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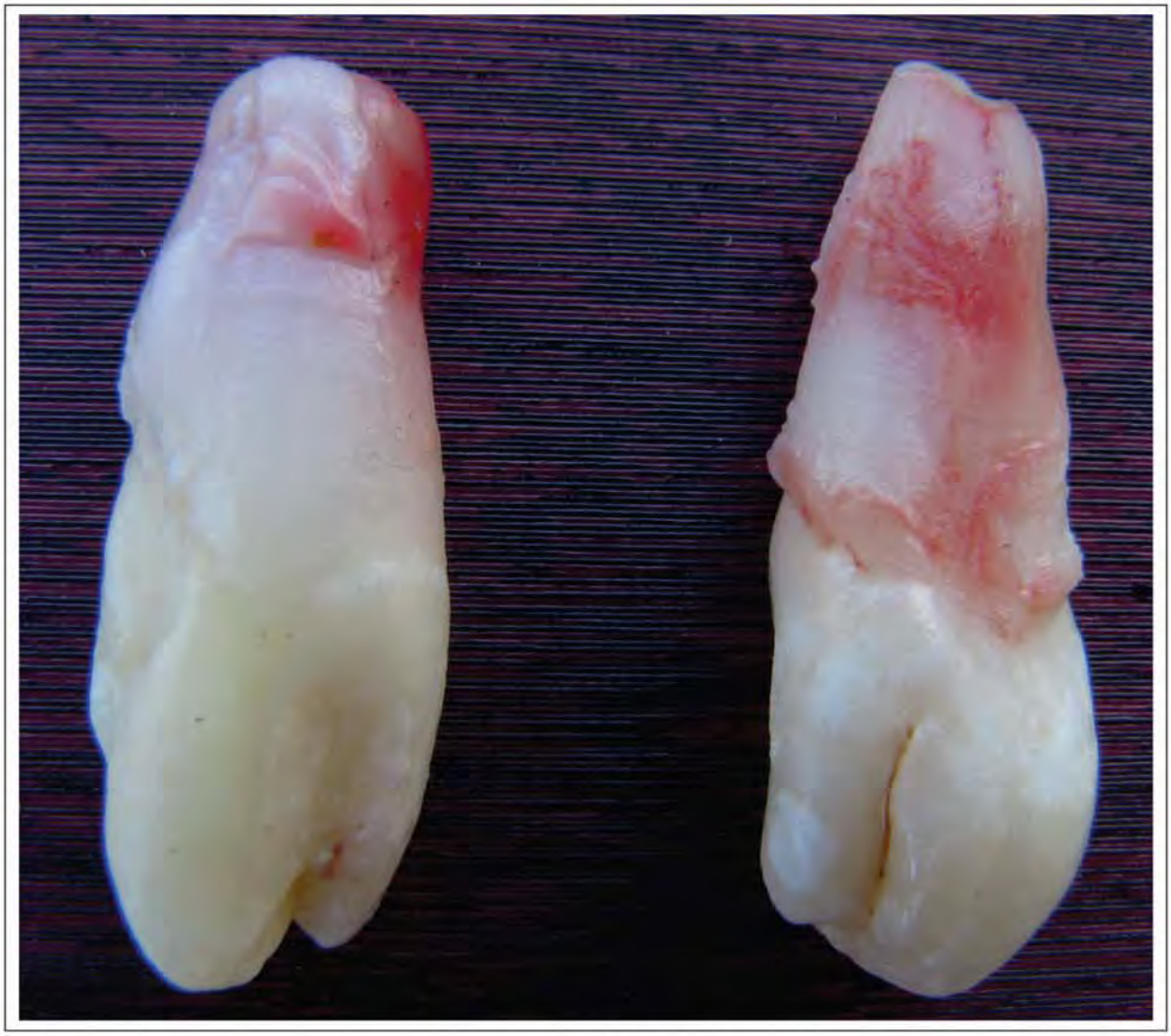

January to June 2014; Volume 1, Issue 1

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Manuscripts: Articles should be type written on one side of A4 size (21x28cm) White paper in double spacing with a sufficient margin. One Original and two high quality photostat copies should be submitted. The author's name is to be written only on the original copy and not on the two photostat copies. **In addition to the printed version, a CD containing the article file also should be submitted compulsorily.** Use a clear and concise reporting style. Journal of Maxillofacial Science and Research reserves the right to edit manuscript, to accommodate space and style requirements. Authors are advised to retain a copy for the reference.

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Messages

Message by Chairman

PMS College of Dental Science & Research
Golden Hills, Venkode P.O.,
Vattappara, Trivandrum - 695028

Dental education is a continuous process and it is mandatory to stay up to date with the recent advancements in dentistry. It is the need of the hour to have a good platform to take the advancements in the field to the masses and the best medium for this is to bring out scientific publications of the nature of "JOURNAL OF MAXILLOFACIAL SCIENCE AND RESEARCH".

I understand that that this journal is enriched with original articles, case studies and reports, and research articles from all over India. I congratulate all who have contributed to bring out such a publication and wish all success.

Dr P S Thaha
Chairman

Messages

Message by Principal

PMS College of Dental Science & Research
Golden Hills, Venkode P.O., Vattappara, Trivandrum - 695028

I am very much pleased to notice that the PMS College of Dental Science is coming out with a Journal which is a debut venture of its kind. This will definitely provide a platform to the faculty as well as students of our College to publishing the Scientific articles. More over this will encourage scientific aptitude. I appreciate the editor and the management for all their support. All the very best for the venture.

Principal
Dr. N.O. VARGHESE

Editorial

Journal of Maxillofacial Science and Research (JMFSR)

Dentistry which revolved only around the teeth, has over a short period of time evolved from its humble beginnings to become a very important branch of medicine that involves the health of oral and maxillofacial structures. Imbibing new information and experiences in this fast and ever changing oral and maxillofacial world, is often very challenging. Considering the present day evidence based practice, communicating the information gained for the benefit of our patients, profession and society, is even more challenging. In spite of all developments in communication, documentation and information sharing, scientific journals are still considered the gold standard in providing evidence based information.

Our visionary chairman Dr PS Thaha, a widely travelled educationist, entrepreneur , quite interested in keeping abreast with the latest in dentistry , is the man behind the idea of a scientific journal for our college by our college "JOURNAL OF MAXILLOFACIAL SCIENCE & RESEARCH". By leading us [the editorial board] to create a critical and reactive journal, Dr. Thaha intends to create a space where the students and faculty, can "test the academic waters" and think deeply on their invaluable experiences.

JOURNAL OF MAXILLOFACIAL SCIENCE & RESEARCH [JMFSR], the official publication of PMS College of Dental Science & Research is intended to be a research periodical that aims to inform its readers of ideas, opinions, developments and key issues in dentistry - clinical, practical and scientific - stimulating interest, debate and discussion in all disciplines of dentistry. Rigorous peer review by our excellent review board is the hall mark of JMFSR. The Journal is designed with a view to enable fast and easy access to information for its readers. I take this opportunity to request, all the staff and students of PMS college of dental science and research, to support JOURNAL OF MAXILLOFACIAL SCIENCE & RESEARCH by contributing articles, letters, opinions, and last but not the least, identifying and procuring finance for making the journal self sustainable.

Dr. (Capt) V. Vivek,
Editor in Chief
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CASE REPORT

An inverted impacted maxillary 3rd molar, what to do? A case report with review

Basil. M. Jacob[1], Dheeraj Eldho Paulose[2], Nithin Mathew Cherian[3], Ninan Thomas[4], Sankar Vinod V[5].

Abstract

Surgical extraction of impacted third molars is the most common procedure performed by the oral surgeon. Any tooth including the deciduous and the permanent can be impacted but the more common are the maxillary and mandibular third molars and the maxillary cuspids followed by the premolars and the supernumerary teeth[2]. Among the third molar impactions the most common is the mandibular. Maxillary third molar impactions being equally present but have been overlooked. Still rare are literature about inverted impacted maxillary third molars. Here we present a case report with review of literature.

Keywords : Inverted tooth, Maxillary impaction, impaction

Introduction

Archer defined an impacted tooth as one which is completely or partially unerupted and is positioned against another tooth or bone or soft tissue so that its further eruption is unlikely[1]. An increased awareness among the general population, easyaccess to surgical treatment along with advances in the surgical practice and equipment's have resulted in increased number of surgeries for removal of impacted teeth.

Surgical extraction of impacted third molars is the most common procedure performed by the oral surgeon. Any tooth including the deciduous and the permanent can be impacted but the more common are the maxillary and mandibular third molars and the maxillary cuspids followed by the premolars and the supernumerary teeth[2]. Among the third molar impactions the most common is the mandibular. Maxillary third molar impactions being equally present but have been overlooked. Still rare are literature about inverted impacted maxillary third molars. Here we present a case report with review of literature.

Case report

A 50 year female patient reported to the OPD of Mar Baselios Dental college with complaints of pain with respect to upper right back tooth. A history of pain since 1 week and decay since 6 months were elicited. On examination a grossly decayed upper right maxillary first molar was

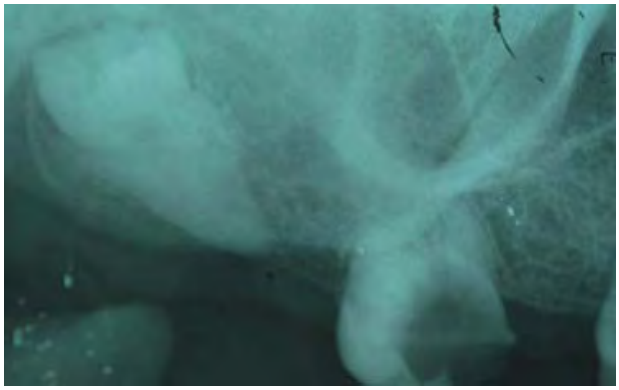

Figure - 1

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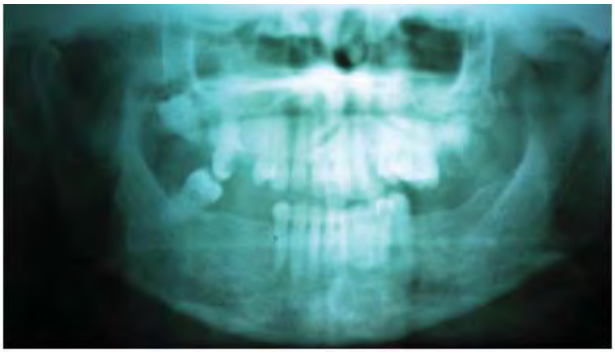
noticed which was tender on percussion. The upper right maxillary second and third molars were missing. A routine IOPA was taken which confirmed the clinical diagnosis of apical periodontitis of upper right maxillary first molar. In addition the IOPA revealed an inverted impacted upper right maxillary third molar.

A panoramic radiograph was taken which confirmed the presence of the inverted impacted right maxillary third molar, with minimum radiolucency around the crown and close proximity to the maxillary sinus.



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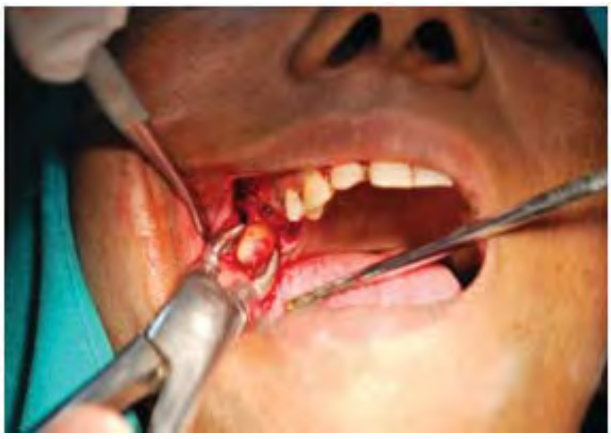

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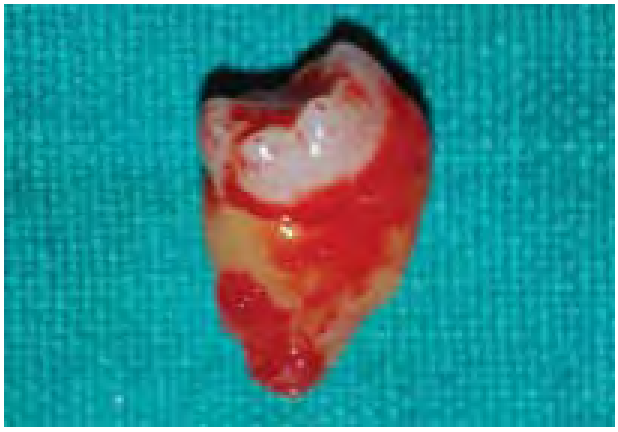

Figure - 4

The patient was informed of the presence of the impacted tooth and pros and cons of surgical removal versus the conservative management were discussed. The patient opted for the surgical removal of the inverted impacted tooth and the same was done along with the removal of the decayed maxillary right first molar under local anaesthesia.

Discussion

Inverted impacted maxillary molar is a rare entity. The first case was reported by Gold and Demby in 1973[3], followed by this another 12 cases of inverted impacted maxillary molars have been reported so far[4-12]. An inverted impacted tooth is called a complicated impaction[5]. Third molars in both the mandible and maxilla may develop at a distance from their normal location because of unusual proliferation of odontogenic epithelium before development of tooth germ thus leading these to get impacted[4]. Indications for surgical removal of impacted teeth include dental caries, recurrent pericoronitis, periodontal disease involving the adjacent tooth, obstruction to eruption of adjacent tooth, resorption of adjacent tooth, to facilitate orthognathic surgery, if related to any pathology, prosthetic considerations (prior to denture construction or implant placement) and to eliminate any potential sources of infection prior to radiotherapy. Contraindications include advanced age, excessive risk of damage to adjacent structures and asymptomatic deeply impacted third molars.

Archer in 1975[1] classified impaction of maxillary third molars as mesioangular, distoangular, vertical, horizontal, buccoangular, linguoangular and inverted. According to depth of impaction compared to the second molar Archer classified maxillary impactions into three categories:

Class A - Occlusal surface of impacted tooth is at approximately the same level as the occlusal surface of second molar.

Class B - Occlusal surface of impacted tooth is at the middle of the crown of the adjacent second molar.

Class C- Occlusal surface of impacted tooth is apical to the cervical line of the adjacent second molar or even deeper.

The factors that increases the difficulty of surgical removal of impacted maxillary third molars are surgical removal performed with mouth opening greater than 45mm, occlusal level is high, elevator tip is high, relation to maxillary sinus is positive (vs patients with no contact) and close relation to root of second molar[13] (vs patients in whom there is no contact or contact only with crown of second molars).

The treatment options for inverted impacted tooth include conservative management with routine radiographic follow up, autotransplantation and surgical removal. In our case an option of surgical removal of impacted inverted third molar along with extraction of grossly decayed upper right first maxillary molar was given and the patient opted for the same.Literature search shows no exact treatment protocols for removing inverted impacted maxillary molars so the most conservative management is considered to be the best protocol.

Complications during removal of inverted impacted maxillary third molars include a significantly large post extraction socket, risk of tooth displacement into infratemporal space or into the maxillary sinus, fracture of maxillary tuberosity and bleeding[14].

Conclusion

As a conclusion inverted impacted third molars can be surgically extracted but a conservative approach is more appropriate considering the complication that is foreseen.

References

1. Archer William H. Oral and Maxillofacial Surgery, Vol.1, edn.5, Philadelphia, WB Saunders Co.1975.
2. R Rajendran, B Sivapathasundrharam (2006) Shafer's textbook of oral pathology, 5thedn. Elsevier, pp 83-86.
3. Gold J, Demby N (1973) Rare inverted maxillary third molar impaction: report of case. J Am Dent Assoc 87: 186-188.
4. Held HW (1979) Inverted maxillary molar. Dent RadiogrPhotogr 52: 87. 10.
5. AlShamrani SM (2001) Inverted and impacted maxillary third molar. Report of two cases. Odontostomatol Trop 24: 15-17.
6. Pai V, Kundabala M, Sequeira PS, Rao A (2008) Inverted and impacted maxillary and mandibular 3rd molars; a very rare case. Oral Health Comm Dent 2: 8-9.
7. Yuvaraj, Agarwal GD (2011) Inverted maxillary third molar impaction – A case report. People's J Sci Res 4: 57-58.
8. Mohan S, Kankariya H, Fauzdar S (2012) Impacted inverted teeth with their possible treatment protocols. J Maxillofac Oral Surg 11: 455-457.
9. Togoo RA (2013) Rare occurrence of inverted maxillary third molar impaction: a case report. J Int Oral Health 5: 85-87.
10. Rammal M, Alfonso C (2013) An unusual case of an inverted and impacted maxillary third molar. Oral Surg(in press).
11. Chhabra S, Chhabra N, Dhillon N (2012) Inverted and impacted maxillary third molar - Removal by lateral transposition method. Int J Dent Exper Dent Sci 1: 26-29.
12. Chen CY, Wang WC, Lin LM, Chen YK (2014) Incidental detection of a rare inverted and impacted maxillary third molar in a patient of mandibular unicysticameloblastoma. Dentistry 4: 213.
13. Carvalho, De AraujoFilho, Do Egito Vasconcelos (2013) Assessment of factors associated with surgical difficulty during removal of impacted maxillary third molars. J Oral MaxillofacSurg 71: 839-845.
14. Togoo R A (2013) Rare occurrence of inverted maxillary third molar impaction: A case report. J Int Oral Health 5(3):85-87.

