographic necrosis, and displays strong S-100 protein staining provides good support for benignancy in such cases.[6]

Intraosseous Schwannoma rarely occurs in the oral cavity less than 1%, but when they occur mandible is the most common site.[4,8] First identified by Virchow in 1908, Verocay later termed it Neurinomas. Approximately 25-40% of all schwannomas are seen in head and neck region.[9,10]

Schwannomas arise from neural sheath of peripheral sensory, motor, sympathetic and cranial nerves with the exception of olfactory and optic nerves since they lack sheaths that contain Schwann cells.[10,11] Etiology is still unknown and mostly they are asymptomatic.[9,10]

These tumors are characteristically slow growing, well encapsulated, round or fusiform and closely associated with the nerve of origin and are usually solitary and painless.[2,6,10] It is difficult to identify the nerve from which they originate.[7,9,11]

Considering the site of our case an assumption of the posterior superior alveolar branch of the maxillary division of the trigeminal nerve can be made although the nerve was not identified at the time of surgery.

Reviewed literature revealed a lot of schwannomas of the mandibular division but rarely any case arising from maxillary division of trigeminal nerve causing swelling intraorally. The clinical course and radiographic appearance of schwannoma is not characteristic, therefore clinical diagnosis not possible and can only be established on histologic examination.[5] Radiographically, schwannomas of either jaw are well demarcated , unilocular radiolucencies with a thin sclerotic border. Additional features such as external root resorption, cortical thinnin, spotty calcification, cortical expansion and peripheral scalloping can be evident.[3]

Histologically, schwannomas are typically encapsulated, but unencapsulated cases have been reported. The tumor consists of Antoni A and Antoni B zones arranged in a palisading pattern interspersed with verrocay bodies.[8] Immunohistochemically schwannomas show strong S-100 and CD34 staining. The recommended treatment for intraosseous schwannomas is surgical excision with periodic follow ups.[4]

Conclusion

Five percent of all schwannomas have a cellular distribution in a plexiform pattern. Mostly such cases are present subcutaneously, intraosseous sites are rare for this type of schwannoma. Proper histologic assessment is essential for there are chances of missing a tumor nodule during surgery which could be the cause of recurrence.