Source of Support	:	Nil
Conflict of Interest	:	Nil

CASE REPORT

Clinical and surgical treatment dilemma of ankyloglossia: A case report with review of literature

Varun Menon[1], Surej Kumar LK[2], Rakesh Koshy Zacariah[3], Sherin.A.Khalam[4].

Abstract:

Ankyloglossia, or tongue-tie, refers to an abnormally short lingual frenulum. It is a well recognized but poorly defined condition and has been reported to cause difficulties in feeding, dysarthria, dyspnea, and social or mechanical problems. Questions pertaining to the age, appropriate time of treatment and the most optimal method to treat are still a dilemma to the clinicians. It has been reported to cause feeding difficulties, dysarthria, dyspnea, and social or mechanical problems, which may warrant surgical correction in symptomatic cases. The literature regarding treatment remains inconclusive. In this article we report a case of 18 year old girl with tongue-tie who underwent the frenectomy procedure under local anaesthesia, along with review of literature.

Keywords : Ankyloglossia, tongue, surgical treatment.

Introduction

Ankyloglossia, or tongue tie, is an uncommon congenital anomaly characterized by an abnormally short lingual frenum which may restrict tongue-tip mobility and may subsequently lead to a range of problems such as difficulties in breast feeding during infancy, inability to chew age-appropriate solid foods, speech impediments, poor oral hygiene and behavioural problems[1]. Ankyloglossia has been defined as the condition in which the tongue cannot make contact with the hard palate or cannot protrude more than 1–2 mm past the mandibular incisors[2]. According to Lalakea and Messner incidence of ankyloglossia reported vary from 2 to 4.8% and were observed to occur more commonly in males with a male to female ratio of 3 to 1 with no racial predilection[3].The reported prevalence varies from 0.02 to 4.8%[4].

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Case Report:

An 18year female patient reported with complaints of difficulty in protruding the tongue and to approximate the tip of the tongue to the palate .She also finds difficulty in speaking certain letters like t; d etc.She had a history of previous surgical procedure done for correction of same difficulty during childhood. Morphologically, lingual frenum was abnormally short and thick and functionally; the tongue was unable to protrude past the gum line and could not contact the palatal vault (Figure 1a). Even lateral movements of tongue were

Figures and Legends

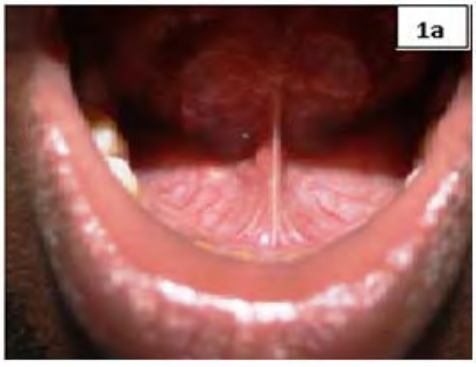

Figure 1a: Ankyloglosia showing short and thick lingual frenum with restricted tongue movement

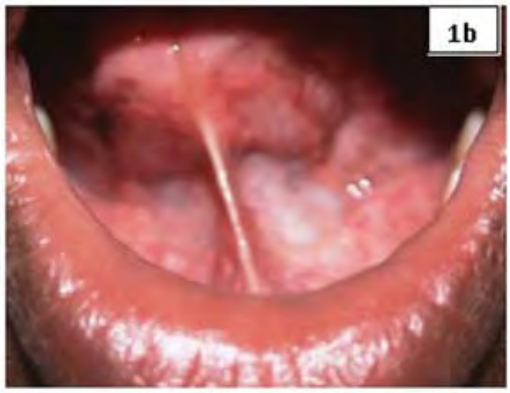

Figure 1b: Lingual frenum with limited lateral movement

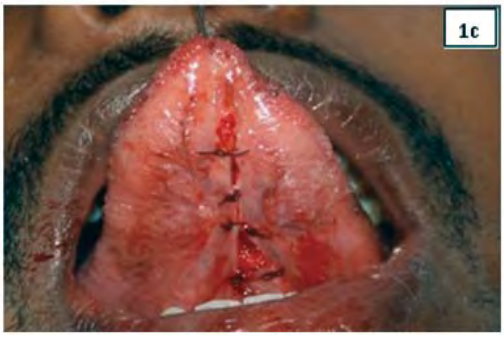

Figure 1c: Surgical correction and closure of Ankyloglosia

mildly restricted (Figure 1b). The surgical correction was performed sequentially, taking into consideration the vital anatomical structures present in the vicinity ,a vertical incision was placed along the lingual frenum.Fibrotic bands excised, improved mobility of the organ was visible immediately after post intervention . Following total release, closure was done with 3, 0 vicryl (Figure 1c). Haemostasis was achieved.

Discussion

Ankyloglossia, or tongue-tie, refers to an abnormally short lingual frenulum. Ankyloglossia is derived from the Greek words 'ankylo' meaning stiff and 'glossa' meaning tongue. The exact pathophysiology of tongue-tie is unknown and the most likely cause of abnormal frenulum length and attachment is the deviation from normal development. The clinical finding of ankyloglossia has been associated several syndromes, including Ehlers-Danlos syndrome, Beckwith-Wiedemann syndrome, Simosa syndrome, X linked cleft palate, orofaciodigital syndrome, and several others[5, 6].

In the early 1900s, tongue-tie was believed to impact breastfeeding and was routinely divided. As formula milk gained popularity, tongue-tie release fell out of favor as infants could bottle feed as an alternative[7]. History reveals treatment of ankyloglossia dates back to the New Testament: "one...had an impediment in his speech...the string of his tongue was loosed, and he spake plain." (Mark 7:32) and midwives ,in the 18th century would use their fingernails to divide the frenulum.Since then a variety of treatment options, including simple division, frenulotomies, and four-ap Z-frenuloplasty have been described in literature[2].

Kotlow LA[8] ,from his clinical research gave a comprehensive classification to describe the severity of ankyloglossia

1. Clinically acceptable, normal range of free tongue: greater than 16 mm
2. Class I: Mild ankyloglossia: 12 to 16 mm
3. Class II: Moderate ankyloglossia: 8 to 11mm
4. Class III: Severe ankyloglossia: 3 to 7 mm
5. Class IV: Complete ankyloglossia: less than 3 mm

According to American Academy of Paediatrics[9] different grading system for tongue tie: Grade 1: is the attachment of the lingual frenulum to the tip of the tongue, in front of the alveolar ridge in the lower lip sulcus.

Grade 2**:** is 2-4mm behind the tongue tip and attaches on or just behind the alveolar ridge.

Grade 3: is the attachment of the lingual frenulum to the midtongue and the middle of the floor of the mouth and is usually tighter and less elastic.

Grade 4: is essentially attached against the base of the tongue and is thick, shiny and inelastic.

The Hazelbaker Assessment Tool for Lingual Frenulum Function [10] (HATLFF) was developed to provide a quantitative assessment of ankyloglossia and has been proven to be highly reliable. It includes five appearance items, such as length, attachment site, and elasticity, as well as seven functional items, such as extension, spread, cupping, and peristalsis of the tongue[4],[10,11].

Ankyloglossia can be diagnosed using the following criteria: impossibility of touching palate with tongue tip when the mouth is open; bifid tongue during protrusion; curvature of the intermediate part of the tongue, preventing it from moving forward out of the oral cavity;

and reduced sublingual space. The treatment options for ankyloglossia involve observation, language articulation therapy and three possible surgical techniques: frenotomy, which consists of simply cutting the frenulum; frenectomy, complete excision of the frenulum; and frenuloplasty, which involves freeing the tongue and correcting its anatomy[12,13,14]. Depending on the consequences of the abnormality, radical and urgent treatment may be required.

Berg[15] reported that frenectomy is normally performed in 1–3-year old children, under general anaesthesia, in most cases. It should be performed as soon as possible, because if performed later, the child might have incorrect swallowing and disturbed speech muscle movement patterns. There are no conclusive parameters about the age for performing the surgical technique in the literature[16].

Conclusion

Though Clinical and surgical treatment dilemma of Ankyloglossia still exist and it varies from clinicians to clinicians but from our experience, we feel timely and appropriate surgical intervention, followed by speech therapy when indicated is mandatory for optimal management of tongue tie; It is being more and more accepted across by all clinicians associated with infants, children and adults with tongue tie that , early intervention is the optimal form of management when the frenum has been identified and diagnosed as abnormal.

Reference

1. Lalakea M.L., Messner A.H. Ankyloglossia: Does it matter? Pediatric Clin North Am 2003;50:381-97.
2. Heller.J,Gabbay J, O'Hara .C, et al., Improved ankyloglossia correction with four-ap Z frenuloplasty, Ann. Plast. Surg 2005;54: 623–628.
3. Messner A.H., Lalakea M.L.Ankyloglossia: Controversies in management. Int J Pediatr Otorhinolaryngol 2000; 54:123-31.
4. Amir LH, James JP, Donath SM. Reliability of the Hazelbaker Assessment Tool for lingual frenulum function. Int Breastfeed J 2006; 1:3–8
5. Kummer,A: Ankyloglossia: to clip or not to clip? that's the question. ASHA Leader, 2005; 10(17):6,7-30.
6. De Felice C, Toti P, Di Maggio G etal. Absence of the inferior labial and lingual frenula in Ehlers-Danlos syndrome. *Lancet*. 2001;357(9267):1500–1502.
7. Wallace H, Clarke S. Tongue tie division in infants with breast feeding difficulties. Int J Pediatr Otorhinolaryngol. 2006;70(7):1257–1261
8. Kotlow L.A. Ankyloglossia (tongue-tie): A diagnostic and treatment quandary. Quintessence Int 1999; 30:259-62
9. *Shafer* WG, *Hine MK*, Levi *BM*. Developmental disturbances of oral and para oral structures. In: Oral Pathology. 4th ed. Philadelphia, Pa: Harcourt; 2000
10. Hazelbaker A. The Assessment Tool for Lingual Frenulum Function (ATLFF): Use in a Lactation Consultant Private Practice. Pasadena, CA: Pacific Oaks College; 1993
11. Hogan M, Westcott C, Griffiths M. Randomized, controlled trial of division of tongue-tie in infants with feeding problems. J Paediatr Child Health.2005;41(5–6):246–250
12. Cunha RF, Silva JZ, Faria MD. Clinical approach of ankyloglossia in babies:report of two cases. J Clin Pediatr Dent 2008: 32: 277–281.
13. Kupietzky A, Botzer E. Ankyloglossia in the infant and young child: clinical suggestions for diagnosis and management. Pediatr Dent 2005: 27: 40–46.
14. Suter VG, Bornstein MM. Ankyloglossia: facts and mytis in diagnosis and treatment. J Periodontol 2009: 80:1204–1219.
15. Berg KL. Two cases of tongue-tie and breastfeeding. J Hum Lact 1990: 6: 109–112.
16. Segal Lm. Stephenson R, Dawes M, Feldman P. Prevalence, diagnosis, and treatment of ankyloglossia. Can Fam Physician 2007: 53: 1027–1033.

Source of Support	:	Nil
Conflict of Interest	:	Nil

CASE REPORT

Pyogenic granuloma in a one and half year old child: A case report

Vadi V. Bharatham[1], Raj Gopal Singh Mehta [2], Reshma V J[3], Shihab Anwar A[4]

Abstract

Oral pyogenic granuloma (OPG) is defined as kind of inflammatory hyperplasias on any kind of soft tissues of the oral cavity. It is very similar to pyogenic granuloma that occurs on the other part of the body. The term pyogenic granuloma is a misnomer because the lesion is unrelated to infection and in reality arises in response to various stimuli such as low grade local irritation traumatic injury or hormonal factors. This lesions is predominantly seen in younger individuals, specially in the females and around the second decade of life. The reason could be possibly because of the high vascularity and due to changes in the hormones in these individuals. Clinically, the oral pyogenic granuloma is a smooth or lobulated exophytic lesion manifesting as small red erythematous papules on a pedunculated or sometimes sessile base which is usually hemorrhagic. The surface ranges from pink to red to purple depending on the age of the lesion. Excisional surgery has the treatment of choice although some other treatment protocols such as the use of ND YAG laser, flash lamp, pulsed dye laser, cryosurgery, intralesional injection of ethanol or corticosteroid and sodium tetradecyl sulfate, sclerotherapy have been proposed.

Keywords : Pyogenic granuloma, child.

Introduction

OPG is a kind of inflammatory hyperplasia. The term inflammatory hyperplasia is used to describe a large range of nodular growths of the oral mucosa that histologically represents inflamed fibrous and granulation tissues. It includes fibrous inflammatory hyperplasia palatal papillary hyperplasia, giant cell granuloma and pregnancy epulis.

The term Pyogenic granuloma was first introduced by Hartzell in 1904 although Hullihen was the first person to describe the lesion in 1844 for the first time in the English literature.

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Although it is a common disease in the skin, it is extremely rare in the gastrointestinal tract except for the oral cavity where it is often found on keratized tissue. There are two kinds of PG namely the lobular capillary hemangioma and non lobular capillary hemangioma which differs in their histological appearances.

Because of the high incidence of oral PG especially in pregnant women and the critical need for its proper diagnosis management and treatment, it is important to recognize this problem and deal with it suitably.

The etiology for oral pyogenic granuloma is unknown. Some investigators believe it to be a kind of benign neoplasm. It is also a reactive-tumor like lesion which arises in response to various stimuli such as a low grade local irritation, traumatic injury, hormonal factors or certain kinds of drugs.

Oral pyogenic granuloma is a smooth or lobulated exophytic lesion manifesting as a small red erythematous papule or a pedunculated or sometimes sessile base which is usually hemorrhagic and compressible and may develop as dumb bell shaped masses. The size varies in diameter from a few millimeters to several centimeters and usually reaches its full size within weeks or months remaining inefinitely thereafter. Clinically the development of the lesion is slow asymptomatic and painless but it may also grow rapidly. The surface is characteristically ulcerated and friable which may be covered by a yellow fibrinous membrane and its color ranges from pink to red to purple depending on the age of the lesion. Young oral pyogenic granulomas are highly vascular in appearance because they are composed of predominantly of hyperplastic granulation tissue in which capillaries are prominent. Minor trauma to the lesion may cause considerable bleeding due to its pronounced vascularity where as older lesions tend to become more collagenized and pink.

Although, it has been reported even in children, it is a rare finding in the children. Here we report such a case of oral pyogenic granuloma in a one-and-half year old child in the upper maxillary gingiva.

Case report

An 15-month-old male child patient reported to the department of oral medicine and radiology at MES Dental college and Hospital with a chief complaint of bleeding from swelling on the gums since 15 days.

History of presenting illness revealed that the patient was apparently normal about a month days ago when he had a fall. The upper front tooth was broken in the in the event with a laceration of the upper lip. The patients' parents rushed the child patient to a child health care provider where he was subsequently treated for the broken tooth and laceration of the upper lip with antibiotics and analgesics. About 15 days ago the patients' parents noticed a growth on the upper gums in the place of the broken tooth. The swelling was bright red in color and would easily bleed during feeding. The child patient was then subsequently referred to this hospital for further diagnosis and management.

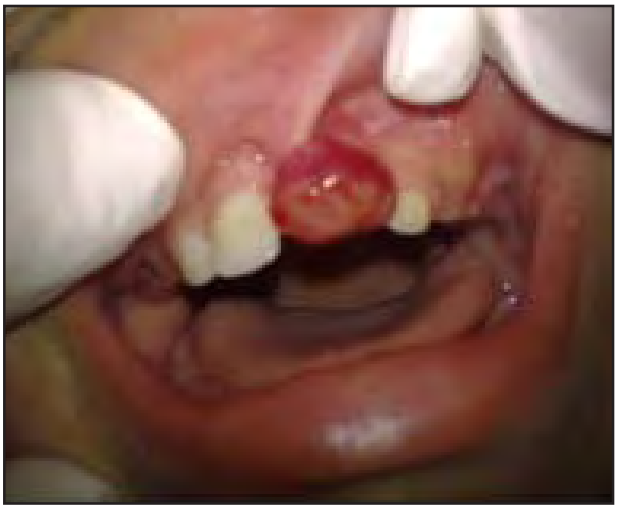

Figure-1 Clinical photograph showing he gingival growth

Clinical examination revealed that the patient had gingival overgrowth in relation to 61 Description of the lesion: a solitary reddish peduculated growth, measuring roughly about 1.5 cm in diameter, round to ovoid in shape was present on the upper labial gingival in relation to 61. the growth was smooth with no surface ulceration, discharge, or bleeding. On palpation, all the inspectory findings were confirmed. The swelling was soft in consistency, attached to the maxiallary labial gingival with a pedicle and immobile. The growth bled easily with manipulation.

The remaining hard tissue such as the decidous teeth were intact with no significant pathological findings. Similarly, other soft tissue such as tongue buccal mucosa, vestibule, labial mucosa, tongue had no noticeable change or abnormality.

Based on chief complaint, history and clinical examination a working diagnosis of oral pyogenic granuloma was given.

DIFFERENTIAL DIAGNOSIS

Differential Diagnosis of Peripheral giant cell granuloma, Peripheral ossifying fibroma, Metastatic cancer, Hemangioma, Conventional granulation tissue, Kaposi's sarcoma, Angiosarcoma and hon hodgkins lymphoma was also considered

As part of the routine investigative procedure an Intra Oral Periapical Radiograph was advised in relation to 61 and an anterior maxillary occlusal view was taken.

The radiographs revealed no change in the bone morphology and indicating the lesion was arising from the soft tissue.

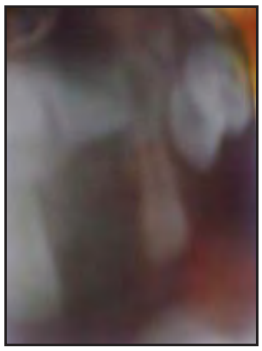

Figure - 2 Radiograph showing the missing 61

The patient was then referred to the Department of Pedodontics for further management where an excisional biopsy was done under local anesthesia and the specimen subsequently sent to the Oral pathology department for histopathological studies.

Biopsy report

Electron microscopy of the fixed lesion revealed parakeratized stratified squamous epithelium in focal areas and connective tissue stroma, loosely arranged collagen fibers numberous blood vessels filled with RBCs and inflammatory cells. Connective tissue stroma also exhibited moderate infiltration of mixed inflammatory cells like neutrophills lymphocytes plasma cells.

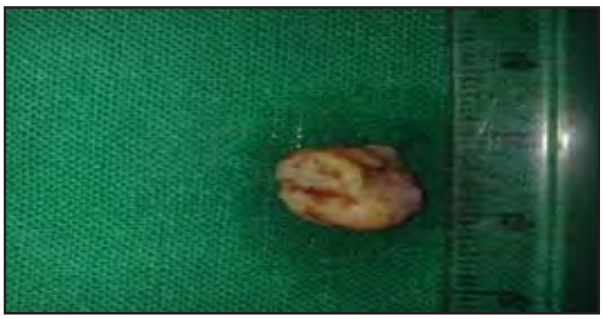

Figure - 3 Photograph of the excised specimen

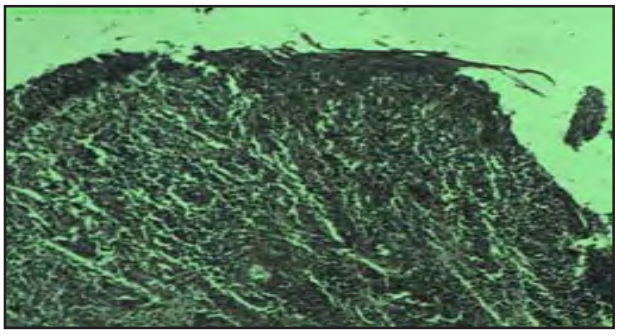

Figure - 4 photohistomicrograph of the oral pyogenic granuloma

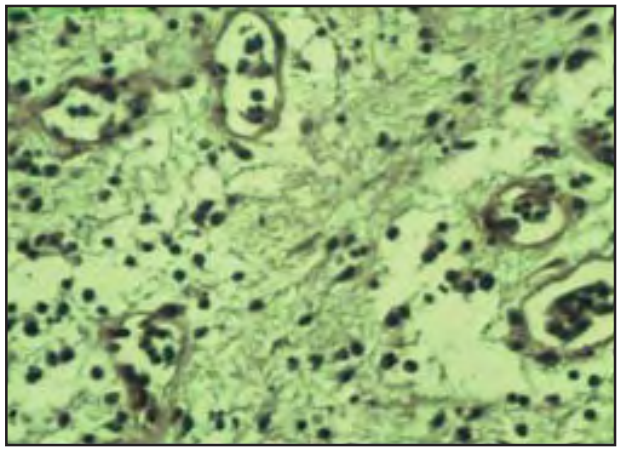

Figure - 5 photohistomicrograph of the oral pyogenic granuloma

Based on the histopathological findings a final diagnosis of Oral pyogenic granuloma was given.

The pyogenic granuloma also known as, granuloma pyogenicum, is a response of tissues to nonspecific infection of staphylllococci or streptococci.

Oral pyogenic granuloma is relatively common and accounts for 3 to 7 % of all biopsy findings fromm oral cavity lesions. In study the most common intra oral site was the gingival followed by the lips tongue palate and buccal mucosa. Oral pyogenic granuloma on gingiva is more common in the maxilla than in the mandible. Oral pyogenic granuloma if on the gingival tissue is present is more common in the anterior region than in the postherior region

Oral pyogenic granuloma is a non neoplastic growth in the oral cavity proper most commonly seen in young individuals, especially the females. Although its occurrence in children and infants is rare, many cases have, however, been reported previously. Since it is seen pretty commonly in pregnant women and young individuals, its diagnosis prevention, management, and treatment of the lesion are very important. The exact etiology for this lesion is unknown although it is seen arising in response to various stimuli such as low grade local irritation, traumatic injury, sex hormones or certain kinds of drugs. Hence , our primary objective should be aimed at removal of causative irritants such as plaque calculus foreign materials and source of trauma if such is the cause followed by treatment by excisional surgery. New modalities of treatment

include cryosurgery excision by ND YAG laser, flash lamp, pulsed dye laser, injection of ethanol or corticosteroid and sodium tetradecyl sulfate. In spite of these treatments, recurrence is not infrequent. So, in some case re-excision may be necessary. It should be emphasized that one of the most important point about pyogenic granuloma is the effect of sex hormonal imbalances during pregnancy and the body's over zealous response to irritants. Although it is rarely seen in children, it is some times seen in these individuals also

References

1. Ramirez. K, Bruce G: carpenter. pyogenic granuloma: case report in a 9-year old girl. General Dentistry 2002, 50(3):280-1.
2. Nevile BW, Damm DD, Allen CM, Bouquot JE: oral and maxillofacial pathology. Second edition. W.B. saunders co; 2004:444-449.
3. Svirsky J: Oral pyogenic Granuloma.[http://emedicine.medscape.com/article/1077040-overview] webcite 2007. (10 pages), web med Accessed Mar 10, 2007
4. karthikeya Patil, mahima VG, Lahari K: Exragingival pyogenic granuloma. Indian journal of dental research 2006, 17(4):199-202.
5. Regezi JA, sciubba , James J, Jordan Richors CK: Oral Pathology, clinical pathologic correlation. Fourth edition. Sanders Company; 2003:115-76.
6. Akyol MU, Yalciner EG, Dolan AI: Pyogenic granuloma (lobular capillary hemangioma) of the tongue. Int J Pediatr Otorhinolaryngol 2001, 58(3):239-241.
7. Glorgi VD, sestini S, Nardini p, cali p: A 42-year – old man with a rapidly growing lesion of the soft palate. CMAJ 2005, 173(4):367.
8. Greenberg MS, Glick M: Burket's oral medicine Diagnosis & treatment. Tenth edition. B C Decker inc; 2003:141-142.
9. Cawson RA, Odell EW: Cawson's essentials of oral pathology and medicine. 7th edition. Churchill Livingston; 2002:277
10. Mueller BU, Mulliken JB. The infant with a vascular tumor. Semin Perinatol. 1999;23:332-340.
11. Mills SE, Cooper PH, Fechner RE: Lobular capillary hemangioma: the underlying lesion of pyogenic granuloma: a study of 73 cases from the oral and nasal mucous membranes. Am J Surg Pathol. 1980;4:470-479.
12. Isaacs H Jr. Tumors of the Fetus and Newborn. Vol 35. Philadelphia, Pa: WB Saunders Co; 1997.
13. Dilley DC, Siegel MA, Budnick S. Diagnosing and treating common oral pathologies. Pediatr Clin North Am.1991;38:1227-1264.
14. Sato M, Tanaka N, Sato T, Amagasa T. Oral and maxillofacial tumours in children: a review. Br J Oral Maxillofac Surg. 1997;35:92-95.
15. Kershisnik M, Batsakis JG, MacKay B. Pathology consultation: granular cell tumors. Ann Otol Rhinol Laryngol. 1994;103:416-419.

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CASE REPORT

The myth and reality of " The Free Fibula"

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Abstract

Segmental resection of the mandible may cause considerable functional, esthetic, and social problems. Immediate reconstruction of the mandible is, therefore, preferred, with the aim to achieve restoration of function and esthetics. Over the past decade, the use of reconstructive plates alone for the reconstruction of large mandible defects has essentially been replaced by the use of osseous microsurgical free flaps, of which the free fibula flap has emerged as the primary choice among surgeons. The free fibula flap has improved the functional and cosmetic outcomes after reconstruction of extensive mandible defects. I present a case of free fibula microvascular flap transfer for reconstruction of the mandible.

Keywords : Free Fibula flap, mandibular defects.

Introduction

Over the past few decades, the use of reconstructive plates alone for the reconstruction of large mandibular defects has almost completely been replaced by osseous microsurgical free flaps, such as the fibula flap, iliac crest, and scapula free flap. Such reconstructive options have improved the functional and cosmetic outcomes after reconstruction of extensive mandibular defects.
In 1973, the fibula transfer was developed simultaneously by O'Brien[1] in Australia and Ueba[2] in Japan and was first published by Taylor et al [3] from Melbourne in 1975 & Gilbert[4] in 1979. The osteocutaneous tissue transfer from the calf region was first described by Chen & Yan[5] in 1983. Hidalgo[6] popularised the technique and the flap came into widespread use for mandible microsurgical reconstruction. Since then, several refinements for flap harvesting and insetting has been described for meeting better aesthetic and functional demands.

Free fibula graft allows for adequate prosthetic rehabilitation of the patient through implants and thus has greatly enhanced masticatory and speech functions of patients with mandibular defects. This flap has thus emerged as the workhorse for microsurgical mandibular reconstruction[7,8].

Case report

We report a case of reconstruction of a segmental defect of the mandibular body using free fibula flap. The patient reported with a swelling on the right mandibular body which was investigated and diagnosed to be ameloblastoma. The patient underwent segmental resection of the right mandibular body and reconstruction with a plate at our centre. Due to certain technical constraints we were not able to rehabilitate with a free flap at the time. Three years later the patient reported with pain and swelling, which was investigated to reveal a fractured reconstruction plate. We then decided on rehabilitation with a free fibula flap. A free

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fibula transfer was planned and executed and adequate results were achieved using the procedure.

The prosthetic rehabilitation of the patient is being planned currently.

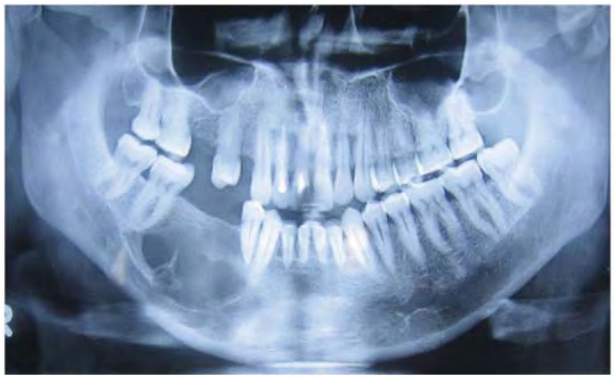

Figure - 1 OPG revealing the initial lesion - ameloblastoma of right mandibular body