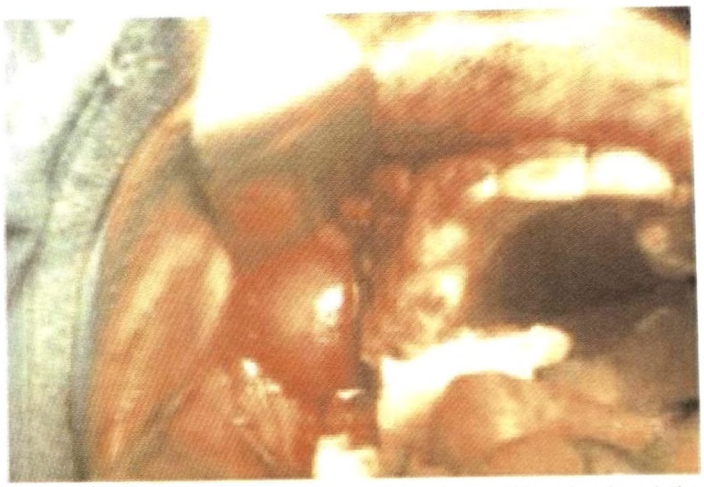

Figure 1. Intra operative view of capsulated swelling in the right maxilla

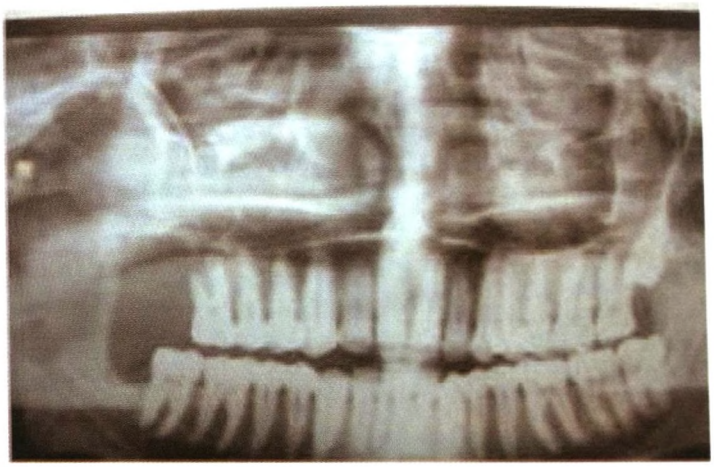

Figure 2. OPG to show the diffuse radiolucency of approximately 2 cm in diameter, distal to the roots of 16 causing root resorption in the apical region of distal root of 16.

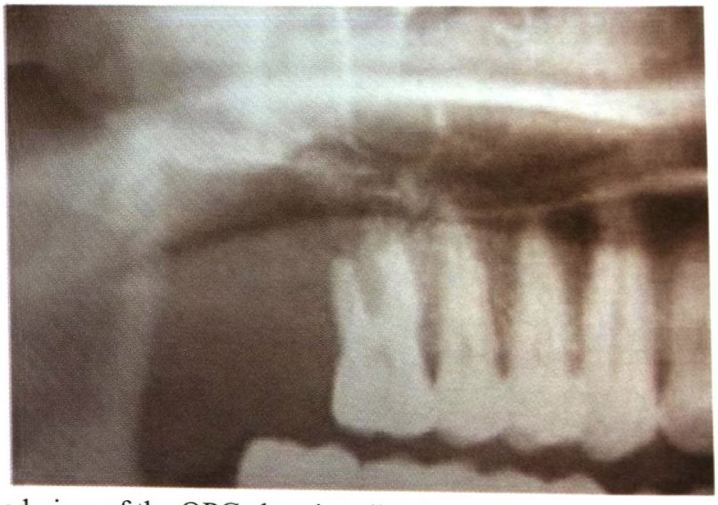

Figure 3. Cropped view of the OPG showing distal root resorption and diffuse radiolucency

Figure 4. Multi nodular tan colored specimen of approximately 7cm in diameter

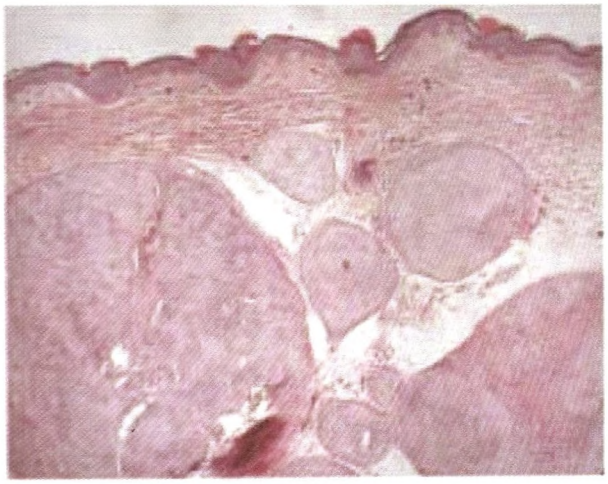

Figure 5. Low power view of the H&E section to show the multiple nodules of lesional tissue

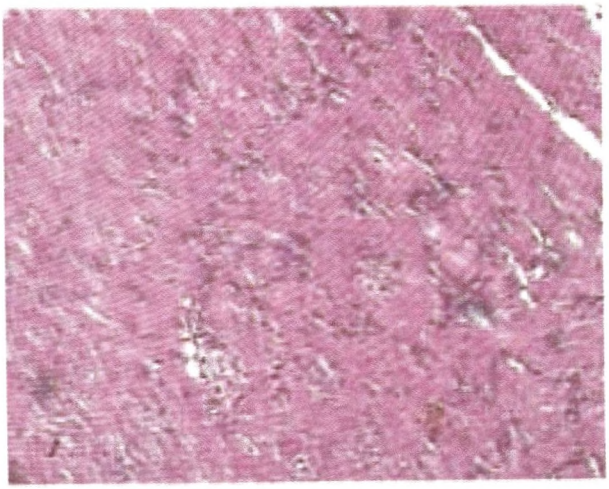

Figure 6. High power view of the H&E section to show Antoni type A Cells (Palisading arrangement)