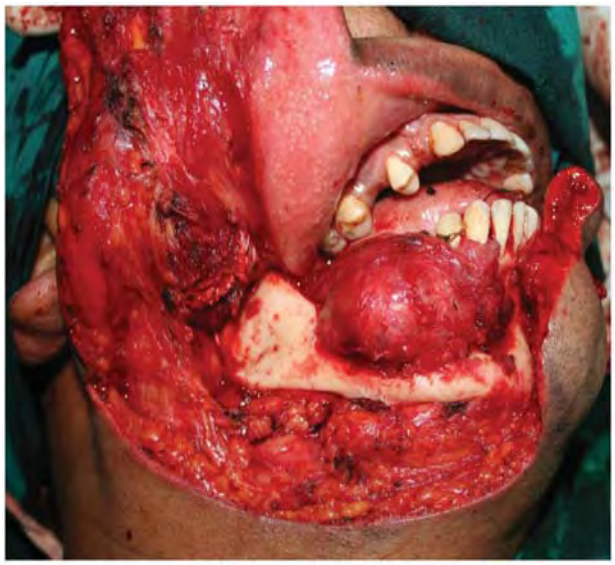

Figure - 2 Intraoperative view of the lesion

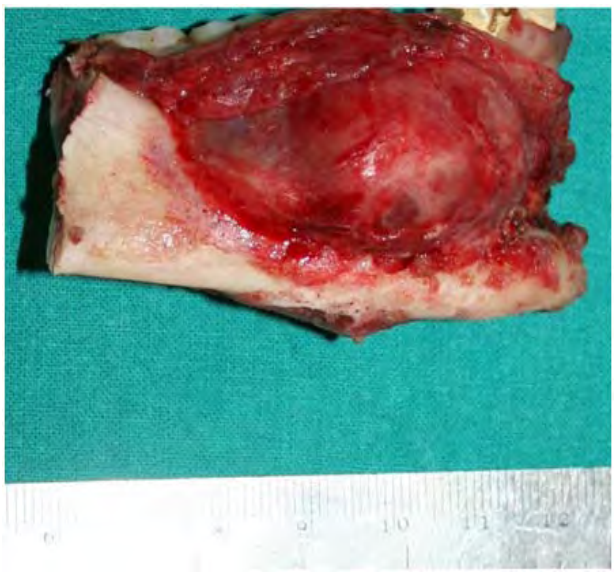

Figure - 3 Segmental resection of the mandibular body done

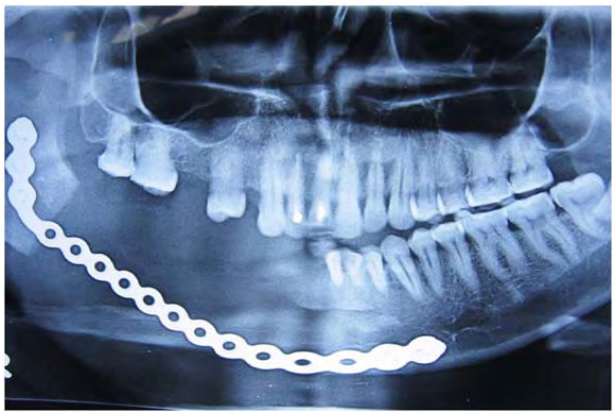

Figure - 4 Post operative OPG showing reconstruction plate in place

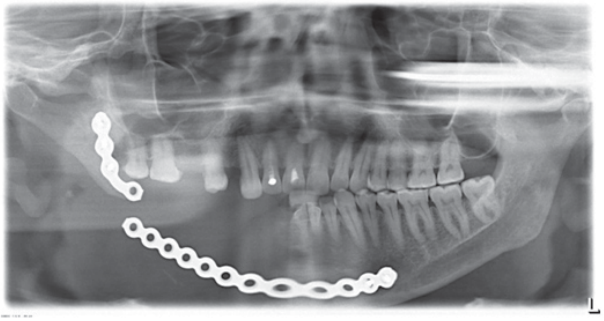

Figure - 5 OPG showing fractured reconstruction plate

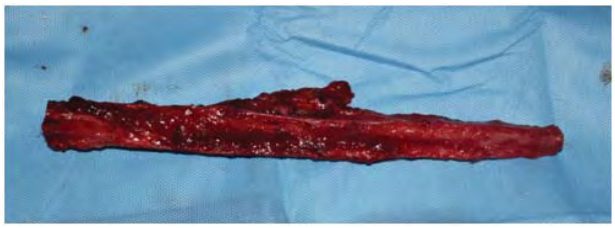

Figure - 6 Harvested Free fibula free flap

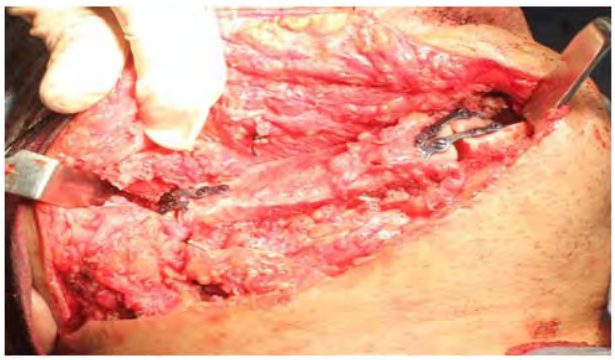

Figure - 7 Free fibula fixed with plates

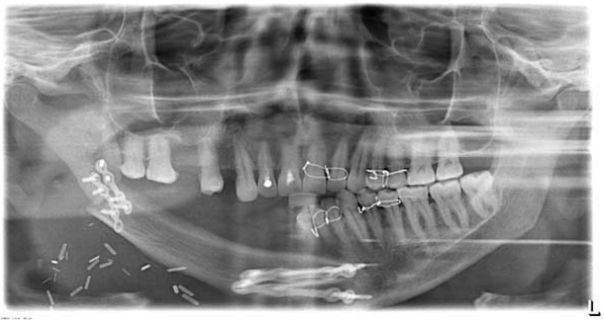

Figure - 8 OPG showing flap in place

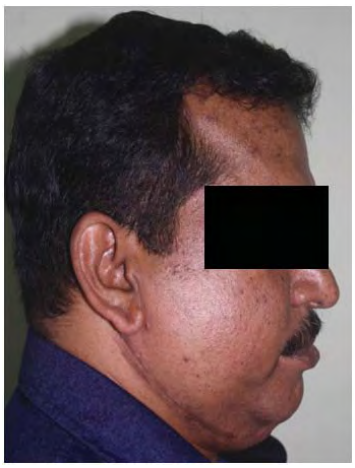

Figure - 9 One month postoperative review

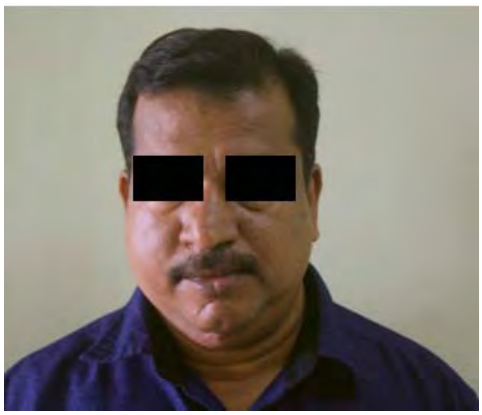

Figure - 10 One month postoperative review

Discussion

There are wide varieties of mandibular defects and no single type of free flap is capable of meeting the demands of all the defects. The choice of flap depends on several factors including the type of bone and soft tissue (skin and/or oral) defect, vessel geometry of the donor and recipient sites, donor site morbidity, whether the patient is dentate or edentulous, need for dental implant rehabilitation, and often the type of training and individual preference of the surgical team[9].

The fibula free flap remains the first choice for the edentulous mandibles or for extensive mandibular resections [9]. Blood supply to the fibula is both intraosseous and segmental which allows multiple osteotomies to be made. This free flap can provide up to 30cm of bone length which is sufficient for reconstructing any length of the mandible. The height of bone often becomes inadequate relative to a dentate mandible which may become a problem when dental implants are planned for prosthetic rehabilitation. This problem may be overcome by the use of a 'double barrel' flap[10] or a vertical distraction of the fibular flap[11]. Donor site morbidity often is relatively mild but pre-existing peripheral vascular disease may preclude use of this flap.

Ghara et al[12] have summarized, the potential advantages of the fibula flap, as follows:

1. Straight shape and high mechanical resistance to pressure and torsion
2. Rapid incorporation and healing of the highly vital flap due to the excellent perfusion,
3. Composition - with a high content of cortical bone,
4. Great length, which allows the bridging of large defects,
5. Possibility of osteotomizing it at various points allowing adaptation
6. Relatively simple harvesting of the flap with conveniently sized blood vessels for anastomoses
7. Low morbidity of the donor region
8. Can be elevated with skin and muscle as an osteomyocutaneous flap Ferri etal[13] have listed several limitations in using the fibula flap. When bone modelling is required, and achieved by one or more osteotomies, there is destruction of the centromedullary fibular pedicle. Although many authors consider periosteal vascularisation adequate, Ferri etal found that a large number of osteotomies of the fibular diaphysis led to reduced vitality of distal fragments, especially in cases of small fragments. This risk is greater the smaller the fragment. For this reason, we attempt to limit the number of osteotomies of the fibula to a maximum of two. This problem is not seen with the other bone flaps, which can be modelled without risk. Aesthetic sequele can sometimes be important, especially in women. A scar may run along the entire length of the lateral part of the leg, which many may consider as unsightly.

The fibula flap procedure is not be used in patients with ischemic disease involving the lower limbs. In patients with arteritis, the fibular artery is often the lone remaining permeable vascularising

vessel in the lower limb. Arteriosclerotic disease involving the fibular artery does not allow for good vascularisation and can lead to even failure of the flap. .

Another limitation of the fibula flap is the small length of its pedicle, which makes it difficult to perform anastomoses. When the entire fibula is removed, no more than 5 cm is available, compared with 10 cm for iliac crest, lateral brachialis, and parascapular flaps. When a considerable quantity of soft tissue is to be reconstructed, the skin paddle of the fibula flap often becomes inadequate because of its limited thickness.

For our case, careful treatment planning and methodical execution as per planned was adhered to, throughout the procedure. We did not wish to compromise the vasculature by osteotomising the fibula for use of a double barrel, as adequate results were achieved by the procedure alone. At the time of preparation of this article, prosthetic rehabilitation of the patient is being planned, which will thus add to the benefit the patient, functionally.

Conclusion

The free fibula flap does have its advantages and limitations, but the fact that, it is one of the most versatile and reliable option for microsurgical reconstruction of large mandibular defects; is without question. It provides a large quantity of bone, which can be shaped easily to adapt to the remaining mandible passively. The bone height is adequate for implant-based prosthetic rehabilitation. Preoperative mapping of the cutaneous perforators help in improving the versatility of the flap design, thereby decreasing the donor site morbidity.

While opting for free flaps, one should bear in mind that most patients with head and neck tumours often have medical co morbidities. A proper and critical preoperative assessment of the risk of postoperative complications is essential before such patients are selected for extensive oncological and reconstructive surgery. The free fibula is the ideal tool for a good surgeon wanting to put a smile back onto the faces of his patients.

References

1. O'Brien BMcC, Morrison WA. Reconstructive microsurgery. New York: Churchill Livingstone, 1987
2. Ueba Y, Fujikawa S; Nine years follow up of a free vascularised fibula graft in neurofibromatosis -a case report and literature review. J Orthop Trauma Surg 1983: 26: 595-600.
3. Taylor GI, Miller DH, Ham FJ: The free vascularised bone graft: A clinical extension of microvascular techniques. Plast Reconstr Surg 55:533, 1975
4. Gilbert A: Vascularised transfer of the fibular shaft. Int J Microsurg 1:100, 1979
5. Chen ZW, Yan W The study and clinical application of the osteocutaneous flap of fibula. Microsurgery 1983: 4: 11-6.
6. Hidalgo A: Fibula free flap: A new method of mandible reconstruction. Plast Reconstr Surg 84:71, 1989
7. P. Sieg, A. Hasse, C E. Zimmermann. Versatility of vascularised fibula and soft tissue graft in the reconstruction of the mandibulofacial region. Int. J. Oral Maxillofac. Surg. 1999; 28." 356-361.
8. López-Arcas JM, Arias J, Del Castillo JL, Burgueño M, Navarro I, Morán MJ, Chamorro M, Martorell V. The Fibula Osteomyocutaneous Flap for Mandible Reconstruction: A 15-Year Experience. J Oral Maxillofac Surg 68:2377-2384, 2010
9. TinGoh B, Lee S,Tideman H, Stoelinga PJW: Mandibular reconstruction in adults: a review. Int. J. Oral Maxillofac. Surg. 2008; 37: 597–605
10. Horiuchi K, Hattori A, Inada I, Kamibayashi T, Sugimura M, Yajima H, Tamai S. Mandibular reconstruction using the double barrel fibular graft. Microsurgery 1995: 16: 450–45.

11. Nocini PF, Wangerin K, Albanese M, Kretschmer W, Cortelazzi R. Vertical distraction of a free vascularised fibula flap in a reconstructed hemi mandible: a case report. J Craniomaxillofac Surg 2000: 28: 20–24.

12. Ghara A, Darwich K, Li L, et al: Long-term results of jaw reconstruction with microsurgical fibula grafts and dental implants. J Oral Maxillofac Surg 65:1005, 2007.

13. Advantages and Limitations of the Fibula Free Flap in Mandibular Reconstruction. Ferri J, Piot B, Ruhin B, Mercier J. J Oral Maxillofac Surg 55440-448, 1997.

Source of Support : Nil
Conflict of Interest : Nil

CASE REPORT

Emotional woes : A strain to the periodontium

Anju Samuel[1], Nithin Mathew Cherian[2]

Abstract

Stress poses as a health risk to all individuals in day today life. It affects the immune and inflammatory responses. They have got an impact on oral diseases, wound healing and alter the management of periodontal diseases.

Keywords : Stress, Perodontal disease

Birth of stress

Stress has become such an ingrained part of our vocabulary and daily existence, that it is difficult to believe that our current use of the term originated only a little more than 50 years ago, when it was essentially "coined" by Hans Selye. Selye observed that patients suffering from different diseases often exhibited identical signs and symptoms. They just "looked sick". This observation may have been the first step in his recognition of "stress"[1].

Psychological disturbances can lead patients to neglect oral hygiene with resultant unfavorable effects on the periodontal tissues. The association of stress with periodontal disease is difficult to prove as there are many factors influencing the incidence and severity of periodontal disease, some of which are assumed and have not been identified. Nevertheless, more recent studies indicate that psychosocial stress represents a risk indicator for periodontal disease and should be addressed before and during the treatment[2].

Definition of stress and stressor

The term stress is used loosely, in layman terms, to describe adverse emotions or reactions to unpleasant experiences. However, the term stress has a precise physiological definition. It is "a state of physiological or psychological strain caused by adverse stimuli, physical, mental, or emotional, internal or external, that tend to disturb the functioning of an organism and which the organism naturally desires to avoid". Thus,stress can be viewed as a process with both psychological and physiological components[3].

A stressor is any stimulus, situation or circumstance with the potential to induce stress reactions[3].

Pathophysiology of the stress response

In response to a variety of stressful stimuli, an elegant sequence of events is initiated. Activation of the hypothalamic–pituitary–adrenal axis by stress results in the release of an increased concentration of corticotropin-releasing hormone from the hypothalamus. The pituitary gland is connected to the hypothalamus by the infundibulum, a stalk of tissue that contains nerve fibersand small blood vessels. Corticotropin-releasing hormone, in turn, acts on theanterior pituitary, resulting in the release of adrenocorticotropic hormone (corticotropin). The

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adrenocorticotropic hormone then acts on the adrenal cortex and causes the production and release of glucocorticoid hormones into the circulation. The glucocorticoids then produce a myriad of effects throughout the body, such as suppressing the inflammatory response, modifying cytokine profiles, elevating blood glucose levels, and altering levels of certain growth factors . Importantly, it has been established that proinflammatory cytokines, such as interleukin-1, can also activate the hypothalamic–pituitary–adrenal axis, leading to a feedback loop . Immune function plays a critical role early in the wound healing cascade. Pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor, are just two of the essential cytokines in this regard. It has been speculated that success in the later stages of healing is critically dependent on these early events[3].

The second major pathway to be activated is thesympathetic nervous system. A well-known exampleof this is the so-called flight or Figureht response to potentially harmful stimuli. Stress activates the nerve fibers of the autonomic nervous system, which innervate the tissues of the immune system. The adrenal medulla is actually a modified sympathetic ganglion. Its nerve bodies, instead of possessing axons, secrete their products directly into the bloodstream. The release of catecholamines results in the hormonal secretion of norepinephrine and epinephrine from the adrenal medulla, which results in a range of effects that may act to modulate immune responses. Catecholamines, released during stress, contribute to the development of hyperglycemia by directly stimulating glucose production and interfering with the tissue disposal of glucose . In addition, the sympathetic nervous system has a role in regulating immune cell activities[3].

Cases for Periodontal Destruction Due to Stress

Neglect of oral hygiene

It is obvious that proper oral hygiene is partially dependent on the mental health status of the patient. It has been reported that psychological disturbances can lead patients to neglect oral hygiene and that the resultant accumulation of plaque is detrimental to the periodontal tissue. Academic stress was reported as a risk factor for gingival inflammation with increasing crevicular interleukin-b levels and a diminution of quality of oral hygiene.

Changes in dietary intake

Emotional conditions are thought to modify dietary intake, thus indirectly affecting periodontal status. Psychological factors affect the choice of foods, the physical consistency of the diet, and the quantities of food eaten. This can involve, for instance, the consumption of excessive quantities of refined carbohydrates and softer diets requiring less vigorous mastication and therefore predisposing to plaque accumulation at the approximal risk site.

Smoking and other harmful habits

Circulating nicotine results in (i) vasoconstriction, produced by the release of adrenaline and noradrenaline, which is supposed to result in a lack of nutrients for the periodontal tissue; (ii) suppression of in vitro secondary antibody responses and (iii) inhibition of oral neutrophil function.

Gingival circulation

The tonus of the smooth muscle of blood vessels may be altered by the emotions by way of the autonomic nervous system. Furthermore, in long or continued emotions, a constant constriction of blood vessels could alter the supply of oxygen and nutrients to the tissues.

Alteration in salivary flow and components

It is assumed that both increase and decrease in salivary flow, induced by emotional disturbance, may affect the periodontium adversely. Emotional distress may also produce changes in saliva pH and chemical composition like IgA secretion. These relationships between salivary physiology and psychological status do not necessarily demonstrate causation of periodontal disease, but they show a pathway in which periodontal health is influenced by salivary changes.

Oral habits

Neurotic needs find oral expression. The mouth may be used to obtain satisfaction, to express dependency or hostility, and to inflict or receive pain. Sucking, biting, sensing, and feeling may become habitual as in thumb sucking, tongue thrusting, infantile swallowing, and biting of tongue, lip, cheek or fingernail. These actions also Figure in bruxing, clenching, tooth doodling, and smoking. Such habits may lead to tooth migration, occlusal traumatism, and occlusal wear.

Lowered host resistance

Under stress, the release of adrenaline and noradrenaline may not only induce a decrease in blood flow, but possibly also in those blood elements necessary for maintaining resistance to disease-related microbes. It may be that glucocortiocoids, released during the stress prolong this vascular response.

Bruxism

Evaluations from psychometric and health inventories suggest that stress bruxists have more muscular symptoms and seemed more emotionally disturbed. Bruxism has been considered of etiological importance in chronic inflammatory periodontal disease. However, it is difficult to find scientific evidence to substantiate this claim, which seems to be basically supported only by clinical observations.

Clinical Implications

1. Studies have demonstrated that individuals under psychological stress are more likelyto develop clinical attachment loss and loss of alveolar bone.One possible link in this regard may be increases in production of IL-6 in response toincreased psychological stress. Perhaps the relationship is simply due to the fact that individuals under stress areless likely to perform regular good oral hygiene and prophylaxis[4].

2. Recent studies were well-designed and confirm positive correlations between stress, depression and periodontal disease by demonstrating convincing linkages between depression and tooth loss; stress and attachment loss; stress/ depression and neglect of oral hygiene; and elevated cortisol levels and pocket depth/tooth loss.These studies firmly support both the biological and behavioural mechanisms for this perio–systemic connection. These findings have important clinical implications because they suggest that addressing psychological factors such as stress and depression represents an important part of overall preventive periodontal maintenance and, more importantly, may also prevent oral inflammation from developing into systemic inflammation in susceptible individuals. The simple stress profile and depression scale used in one recent study4 provides information on the psychological status of a patient (stress levels related to employment, domestic and health environments; attitudes and personality traits related to relaxation, anxiety, hostility, obsessive compulsiveness and positive/ negative perceptions of life events) and may be valuable tools in a modern periodontal practice that emphasizes individualized diagnosis, treatment planning and maintenance[5].