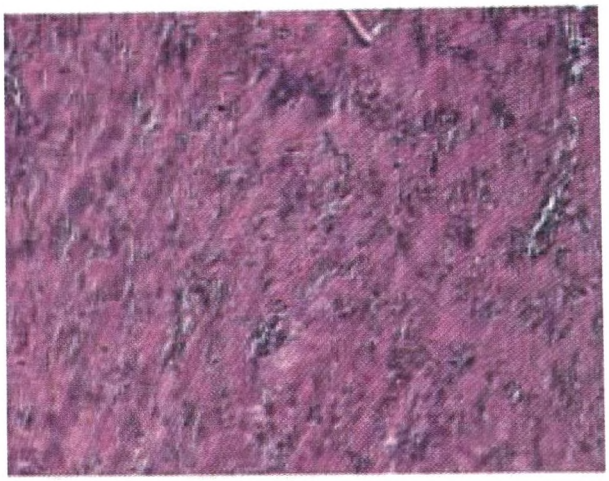

Figure 7. Low power view of the H&E Stained section shows Antoni type A & Type B with homogenous eosinophilic areas-Verrocay bodies.

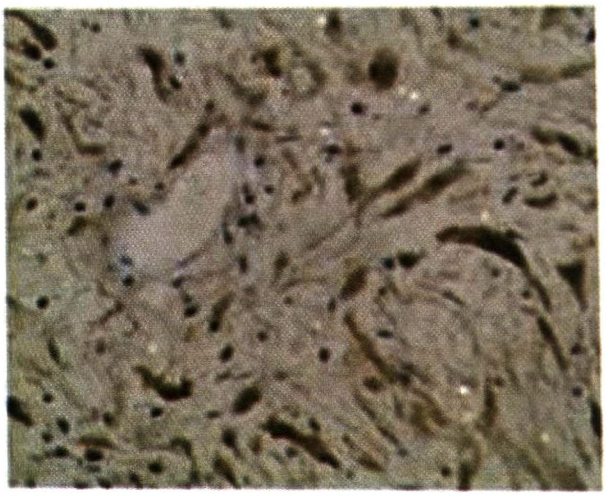

Figure 8. S-100 immuno positivity of the lesional tissue

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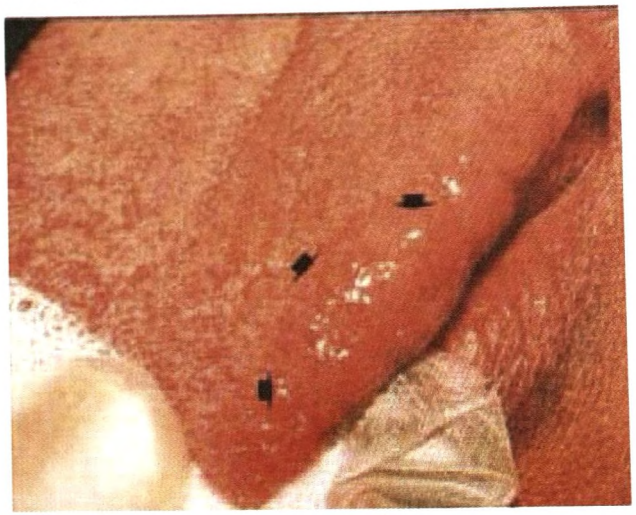
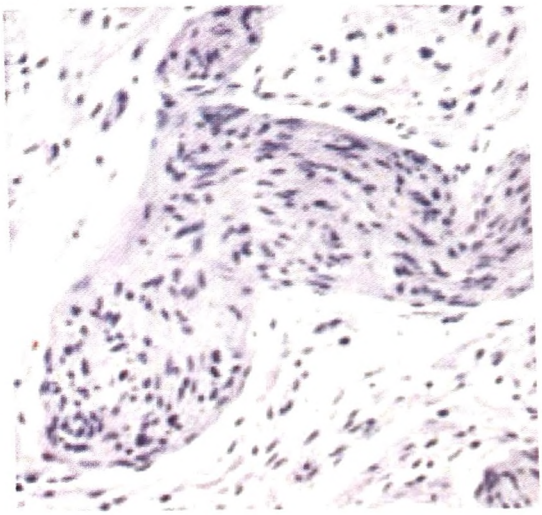
Self Assessment - Oral Diagnosis (SAOD)