3. Studies conducted by RadafsharG,Zarabi.H, on relationship of stress and coping styles to periodontal disease;a case control study came to a conclusion that psychological stress associated with various ife events is a significant risk indicator for periodontal disease[6].

References

1. Paul J. Rosch, M.D., F.A.C.P. Reminiscences of Hans Selye, and the birth of "Stress". The American Institue of Stress.

2. Sachin Goyal, GarimaGupta,Betsy Thomas, K. M. Bhat, and G. S. Bhat.Stress and periodontal disease: The link and logic!! . Ind Psychiatry J. 2013 Jan-Jun; 22(1): 4–11

3. Lakshmi Boyapati & Hom-Lay Wang. The role of stress in periodontal disease and wound healing.Periodontology 2000, Vol. 44, 2007, 195–210.

4. Matthew Locke Consultant in Restorative DentistryCardiff University School of Dentistry. Periodontal Disease Risk Factors

5. Relationship Between Stress, Depression and Periodontal Disease; Anthony M. Iacopino, DMD, PhD; JCDA, June 2009, Vol. 75, No. 5

6. Radafshar G., Zarrabi H., Jalayer S. Relationships of Stress and Coping Styles to Periodontal Disease: A Case-Control Study. J Dent Shiraz Univ Med Scien 2012; 13(4): 169-175

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Conflict of Interest : Nil

CASE REPORT

Zygomatic implants - A case report with review of literature

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Abstract

Zygomatic implants are a good rehabilitation alternative for upper maxilla with severe bone reabsorption.These implants reduce the need for onlay-type bone grafting in the posterior sectors and for maxillary sinuslift procedures - limiting the use of bone grafts to the anterior zone of the upper jaw in those cases where grafting isconsidered necessary. Zygomatic implants are designed for use in compromised upper maxilla. They allow theclinician to shorten the treatment time, affording an interesting alternative for fixed prosthetic rehabilitation. Thisstudy confirms that zygomatic bone offers predictable anchorage and acceptable support function for prostheses in atrophic jaws. However, these implants are not without complications. Longer-term evaluations are needed of zygomatic implant survival in order to establish a correct clinical prognosis.

Key words: Zygomatic implants, atrophic upper maxilla, edentulism, dental implants.

Introduction

Introduced by Brånemark in 1988, zygomatic implants were designed to rehabilitate atrophic upper maxilla, or upper jaws subjected to resection for oncological reasons or with bone loss secondary to trauma. Their use has made it possible to reduce bone grafting procedures in patients seeking a permanent solution with a minimum number of surgical operations and the shortest treatment time possible - without losing the expectations for successful treatment.The insertion of standard dental implants and the preparation of a prosthesis are limited in patients with atrophic upper jaws, due to the limited amount and quality of the available bone, as well as because of the presence of highly pneumatized maxillary sinuses. In these cases it is necessary to resort to advanced bone graft surgery, such as the bloc iliac crest Le Fort I osteotomy, onlay-type bone grafting techniques, or maxillary sinus lift procedures in the posterior sectors of the maxilla[1-4]. These techniques pose a series of inconveniences, such as the need for multiple surgical interventions, the use of extraoral bone donor sites (e.g., iliac crest or skull) - with the morbidity involved in surgery of these zones - and the long time for which patients remain without rehabilitation during the graft consolidation and healing interval[5]. These factors complicate patient acceptance of the restorative treatment and limit the number of procedures carried out. Zygomatic implants are an effective treatment alternative that reduces the use of bone graft procedures, employing the zygomatic bone as anchorage. When contemplating zygomatic implant rehabilitation, the patient must present not only posterior alveolar crest reabsorption precluding the placement of additional fixations for supporting the prosthesis, but also sufficient bone volume in the anterior zone of the upper jaw - with a minimum height of 10 mm and a width of 4 mm - to allow the placement of 2-4 conventional fixations. If the bone volume in the anterior upper maxillary zone is insufficient, there must be ideal

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conditions for onlay-type bone grafting and guided bone regeneration (GBR) techniques.Different therapeutic options are available for the rehabilitation of atrophic upper maxilla[6-12]. Maxillary sinus lift procedures are accepted and are predictable for implant placement. In order to apply this technique, a bone donor site must be available. This site can be intraoral (chin, ascending mandibular ramus, retromolar trigone, etc.) or extraoral (iliac crest, skull, tibia, etc.), depending on the size of the maxillary sinus and on whether sinus lifting is to be uni- or bilateral.

Description of the zygomatic implant

The zygomatic implants (Figure. 1) are self-tapping screws in c.p. titanium with a well-defined machined surface. They are available in eight different lengths ranging from 30 to 52.5 mm. They present a unique 458 angulated head to compensate for the angulation between the zygoma and the maxilla. The portion that engages the zygoma has a diameter of 4.0 mm, and the portion that engages the residual maxillary alveolar process a diameter of 4.5 mm. At the maxillary level the angulated implant platform extremity offers the possibility to screw any kind of abutmentfrom the Brainemark system[1]. However, for the newest generation of abutments a separate slightly shorter abutment screw must be utilized for the construction of conventional screwed prosthesis.

Figure 1. Zygoma implant ® (Nobel Biocare, Götenborg, Sweden).

Case report

A 45 year old male patient reported with atrophic maxilla and partially edentulous area in relation to 35, 36 and 37 region. Due to severe ridge resorption authors planned for zygomatic implants in maxilla under general anaesthesia.

Surgical procedure

The zygomatic implant surgical procedure should involve atraumatic surgery, avoiding overheating in the zygoma bone as well as in the maxilla under sterile circumstances with what is in reality still a two-stage approach. Although the operation can be carried out underlocal anesthesia, for the patient's comfort, it has beendone up to now under total anesthesia or neuroleptic deconnection[13]. After a palatal incision of the soft tissue along the entire maxillary crest, the soft tissue is completely reflected from maxillary crest to zygomatic buttress and the suborbital nerve identified. A window is then made by drilling at the upperlimit between the zygoma and the sinus to determine the orientation of the zygoma and to reflect the Schneidarian membrane. This window will also be helpful during the surgical procedure

for cooling the drills to avoid overheating . Different drills are used with increasing diameters

ending with the insertion at low speed of the self-tapping zygomatic implant. Thelength of this is carefully chosen by means of a special gauge. After insertion of the implant, a cover screw is placed on the top of the zygomatic implant and the soft tissue closed. There are no evidence-based arguments that advocate the use of a membrane to cover the hole made in the sinus. The other implants are placed during the same surgical procedure. At the second stage surgery 6 months later, the abutments are screwed on the implants and an immediate (same day) provisional prosthesis is provided for the patient.

Prosthetic procedure

The prosthesis is made of gold and acrylic or gold and porcelain routinely like a standard screwed reconstruction on standard implants. Although screwed bridges allow a better adjustment of the occlusion, overdentures retained by rigid bars are also considered sometimes because of the important cantilever due to the palatal emergence of the zygomaticimplants and to the distance between the two maxillas or simply the resorption of the maxilla. Considering the biomechanical aspects of the prosthetic reconstructions on zygomatic implants, it is well known that when masticatory load is applied to a rigid semicircular arch connectingfour anterior implants and two zygomatic ones, the masticatory load in the posterior region is transferred to the bony support situated in the zygoma. Occlusal view of a bridge made of gold and porcelain on topof two zygomatic implants and four standard implants.

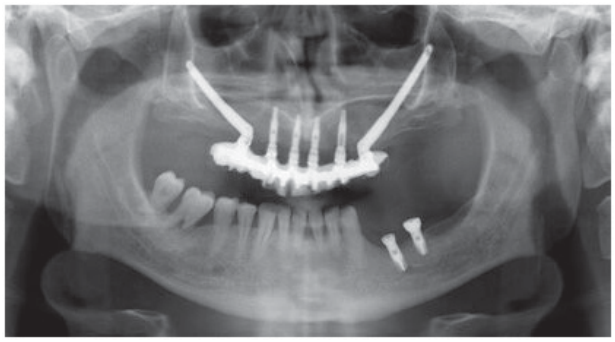

Figure-2. Postoperative panoramic X-ray viewof rehabilitation with two zygomatic implants and three standard implants in the anterior zone of the upper maxilla.

Review of literature

Depending on the remnant bone volume and quality as established by the classification of Lekholm and Zarb 1985[7], maxillary sinus lift and implant placement are carried out in one or two phases.When the bone height is insufficient to secure the necessary primary implant stability (type 4 bone with type D or Ereabsorption), bone grafting is performed in a first phase, followed 6 months later by implant placement[2]. Lekholm et al.[8]conducted a multicenter retrospectivestudy with a follow-up period of three years, involving 150 patients with edentulous upper jaws that were treated with different bone grafting techniques (onlay, inlay, or using the Le fort I osteotomy plus bone grafting). These authors reported a 23% failure rate when placing the implants in the same surgical step as bone grafting, and a 10% failure rate when two surgical phases were used. The success rate of prosthetic rehabilitation in the patients was 85%. Keller et al.[9] presented the long-term results (with 12 years of follow-up) corresponding to 118 inlay autologous bone grafts placed in the nasal and sinusal zones in patients with compromised upper maxilla. The survival rate was 87%, while the correct prosthetic function rate was 95%. These procedures may require several surgical interventions, and the duration of treatment associated with such bone grafting procedures, plus the time needed to prepare the definitive dental rehabilitation, is considerable. Nevertheless, the end results usually offer high percentage success rates. Zygomatic implants have been proposed to facilitate thetreatment of atrophic upper maxilla, since they reduce the need for a range of surgical interventions, and moreover shorten the overall treatment time [10,11].Different authors [8-13] have described the use of zygomatic implants for the functional and esthetic reconstruction of palatal deformities, post-maxillectomy defects or other mutilating disorders, and in developmental anomalies of the craniofacial skeletal components (e.g., ectodermal dysplasia) - with satisfactory results in all cases.

Summary

The management of the posterior maxilla with implant placement, including zygomatic implants, can be planned using the zones of available bone concept as a guideline. The use of anteriortilted or posterior-tilted implants allows for stablishing posterior implant support for a fixed prosthesis. The use of 2 premaxillary implants in onjunction with 2 posterior-tilted implants accomplishes this objective in a predictable manner. However, any of the implants may fail, interfering with the ability to continue use of the prosthesis. resented here is the expanded use of the zygoma implant asa "rescue implant". The removal of a failed implant is immediately replaced with a zygoma implant to reestablish support needed for continuation of the prosthesis. In cases of rare, usually unilateral sinus infection associated with zygomatic implants, the physiologic drainage of the osteomeatal complex must evaluated and be attended to, including the possible need for a FESS procedure.

Conclusion

The zygomatic implant – the zygomaticus fixture - appears to be a promising development in implant technology. It offers an interesting alternative solution to heavy bone grafting in the severely resorbed posterior maxilla. It has been in use for more than

10 years and gives a predictable outcome in the rehabilitation of totally as well as partially edentulous patients. More published reports are needed and more follow-up has to be provided to assess its final goal and predictability.

References

1. Aparicio C, Brainemark PI, Keller EK, Olive´ J. Reconstruction of the premaxilla withautogenous iliac bone with

osseointegrated implants. Int J Oral Maxillofac Implants 1993: 8:61–67.

2. Bahat O. Brainemark System Implants in the posterior maxilla:clinical study of 660 implants followed for 5 to 12 years. Int J Oral Maxillofac Implants 2000: 15: 646–653.

3. Balshi TJ, Wolfinger GJ. Treatment of congenital ectodermal dysplasia with zygomatic implants: a case report. Int J Oral Maxillofac Implants 2002; 17: 277–281.

4. Bedrossian E, Stupel L. Immediate stabilization at stage II of zygomatic implants: rationale and technique. J Prosthet Dent 2001: 86: 10–14.

5. Bedrossian E, Stumpel L III, Beckely M, Indersano T. The zygomatic implant: preliminary data on treatment of severely resorbed maxillae. A clinical report. Int J Oral Maxillofac Implants 2002: 17: 861–865.

6. Bra°nemark PI. An experimental and clinical study of osseointegrated implants penetrating the nasal cavity and maxillary sinus. J Oral Maxillofac Surg 1984: 42:497–505.

7. Carmagnola D, Berglund T, Araujo M, Albrektsson T, Lindhe J. Bone healing around implants placed in a jaw defect augmented with Bio-Oss. An experimental study in dogs. J Clin Periodontol 2000: 27: 799–805.

8. Champy M, Lodde JP, Kahn JL Kielwasser P. Attempt at systematization in the treatment of isolated fractures of the zygomatic bone: techniques and results. J Otolaryngol 1986: 39–43.

9. Esposito M, Grusovin MG, Worthington HV, Coulthard P. Interventions for replacing missing teeth: bone augmentation techniques for dental implant treatment. Cochrane Database Syst Rev. 2006 Jan25;(1)

10. Gosain AK, Song L, Capel CC, Corrao MA, Lim TH. Biomechanical and histologic alteration of facial recipient bone after reconstruction with autogenous bone grafts and alloplastic implants: a 1-year study. Plast Reconstr Surg 1998: 101: 1561–1571.

11. Higuchi KW. The zygomaticus fixture: an alternative approach for implant anchorage in the posterior maxilla. Ann R Australas Coll Dent Surg 2000: 15: 28–33.

12. Izzo SR, Berger JR, Joseph AC, Iazow SK. Reconstruction after total maxillectomy using an implant-retained prosthesis: a case report. Int J Oral Maxillofac Implants 1994: 9: 593–595.

15. Kahnberg KE, Ekestubbe A, Gro¨ndahl K, Nilsson P, Hirsch JM. Sinuslifting procedure. I. One-stage surgery with bone transplant and implants. Clin Oral Implants Res 2001: 12: 479–487.

13. Langer B. What are the clinical limitations of wide-diameter(4 mmor greater) rootform endosseous implants? Int J Oral Maxillofac Implants 2000: 15: 293–296.

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Conflict of Interest	:	Nil

CASE REPORT

Intraoral nevi - A review

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Abstract

Nevi are benign, non-neoplastic proliferations of nevus cells within the epithelium or the connective tissue. Intraoral nevi are uncommon relative to those in the skin. The most common intra oral nevus is theintramucosal type characterized by a benign, unencapsulated proliferation of small, ovoid cells nevus cells organized into small, round aggregatescalled thèques. In this article we review the pathogenesis, clinical features and histopathological features of intra oral nevi.

Key Words : Intraoral nevi, Melanocytic nevus, Theques, Blue nevus, Nevogenesis.

Introduction

Nevi are benignnon-neoplastic proliferations of nevus cells within the epithelium or the connective tissue and are categorized as hamartoma or developmental malformation.[1] The term "*Nevus*" is Latin for "maternal impression" or "birthmark."[2]Most lesions are distributed above the waist,with head and neck region being a common site of involvement.Although nevi are common lesions that are seen on the skin in the large majority of the population, they are rare intraorally.[3] In 1943, Field and Ackermann reported the first documented case of an intraoral nevus.Later King et al adopted the termintramucosal nevus.[4] Pathologically oral nevi present asjunctional, compound, intramucosal or blue nevi.

Nevogenesis

The origin of nevus cells has been postulated to be from cells that migrate from the neural crest to the epithelium and dermis, or to be from altered resident melanocytes.[5] Nevus cellare large ovoid, rounded, or spindle shaped cells with pale cytoplasm; and may contain granules of melanin pigment in their cytoplasm.[4] The nevus progenitor cells are known as *nevoblats*.

Among the various theories of origin, Masson's theory of dual origin of melanocytes was widely accepted. He believed that the nevus cells in the upper dermis developed from epidermal melanocytes, whereas the nevus cells in the lower dermis develop from schwann cells.The Abtropfung hypothesis states that nevi arise from proliferation of intraepidermal melanocytes within growth centers termed junctional nests or *theques*. An alternate hypothesis states that nevus cells arise from a pluripotential cell of nerve sheath origin.[6]

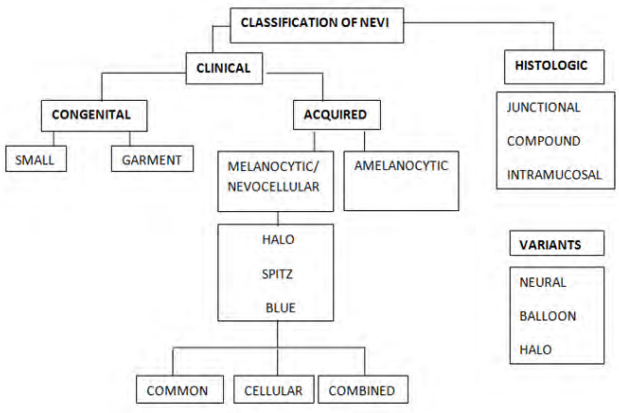

Table - 1 Classification

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Current models of nevogenesispropose that melanocytic neoplasms arise from a single cell of origin.One possibility is that an immature melanocytic stem cell serves as the nevus progenitor cell, on acquiring mutations secondary to UV light exposure or other mutagenic processes.Alternatively, the nevogenic mutation occurs in a dierentiated melanocyte, with the mutation causing the cell to regain proliferative capacity.[7]

Melanocytic lesions follow a characteristic life cyclethat consists of four stages: initiation, promotion, senescence,and involution. Initiation occurs when a nevus progenitor cell acquires a mutation. Promotion occurs when the mutated cell is activated and proliferation begins. After a period of proliferation, nevi stop proliferating through the activation of senescence pathways. This allows them to remain stable for extended periods of time before undergoing involution.[7]

Clinical Presentation

Most oral nevi are asymptomatic, and the lesions are usually detected as an incidental finding on routine dental examination.[4]The most common melanocytic nevi encountered in the oral cavity are the intramucosal type (63%), followed by blue nevi (19%), compound nevi (9%), junction nevi (5%), and combined nevi (4%).[8]

Oral nevi are found in all races and more frequently in whites, in whom 55 percent of reported oral nevi occurred.[4] Studies have revealed that oral mucosal nevi are slightly predominant in women rather than men,at a ratio of 1.5:1. The incidence of oral nevi was highest in patients aged 20-40 years, who accounted for almost one half of the patients.[8]When seen intraorally they are most commonly observed on the hard palate.[9]

Melanocytic nevi can be brown, bluish-gray, or almost black and occasionally non-pigmented. It can be congenital or acquired. Congenital melanocytic nevi sre classified into two types according to their size: giant congenital melacytic nevi(also called as ' garment nevus' , 'bathing trunk nevus' or ' giant hairy nevus') are greater 20 cm in diameter and small congenital nevi, which are usually less than 1.5 cm in their greatest dimension.[1]

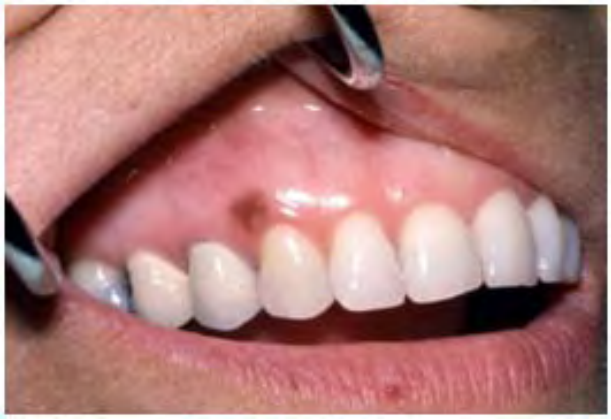

Figure 1. Intramucosal nevus on the gingiva

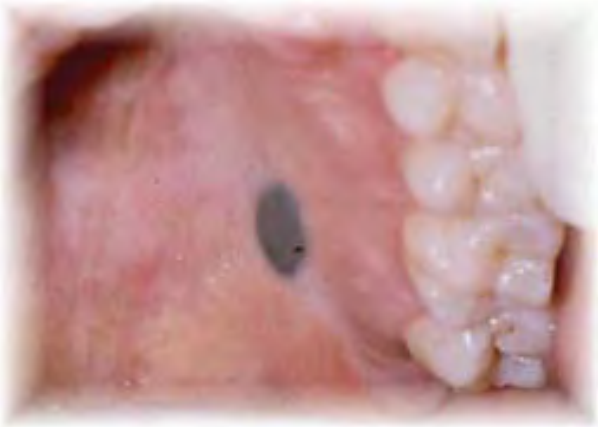

Figure 2. Blue nevus of hard palate

Junctional nevi are dark brown in colour because the nevus cells proliferate at the tips of the rete pegs, close to the surface. Intramucosal (Figure.1) and compound nevi are typically light brown dome shaped lesions.[1]

Blue nevus was first described by Tièche in 1906 as small, sharply defined, blue to blue-black spots mostly seen on the face and extremities[11]. They occur almost equally among males and females[8]. Intraoral blue nevi (Figure 2) present as asymptomatic, slightly raised, blue-black, well-circumscribed lesions.The blue color of the nevus is due to the presence of melanin deep within the dermal melanocytes and the Tyndall effect. Most blue nevi are present at an early age. The most common location of the intraoral blue nevus is the hard palate[10]. Blue nevus is a frequent component of a combined nevus and is usually associated with acommon compound or dermal nevus, or, much less frequently, Spitz nevus.Sclerosing blue nevus, also known as a desmoplastic blue nevus, is an important morphologic variant. Clinically, it

appears as a firm, solitary, variably pigmented papule or nodule.[11]

The cellular blue nevus, established as a distinct entity by Allen and Spitz et al presents clinically as a firm bluish black to bluish gray dome-shaped nodule. Large, long-standing lesions can degenerate, ulcerate, and become painful.Rare CBN can be unexpectedly amelanotic or hypomelanotic.The term *atypical cellular blue nevus* has been applied to rare lesions that show atypical histologic features and raise histologic differential diagnosis of malignant blue nevus.[11]

The Spitz nevus, also known as benign juvenile melanoma or a nevus of epitheloid and spindle cells, was first described by Sophie Spitz in 19481 and is most common in children and adolescents.[12]Other synonyms include juvenile melanoma, Spitz tumor, nevus of large spindle and/or epithelioid cells, and spindle cell and epithelioid nevus.[12,13]A Spitz nevus can arise de novo or in association with an existing melanocytic nevus. [13]They can present in three different ways: solitary nodular, multiple grouped, and multiple disseminated.The occurrence of the intra-oral Spitz nevus is very rare.[12]

Halo nevus or Sutton's nevus is a melanocytic nevus typically appear as a central pigmented papule or macule surrounded by a pale hypopigmentedborder or "halo" of the surrounding epithelium. The halo developsapparently as a result of nevus cell destruction bythe immune system. It is most common on the skin of the trunk during the second decade of life.[6,14]

The clinical differential diagnosis for oral nevi include localized lesions such as amalgam tattoo,oral melanotic macule,melanoacanthoma, melanocytic hyperplasia, post inflammatory hyperpigmentation, malignant melanoma and diffuse lesions such as smoker's melanosis, drug induced pigmentation and melasma. Oral nevi can also be associated with certain syndromes like basal cell nevus syndrome, blue rubber bleb syndrome and B-K mole syndrome.

Histopathology

The melanocytic nevus is characterized by a benign, unencapsulated proliferation of small, ovoid cells callednevus cells.A characteristic microscopic feature is that the superficial nevus cellstend to be organized into small, round aggregates *(thèques)*.Melanocytic nevi are classified histopathologically according to their stage of development, which is reflected by the relationship of the nevus cells to the surface epithelium and underlying connective tissue.[14]Based on this oral nevi are classified asjunctional, compound and intramucosal nevi.

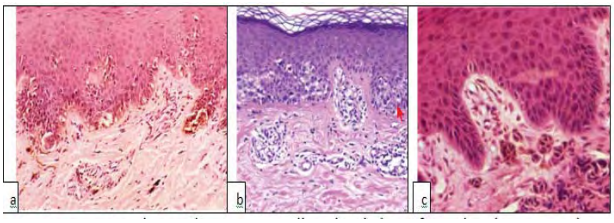

Figure 3- a- Junctional nevus showing nevus cells within the base of reteridges, b- Compound nevus showing theques in both epithelium and connective tissue, c- Intramucosal nevus showing theques within the connective tissue.

Injunctional nevus (Figure 3-a) there is proliferation nevus cell along the epithelial mesenchymal junction.The zone of demarcation is absent and the nevus cells contact and seem to blend into the surface epithelium.[15]

Migration of nevus cells into the connective tissue compartment is seen in compound nevus (Figure3-b). The cells cross the epithelial mesenchymaljunction and grow down into the connective tissue- the so-called *abtropfung or 'dropping off'* effect.[15] In the intramucosal (intradermal) nevus (Figure3-c), the nevus cells are situated within the connective tissue and are separated from the overlying epithelium by a well defined band of connective tissue.[4]

Histological variants of acquired melanocytic nevi such as neural, balloon cell (Figure 4-b) and halo nevi occur. Only the halo nevus is clinicopathologically distinctive. The neural and balloon cell nevi are histologic variants of intradermal or compound nevi.[4]

Blue nevus is characterized by a variety of histologic subtypes, although most are classified as either "common", "cellular" or combined.The common blue nevus, which is the most frequent subtype seen in the oral cavity is characterized by an intramucosal proliferation of elongated, bipolar, spindle-shaped melanocytes that are often grouped

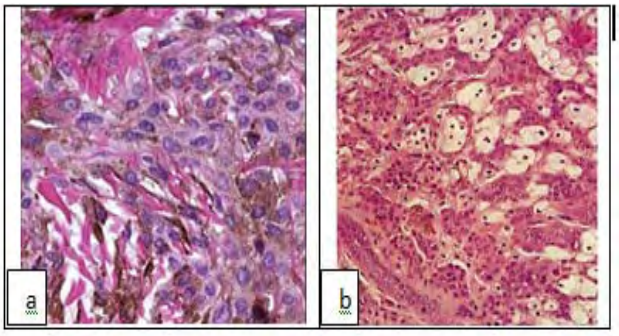

Figure 4- a- Epitheloid blue nevus, b-Balloon cell nevus

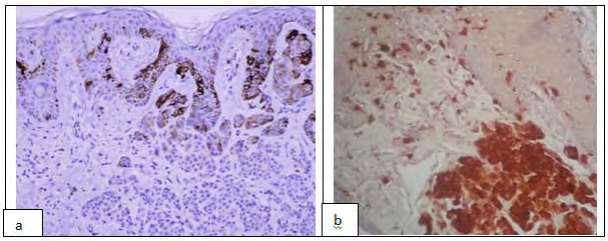

Figure 5- a-HMB 45 positivity , b-S100 positivity in intraoral nevi

in short fascicles parallel arranged to the overlying epithelium.In contrast, a cellular blue nevus is usually characterized by an intramucosal, nodular proliferation of dendritic spindle-shaped, pigmented melanocytes, in addition to tightly-packed aggregates of larger oval-to-round melanocytes with pale cytoplasm and little or no melanin.[16]

Sclerosing blue nevus shows features typical blue nevi,but is associated with exaggerated dermal fibrosis.Amelanotic BN shows a dermal proliferation of spindle cells associated with variably desmoplasticstroma. Diagnosis rests on observing architectural features of BN, such as inverted wedge shape and extension into deep reticular dermis along adnexal structures.[11]

Depending on the location and the size of the cellular areas, cellular blue nevi have different architectural outlines.Melanocytes forming the cellular areas of CBN show a characteristic oval to spindle cells, with moderate amounts of clear or lightly pigmented cytoplasm. The nuclei are usually vesicular with finely stippled chromatin and inconspicuous nucleoli.In amelanocytic CBN, Cellular component is essentially indistinguishable from that of conventional CBN but lacks pigmentation.Most malignant blue nevi arise in association with a preexisting benign BN or CBN.Atypicalcytologic features, abnormal mitosis and tumor necrosis have emerged as the features most specific for malignant blue nevus.[11]

The spitznevus (spindle cell and or epitheloid cell nevus) is basically composed of pleomorphic cells of three basic types: spindle cells, oval or epithelioid cells and both mononuclear and multinucleated giant cells. These are arranged in well circumscribed sheets and there is generally considerable junctional activity.[4] Histopathologically, a Spitz nevus is very similar to a melanoma due to the large size of the spindle or polygonal cells, frequently containing considerable cellular and nuclear pleomorphisms, the presence of inflammatory infiltrate, and, on the base of the lesion, a diminished cell size and spread among the collagen fibers of the connective tissue.[12]

An atypical Spitz nevus shares histologic features with the classic Spitz nevus, but it may have one or more atypical features, which can be characteristic of malignancy. These include pleomorphism, increased cellularity, loss of cellular cohesion, epidermal pagetoid spread, high basal mitotic rate; pushing deep margins into the dermis and nests variable in size, shape, and orientation.[17]

Histopathologically, the halo nevus differs from the routine acquired melanocytic nevus only in thepresence of an intense chronic inflammatory cell infiltrate, which surrounds and infiltrates the nevuscell population.[14]

The special stains used for the demonstation of nevus cells include Masson Fontana, Shmrol's reaction, Churukian'sammonical silver method and DOPA reaction.Immunohistochemical markers such asHMB45 (Figure 5-a), S100 protien, Melan A, MiTF, Tyrosinase, PGP 9.5 and Neuron specific enolaseshows positivity for the nevus cells.

Treatment & Prognosis

Surgical excision is recommended for intraoral nevi as a prophylactic measure becauseoral melanocytic nevi clinically can mimic an early melanoma. Recurrence is rare after surgical excision.[4]Congenital nevi, cellular blue neviand junctional nevi have highest chances for malignant transformation. Although transformation of oral pigmented nevi to melanoma has not been well documented, it is believed that nevi represent precursor lesions to oral mucosal melanoma.[1] Variation is symmetry, border,colour,diameter, evolving and elevation

(ABCDE rule) are considered as alarming or warning signs of malignant transformation of nevi.

References

1. Sabrinath B, Sivapathasundharam B, GauravGhosh, Dhivy. Pigmentation:IJDA 2009;1(1):38- 43.
2. Hiroshi Shimizu. Nevus and neurocutaneous syndrome.In: Hifoshi Shimizu, Shimizu's Textbook of dermatology. Nakayama: Hokkaido university press;2007.p-327-331.
3. V.T Beena, IshaChauhan R, Heera et al .Oral melanotic nevi: a case report and review of literature. Oral & Maxillofacial Pathology Journal 2010.
4. Rajendran R. Benign and malignant tumors of the oral cavity. In: Rajendran R, Shivapathsundharam B, editors. Shafer's Textbook Of Oral Pathology. 7th ed. Delhi: Reed elsevier; 2012.p.81-222.
5. Regezi, Sciubba, Jordan. Oral pathology clinicopathological correlation. 6thed.
6. Elder DE, Elenitsas R, Johnson B, Murphy G, editors. Lever's histopathology of the skin.9th ed. Philadelphia: JB Lippincott; 2005.
7. Andrew L. Ross,Margaret I. Sanchez et al. Molecular Nevogenesis. Dermatology Research and Practice 1-9.
8. www.medscape.com.
9. Craig L. Hatch. Pigmented lesions of the oral cavity. Dental Clinics of North America 2005;49(1):185-220.
10. JunuOjha, Jesse L. Akers, John O. Akers .Intraoral Cellular Blue Nevus: Report of a Unique Histopathologic Entity and Review of the Literature.Cutis 2007;80:189-192.
11. ArturZembowicz, Pushkar A. Phadke. Blue Nevi and Variants- An Update . Arch Pathol Lab Med 2011; 135:327- 336.
12. Ruth Lopes, G Marcelo, C G Hébel et al.Intra-oral spitznaevus: a case report:Clinics 2008;63(1):140-142.
13. Daryl J. Sulit, MD. Spitz nevus: Dermatology Review.com journal; june 2005;1-6.
14. C.Angela .Epithelial pathology.In: Neville, Damm, Allen, Bouquot. Oral and Maxillofacial pathology, 3rded.Saunders; p-362-452.
15. Surej Kumar L.K, Varun Menon . Oral Nevi presenting as a kissing lesion on Lip: A case report .IOSR Journal of Dental and Medical Sciences 2013; 5(2): 16-18.
16. Rogério-Oliveira Gondak, Rogério da Silva-Jorge et al. Oral pigmented lesions: Clinicopathologic features and review of the literature. Med Oral Patol Oral Cir Bucal.2012 ; 17 (6):919-924.
17. LT Daryl J. Classic and Atypical Spitz Nevi: Review of the LiteratureCuti .2007;79:141-146.

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CASE REPORT

Inclusion bodies : A significant clue in pathologic diagnosis

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Abstract

One of the specific and interesting features during histopathologic and hematologic analysis is the observation of certain intracellular and extracellular structures, which show peculiar morphologic and staining characteristics. These are called inclusion bodies, which can be aggregates of proteins, viral, bacterial or fungal organisms, altered cellular structure or calcifications. In this article we have discussed some of the inclusion bodies seen in various pathologic conditions with their microscopic appearance and staining characteristics.