Patient presented with a swelling on lateral border of the tongue





1. What is the most probable diagnosis?
   a) Plexiform neurofibroma
   b) Schwannoma
   c) Granular cell tumour
   d) Fibroepithelial polyp
   e) Lipoma

2. If the patient is having multiple lesions, what could be the systemic disease?
   a) Gorlin-goltz syndrome
   b) Neurofibromatosis type 2
   c) Sjorgren's syndrome
   d) Multiple Endocrine Neoplasia type IIb (MEN type IIb)
   e) Neurofibromatosis type 1

3. What could be the other features that help you to identify the systemic disease
   a) Multiple café-au-lait spots
   b) Two or more Lisch nodules
   c) Scoliosis
   d) Freckling in the axillary or inguinal region
   e) High blood pressure

Prepared by Dr. B.S.M.S. Siriwardena (BDS,MPhil,PhD)
Department of Oral Pathology
Faculty of Dental Sciences
University of Peradeniya

Answers

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

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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 ill vestigations also may be accepted.

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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.

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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.

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.

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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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Results - The Results section should clearly and concisely present the findings of the research, as a rule in the past tense without subjective comments and reference to previous literature. The results should be supported by statistical or illustrative validation. For the sake of clarity this section may have subheadings.

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Journals

Standard journal article

Bartlett IG, O'Keefe P. The bacteriology of the perimandibular space infections. JOral Surg 1979; 37: 407409.

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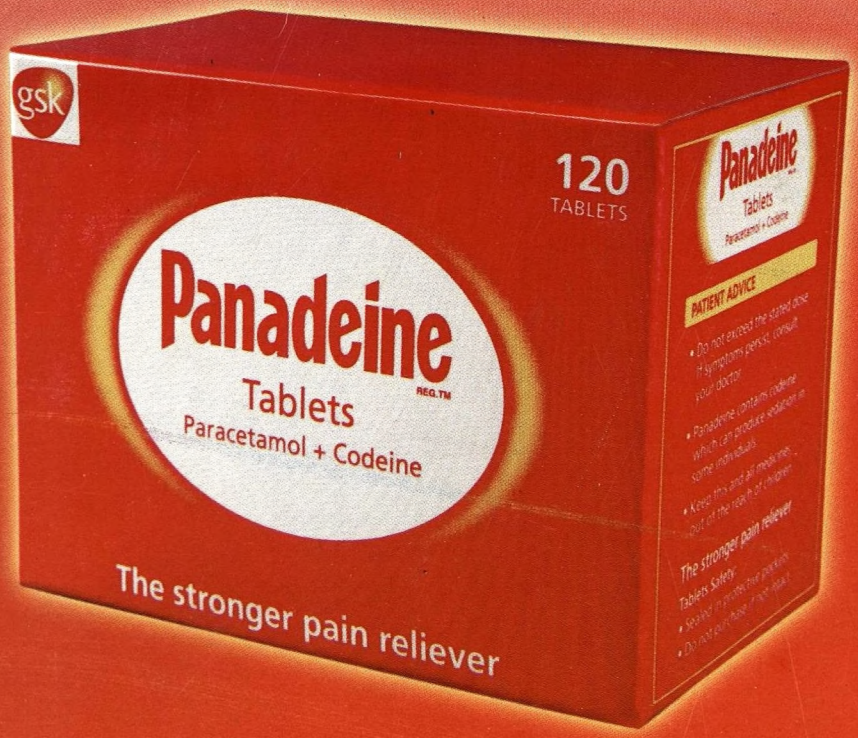

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