Key words: Inclusion bodies, diagnosis, pathology

Introduction

Inclusion bodies are abnormal structures seen in histopathology and haematology, which aids in arriving at proper diagnosis of certain pathologic conditions. They show peculiar morphological alterations giving rise to specific patterns and staining characteristics which make them easy to identify. These bodies can be aggregates of proteins, viral, bacterial or fungal organisms, altered cellular structure or calcifications which are identified either intracellularly or extracellularly. Intracellular inclusion bodies can be seen either in the cytoplasm, in the nucleus or in both.[1] The inclusion bodies seen in specific pathologic conditions are listed (Table I).

1. Inclusion bodies seen in infectious diseases

a. Viral infections:

i) Intra cytoplasmic inclusions

Negri bodies

Negri bodies are intra cytoplasmic, eosinophilic, sharply outlined, pathognomonic inclusion bodies found in certain nerve cells of the brain in patients infected with rabies. They are formed by the accumulation of viral nucleoproteins and cellular organelles and measure around 2–10 μm in diameter.[2] These inclusion bodies were first detected by Adelchi Negri and reported it officially in 1903.[3] Staining with Giemsa can differentiate Negri bodies from other intracellular inclusions by their characteristic magenta colour with dark-blue granules.[4] (Figure 1a)

Guarnier bodies

Guarnier bodies are intra cytoplasmic, round to oval, eosinophilic bodies, which was named after the Italian physician Giuseppe Guarnieri.[5] They are located adjacent to the nucleus and appear as pink blobs and are found in virtually all poxvirus infections. They are formed by the aggregation of viral particles.[6] (Figure 1b)

Henderson Petterson bodies

Henderson–Patterson bodies, also called Molluscum bodies(Figure 1c), are large, round cytoplasmic inclusions, and is the characteristic cytologic feature of molluscum contagiosum.[7,8] These bodies appear about 1-2 layers above the basal cell layer of the epidermis as pink to red inclusions, in hematoxylin and eosin stain. They increase in size as the infected cells move towards the surface. As it reaches the upper layers of the epidermis, it compresses the nucleus appearing as

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PATHOLOGIC CONDITIONS	INCLUSION BODIES
1. Infectious diseases: a. Viral infections.	i. Intra cytoplasmic Negri bodies Guarnier bodies Henderson Petterson bodies
b. Bacterial infections.	ii. Intra nuclear Cowdry type A bodies Cowdry type B bodies Mitosoid bodies Owl's eye bodies
c. Fungal infections	iii. Both intra cytoplasmic and intra nuclear Warthin Finkeldey cell Leishman-Donovan bodies Babes-Ernst bodies Dohle bodies Asteroid bodies
2. Neoplasm.	Psammomatoid Bodies Verocay bodies Kamino Bodies Wagner Meissner bodies Pustulo-ovoid bodies
3. Inflammatory conditions	Russel bodies
4. Immune mediated disorders	L E Body Civatte and colloid bodies
5. Reactive lesions	Toto bodies
6. Cystic lesions	Rushton bodies
7. Blood dyscrasias	Basophilic stipplings Howell jolly bodies Pappenheimer bodies Cabot rings Heinz bodies Fessas bodies

a thin crescent at the periphery of the cell. At the granular layer, the staining reaction of the molluscum body changes from eosinophilic to basophilic.[9] They are regarded as the cellular response to the presence of living foreign body.[7,8] Ultrastructure shows each inclusion is composed of brick shaped viruses with dumbbell shaped nucleoids surrounded by the amorphous layer of capsids.[10]

ii) Intra nuclear inclusion bodies

Cowdry type A and Cowdry type B bodies

Cowdry Type-A inclusion bodies are seen in viral infections caused by herpes simplex virus and varicella zoster virus. In routine histopathology, they appear as droplet-like masses of acidophilic material surrounded by clear halos within nuclei, with margination of chromatin on the nuclear membrane [1,11] measuring 3-8 micrometre in diameter.[12] (Figure 1d)

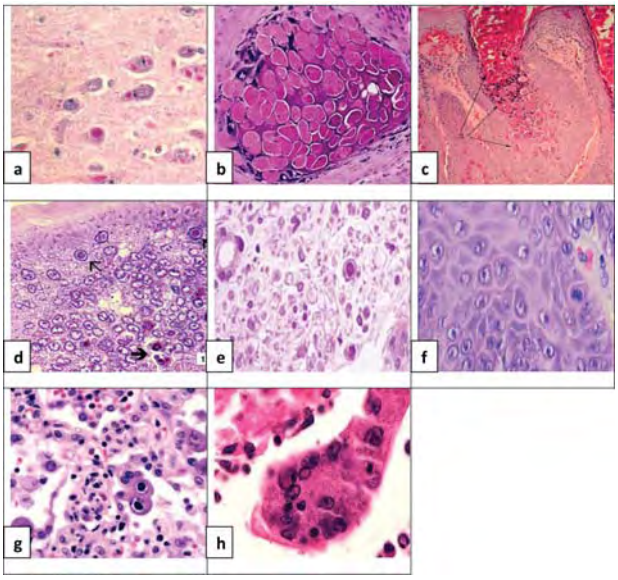

Figure 1.Viral inclusion bodies:
a :Negri bodies, b: Guarnier bodies, c: Henderson Petterson bodies, d: Cowdry type A bodies, e: Cowdry type B bodies, f: Mitosoid bodies, g:Owl's eye bodies, h: WarthinFinkeldey cell

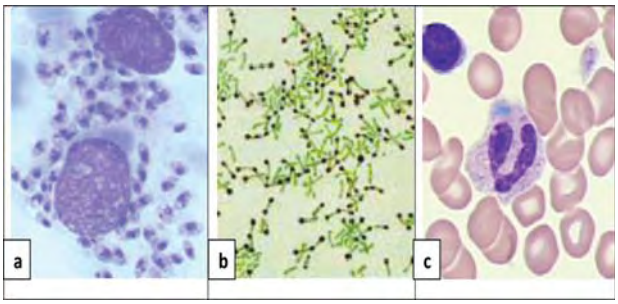

Figure 2. Bacterial inclusion bodies:
a: Leishman-Donovan bodies,
b: Babes-Ernst bodies, c: Dohle bodies

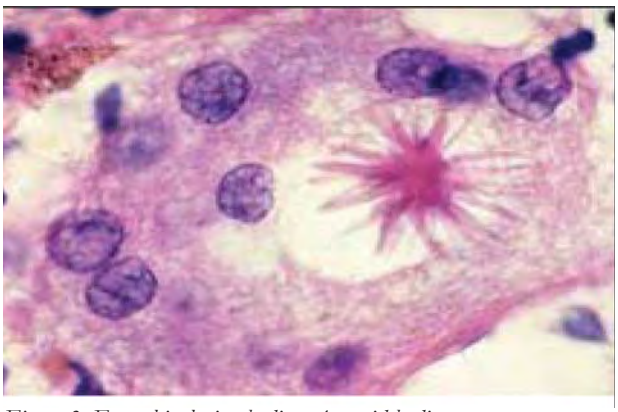

Figure 3. Fungal inclusion bodies: Asteroid bodies

Cowdry Type-B inclusion bodies(Figure 1e) are the eosinophilic amorphous or droplet like bodies surrounded by clear halo without any associated nuclear changes seen in adenovirus and poliovirus infections.[1,11]

Mitosoid bodies

Mitosoid bodies (mitosoid cells, mitosoid Figures) are the hallmark of focal epithelial hyperplasia. These are cells in which the nucleus contains fragmented DNA and has coarsely clumped heterochromatin, resembling a mitotic Figure.[14] They are seen in the middle spinous layer of the epithelium.[13](Figure 1f)

Owl's eye inclusion bodies

Owl's eye inclusion bodies(Figure 1g) are seen in hodgkin's lymphoma and cytomegalo viral infections. Cytomegalo virus causes cytopathic effects in the affected cells thus giving rise to basophilic nuclear inclusions. There is visible margination of the chromatin around the nuclear inclusion resulting in a halo effect or the owl's eye appearance.[15]

ii) Both Intracytoplasmic and Inranuclear inclusions

Warthin–Finkeldey cell

A **Warthin–Finkeldey** cell is a type of multinucleated giant cell found in hyperplastic lymph nodes. It is seen in the early course of measles, HIV-infected individuals, Kimura disease, and rarely in a number of neoplastic (e.g. lymphoma) and non-neoplastic lymph node disorders. It consists of fused lymphocytes and can be seen in a lymph node biopsy after a measles vaccination.Under the light microscope, these cells consist of a large, grape-like cluster of nuclei. The characteristic Warthin-Finkeldey syncytial giant cell may have 50 nuclei, many with intra nuclear inclusions. Hyaline-like cytoplasmic inclusions may also be seen.[16](Figure 1h)

b. Bacterial infections

Leishman-Donovan bodies

They are dull blue grey, small, round to oval bodies(Figure 2a) of 2⁴ 4 ìm in diameter with indistinct cytoplasm, round basophilic nucleus and a small, rod-shaped paranuclear kinetoplast seen in leishmaniasis.[17] Leishman's or Giemsa's stains can be used for the demonstration of these bodies. They are non flagellated form of the parasitic protozoan *Leishmania donovani* and occur once the parasite has invaded the cells of the reticuloendo thelial system.[18]

Babes-Ernst bodies

The cells of cornybacterium diphteriae contains granules composed of polymetaphosphate. These

granules when stained with loeffler's methylene blue, take up bluish purple colour, hence called metachromatic granules. They are called volutin or babes ernst granules(Figure 2b). They are often situated at the poles of the bacilli and are called polar bodies. Special stains such as Albert's, Neisser's and Ponder's have been diviced for demonstrating these granules clearly.[19]

Dohle bodies

Dohle bodies(Figure 2c) are small, one micron size pale irregular light blue grey, basophilic, rounded oval or rod shaped structures seen chiefly in the periphery of neutrophils in patients infected with infections like tuberculosis, diphtheria, and typhoid.[6,4] These cells are seen in the peripheral blood smear and reflect the defective cell production and maturation of neutrophils. Special stains like Leishman-Giemsa stain and Romanowsky stain can be used for the demonstration of Dohle bodies.[1]

c. Fungal infections:

Asteroid bodies

Asteroid bodies(Figure 3), also known as cigar bodie appear as stellate inclusions with numerous rays radiating from the central core. They are found in the disease sporotrichosis. [6,20]

2. Inclusion bodies seen in neoplasms

Psammoma Bodies

Psammoma bodies are seen in numerous benign and malignat epithelial and connective tissue tumours. They are seen in psammomatoid juvenile ossifying fibroma, psammomatoid melanotic schwannoma, cystadenocarcinoma and psammomatoid meningoma. These bodies shows mineralized collagenous foci that vary from small, smoothly contoured round to oval patterns to larger irregularly shaped ossicles like pattern with concentric layering similar to that of psammoma bodies(Figure 4a). The ossicles are identified within bony trabeculae as well as within the adjacent cellular stroma. The ossicles are rimmed by a prominent marginal osteoid and may also contain osteocytes.[21]

Wagner Meissner bodies

Wagner Meissner bodies are oval aggregates of eosinophilic globules. (Figure 4b) containing parallel slits and bordered by nuclei, observed within the cellular sheets of neurofibroma.[1,22,23.] These bodies are identical to the tactile corpuscle-like Meissner bodies

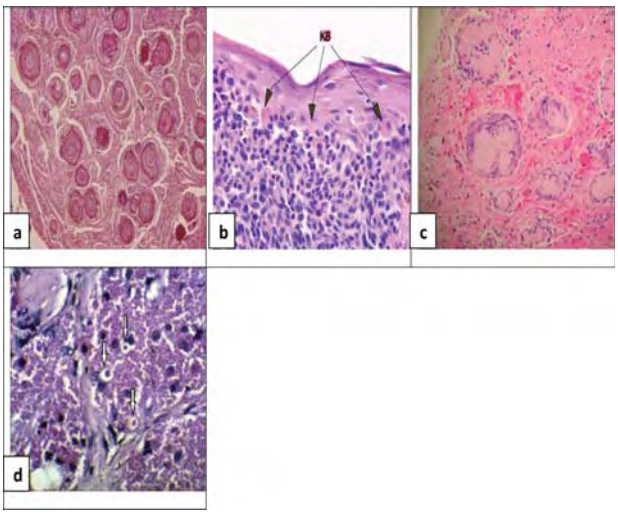

Figure 4. Inclusion bodies seen in neoplasms: a: Psammomatoid bodies, b:Kamino bodies, c:Wagner Meissner bodies, d:Pustulo-ovoid bodies.

Kamino Bodies

Kamino bodies are eosinophilic hyaline globules with scalloped borders commonly located at the dermoepidermal junction.[24] They are found to be associated with Spitz nevus and pigmented spindle cell nevus. Kamino bodies(Figure 4c) are composed of laminin, fibronectin, type IV collagen and type VII collagen and therefore, they give a positive reaction to periodic Acid- Schiff 's and trichrome stains.[25]

Pustulo-ovoid bodies of Milian

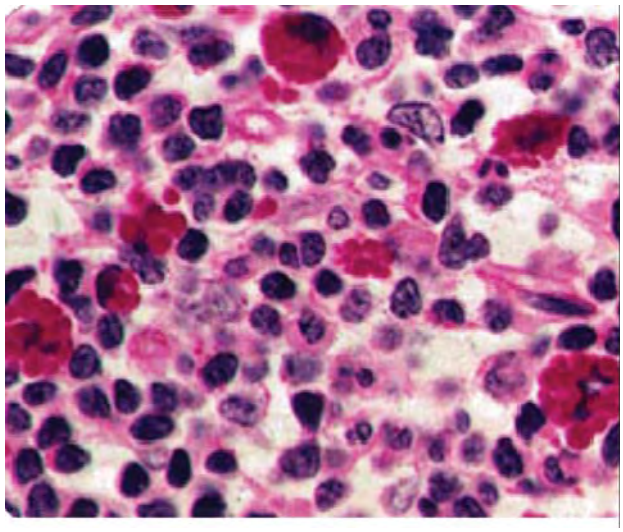

Figure 5. Inclusion bodies seen in inflammatory conditions: Russel bodies

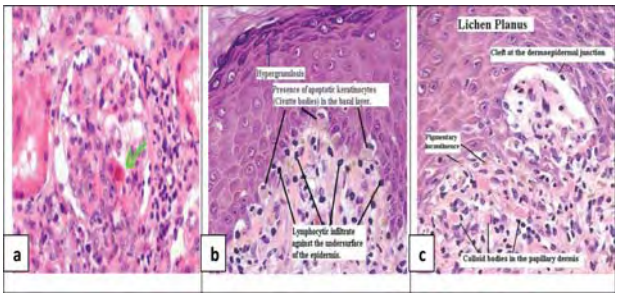

Figure 6. Inclusion bodies seen in immune mediated disorders: a : LE bodies, b:Civatte bodies, c: Colloid bodies

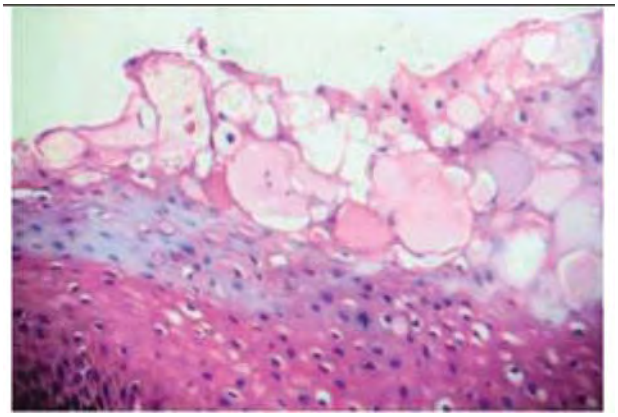

Figure7. Inclusion bodies seen in reactive lesions: Toto bodies

Pustulo-ovoid bodies (bangle bodies)[26] are large eosinophilic intra-cytoplasmic inclusions composed of coalescing granules, representing lysosomes and are surrounded by a clear halo(Figure 4d). They are seen in granular cell tumour of tongue.[6,27]. These granules were diastase-resistant and PAS-positive.[28]

3. Inclusion bodies seen in inflammatory conditions

Russell bodies

Russell bodies(Figure 5) are eosinophilic, large, homogenous immunoglobulin-containing inclusions usually found in a plasma cell undergoing excessive synthesis of immunoglobulin characteristized by distended endoplasmic reticulum. They are found in pathologic conditions such as plasmocytoma, multiple myeloma and rhinoscleroma.[29] They measure about 20- 40 μm in diameter.[6] They are named for William Russell (1852-1940), a Scottish Physician.[30]

4. Inclusion bodies seen in immune mediated disorders

LE body

LE bodies(hematoxylin bodies) are seen in systemic lupus erythematosus. Immunologically this disease is associated with an enormous array of autoantibodies, classically including the antinuclear antibodies. The serum of the patient shows exposed nucleus of the damaged cell which react with ANAs resulting in the formation of a homogenous mass of nuclear chromatin material. These appear as basophilic round bodies called the LE body or haematoxylin body. LE cell is a phagocytic leukocyte commonly PMNs and macrophages which engulf the homogenous nuclear material of the injured cell [31,32,33](Figure 6a)

Civatte or Colloid bodies

Civatte or Colloid bodies are also called as Cytoid, Hyaline or Sabaroud bodies. These bodies are characteristic finding in skin lesions of patients with various dermatoses, particularly lichen planus (LP) and discoid lupus erythematosus(DLE). They are generated by damaged basal keratinocytes through apoptotic cell death, consist largely of keratin intermediate filaments, and are almost invariably covered with immunoglobulins, mainly IgM. Citoid bodies appear as rounded, homogenous, eosinophilic masses on routine H and E staining lying in the deeper parts of epidermis/epithelium and more frequently in dermis/connective tissue. They are known as Civatte bodies (in epithelium/ epidermis) (Figure 6b), colloid bodies or hyaline bodies (in connective tissue) (Figure 6c). They are 10-25 ìm in diameter and situated mostly within or above the inflammatory cell infiltrate.[34]

5. Inclusion bodies seen in reactive lesions

Toto bodies

Toto bodies are homogeneous, eosinophilic pools of material in the superficial spinous layer of the surface epithelium. The epithelial cells of the gingiva in inflammatory fibrous hyperplasia (epulis fissuratum) exhibit mucopolysaccharide keratin dystrophy which replaces individual cells. This is referred to as plasma pooling and this altered appearance is termed as toto bodies.[1, 4](Figure 7)

6. Inclusion bodies seen in cystic lesions

Rushton bodies

Rushton bodies appear as eosinophilic, linear, straight or curved or hairpin shaped, circular or polycyclic forms, often with a granular core and sometimes concentrically laminated(Figure 8). They occur almost exclusively within odontogenic cysts particularly radicular cyst, residual cyst and

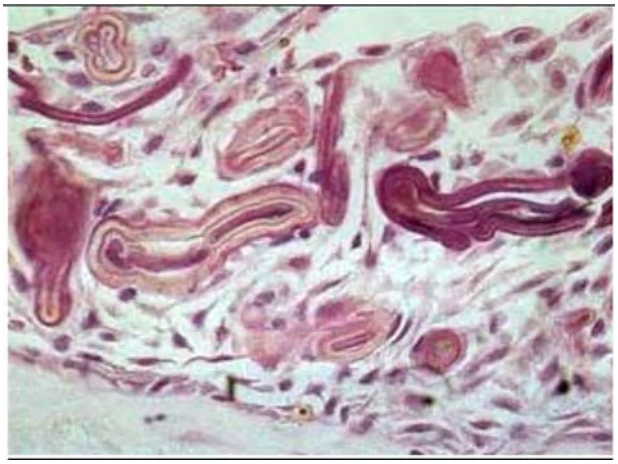

Figure 8. Inclusion bodies seen in cystic lesions: Rushton bodies

plexiform ameloblastoma and are found almost always within the epithelial lining and only rarely in the fibrous capsule. Electron microscopic studies show the lamellar variant to be composed of alternating electron dense and electron lucent layers, whereas the granular form consists of amorphous material in which fragments of red blood cells are seen. They stain strongly with orcein, Mallory aldehyde fuschin, Papanicolaou and Gomori.[6,35]

7. Inclusion bodies seen in blood discrasias:

Basophilic stipplings

Basophilic stipplings are round or irregularly shaped granules of variable size seen in erythrocytes when stained with giemsa.[36] These stipplings represent the spontaneous aggregation of ribosomal RNA in the cytoplasm of erythrocytes.[37] They are frequent in anemias as a result of lead or other heavy metals poisoning.[36](Figure 9a)

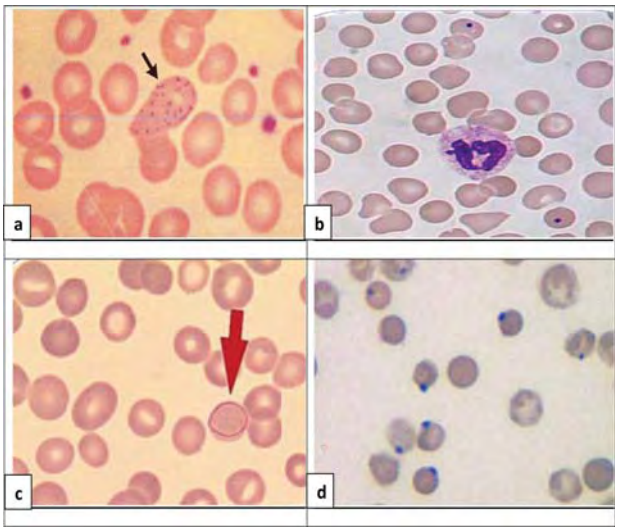

Figure 9. Inclusion bodies seen in blood dyscrasias: a : Basophilic stippling, b: Howell jolly bodies, c: Cabot rings, d: Heinz bodies

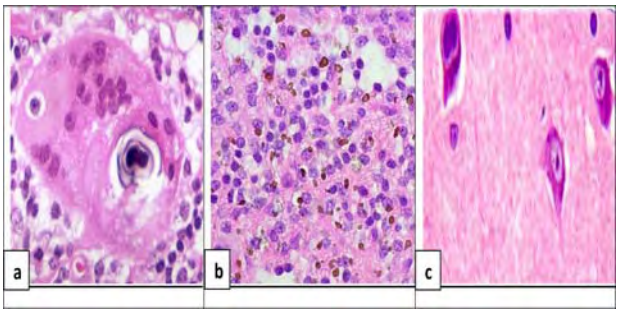

Figure 10. Inclusion bodies seen in granulomatous diseases: a :Schaumann bodies, b: Hamazaki-wesenberg bodies, c: Lafora bodies

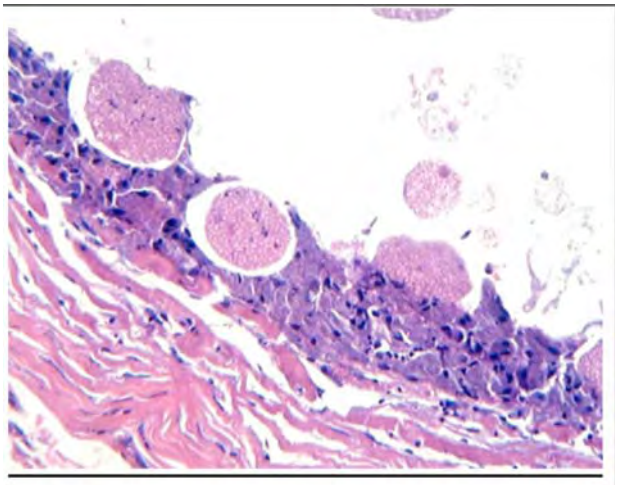

Figure 11. Inclusion bodies seen in foreign body reactions: Parent bodies

Pappenheimer bodies

Pappenheimer bodies are abnormal granules of iron found inside red blood cells on routine blood stain. They are a type of inclusion body formed by phagosomes that have engulfed excessive amounts of iron. They appear as dense, blue-purple granules within the red blood cell and there is usually only one or two, located in the cell periphery. They are seen in iseases such as sideroblastic anemia, hemolytic anemia, and sickle cell disease . Pappenheimer bodies are visible with a Wright and/or Giemsa stain. Confirmation of non-heme iron in the granules is made with a Perls' Prussian blue stain.[38]

Howell jolly bodies

Howell–Jolly bodies are small round basophilic inclusions that are nuclear remnants of DNA in circulating erythrocytes(Figure 9b). During maturation in the bone marrow erythrocytes normally expel their nuclei, but in some cases a small portion of DNA remains. Its presence usually signifies a damaged spleen. It is named for William Henry Howell and Justin Marie Jolly. They are very rarely seen in normal people but common after the removal of the spleen, in some haemolytic anemias and in megaloblastic anemia.[39]

Cabot rings

Cabot rings were first described in 1903 by American physician, Richard Clarke Cabot. They are thin, red-violet staining, thread like strands in the shape of a loop or Figure 8 that are found on rare occasions in red blood cells(Figure 9c). They are believed to be microtubules that are remnants from a mitotic spindle, and their presence indicates an abnormality in the production of red blood cells. Cabot rings stains red or purple with a Wright's stain. Cabot's rings have been described in pernicious anemia, lead poisoning, leukemia, alcoholic jaundice, and in some forms of severe anemia. They can also be seen in patients taking cytostatics.[40]

Heinz bodies

They are small irregular deep purple granules in red blood cells. They are found in glucose-6-phosphate dehydrogenase deficiencies but also found in congenital haemolytic anaemias and in premature infants. They are named after Robert Heinz (1865-1924), a German physician. Also known as Ehrlich or Heinz-Ehrlich bodies[4] they occur as a result of damaged DNA component usually through oxidation or due to change in the internal morphology of the amino acid residue within the red blood cells.[6] They are best seen when blood films are stained with dyes such as crystal violet or methylene blue.[4] (Figure 9d)

Fessas bodies:

Fessas P., 1963noticed the spontaneous formation of precipitated Hb like material within the immature and/or mature erythroid cells of patients with severe or intermediate â- thalassemia. These inclusion bodies were attributed to an excess of á-Hb chains. [41]

8. Inclusion bodies seen in granulomas

Schaumann (conchoidal) bodies:

Large basophilic concentric lamellar calcifications of 100 μm in size, often containing central birefringent calcium oxalate crystals. They are seen in sarcoidosis, and other granulomatous disorders like tuberculosis, leprosy etc. They are composed of lipo mucoglycoproteins impregnated with calcium and iron. Although usually intracytoplasmic they may, if numerous or very large, be extruded into the extracellular space.[1,42](Figure 10a)

Hamazaki-wesenberg (H-W) bodies:

They are oval or spindle-shaped, range up to several microns in size, and appear yellow-brown with H&E stain(Figure 10b). They are found in sarcoidosis. The pigment appears to be lipofuscin. Ultrastructural studies have shown that H-W bodies are giant lysosomes and residual bodies. They are acid-fast and stain with silver stains as well as a variety of other stains. When visualized with silver stains H-W bodies can be easily mistaken for budding yeasts due to clustering of individual bodies.[42]

Lafora bodies:

Lafora bodies are cytoplasmic, basophilic and metachromatic inclusions having a concentric target like lamination(Figure 10c). They are found in nerve cells but may also be found in the liver, muscle and skin of patients with Lafora body disease. The bodies are Alcian blue and PAS positive but resistant to diastase.[4] These inclusion bodies are also seen in the excretory ducts of eccrine and apocrine sweat ducts of clinically normal skin.[6]

9. Inclusion bodies seen in foreign body reactions

Parent body

Parent bodies are seen in a condition called myospherulosis, which is a complication of healing of an extraction wound or soft tissue wound into which was placed antibiotic ointment with a petrolatum base. This treatment results in the formation of multiple cyst like spaces that contain numerous brown-black within the connective tissue within the connective tissue. The collection of spherules sometimes are surrounded by an outer membrane known as parent body (Figure 11), forming structures that resemble a 'bag of marbles'. The spherules represent red blood cells that have been altered by the medication. The unusual dark colouration is due to degradation of haemoglobin.[43]

Conclusion

Inclusion bodies appear as a result of any disharmony to the normal homeostasis of the body and therefore their appearance is pathognomonic. These bodies can be due to the altered cellular and nuclear changes in response to any biochemical process during the course of the disease. Therefore these bodies, if present, are an important aid in diagnosing various pathologic conditions.

References

1. Kulkarni M, Agrawal T, Dhas V. Histopathologic bodies: An insight. Journal of International Clinical Dental Research Organ. 2011; 3: 43-7.
2. Available from: www.encyclo.co.uk/define/NEGRI%20BODIES
3. Bentivoglio M. Intraneuronal inclusion bodies: From negri bodies to proteasomal dysfunction. Rendiconti Lincei .2003; 14 (4): 263-279.
4. Available from: http://pathological bodies.weebly.com/bodies-o-r.html
5. Available from: http://en.wikipedia.org/wiki/B_type_inclusion.
6. Sequeira FF, Kumar A, Kini U, Jayanthi. Named bodies in dermatology. Indian Journal of Dermatology, Venereology and Leprology. 2010; 76:207-13.
7. Krishnamurthy J, Nagappa D K. The cytology of molluscum contagiosum mimicking skin adnexal tumor. Journal of Cytology. 2010; 27(2): 74–75.
8. Gupta RK, Naran S, Lallu S, Fauck R. Cytologic diagnosis of molluscum contagiosum in scrape samples from facial lesions. Diagnostic Cytopathology. 2003; 29(2):84.
9. Xu X, Erickson LA, Elder DE. Diseases caused by viruses. In: Elder DE, Elenitsas R, Johnson B, Murphy G, editors. Lever's histopathology of the skin. 9th ed. Philadelphia: JB Lippincott; 2005. p. 662-3.
10. Available from:http://www.images.missionforvisionusa.org/anatomy/2006/08/what-is-molluscum-contagiosum.html
11. Available from: http://www.drugs.com/dict/cowdry-type-a-inclusion-bodies.html
12. Xu X, Erickson LA, Elder DE. Diseases caused by viruses. In: Elder DE, Elenitsas R, Johnson B, Murphy G, editors .Lever's histopathology of the skin. 9th ed. Philadelphia: JB Lippincott; 2005. p. 652-7.
13. Available from:http://www.rdhmag.com/articles/print/volume-32/issue 60/columns/focal-epithelial-hyperplasia.html
14. Peixoto RF, de Araújo GM, de Araújo RM, Silvestre VD, de Medeiros AMC. Focal epithelial hyperplasia in Brazilian family. RFO, Passo Fundo. 2013;18(2):197-200.
15. Joseph D, Jokowsky, Celestste N. Powers.Infectious diseases. In: Syed Z. Ali, Grace C. H. Yang, editors. Lung and Mediastinum Cytohistology. United States of America: Papinicolaou society of cytopathology; 2012. 32.
16. Available from: http://blog.acrosspg.com/2014/03/warthin-finkeldey-cells.html
17. Sellheyer K, Haneke E. Protozoan diseases and parasitic infestations. In: Elder DE, Elenitsas R, Johnson B, Murphy G, editors. Lever's histopathology of the skin. 9th ed. Philadelphia: JB Lippincott; 2005. p. 635-40.
18. Dacie, John V, Bain, Barbara J, Imelda Bates. Dacie and Lewis practical haematology. Philadelphia: Churchill Livingstone/Elsevier; 2006
19. Ananthanarayanan R, Jairam Panicker CK. Textbook of Microbiology. 8th ed. India: Universities press private limited; 2009.
20. B Sivapathasundaram, N Gururaj. Bacterial infections of oral cavity. Rajendran R, Shivapathsundharam B. Shafer's Textbook of Oral Pathology. 5th ed. Delhi: Saunders; 2006. 435-463.
21. Nair P, Kumar A, Hedge K, Neelakantan S. Psammomatoid Juvenile Ossifying Fibroma: A Case Report with literature review. Journal of Indian academy of oral medicine and radiology.2010; 22(4):53-57.
22. Ben ZP. Head and neck surgical pathology; Soft Tissue Pathology of Head and Neck. 1997; 15: 397-419.
23. Ohno J, Iwahashi T, Ozasa R, Okamura K, Taniguchi K. Solitary neurofi broma of the gingival with prominent differentiation of Meissener bodies: A case report. Diagnostic Pathology 2010;5:61.
24. Kamino H, Flotte TJ, Misheloff et al. Eosinophilic globules in the Spitz nevus. Journal of American Academy of Dermatology. 1982; 7: 324-327.

25. LeBoit PE. Pathology and genetics of skin tumours. Melanocytic Tumors.1959; 6 :115-7.
26. Fernandes BF, Neto RB, Odashiro AN, Pereira PR, Burnier MN Jr. Clinical and histopathological features of orbital granular cell tumor: case report. Arquivos Brasileiros de Oftalmologia. 2012; 75(2):137-9.
27. Epstein DS, Pashaei S, Hunt E Jr, Fitz patrick JE, Golitz LE. Pustuloovoid bodies of Milian in granular cell tumors. Journal of Cutaneous Pathology. 2007; 34: 405-9.
28. Pushpa G, Karve PP, Subashini K, Narasimhan MN, Ahmad BP. Abrikossoff's Tumor: An Unusual Presentation. Indian journal of dermatology. 2013 ; 58(5): 407.
29. Martín-Noya A, Ríos-Herranz E, Rafel-Ribas E. Multiple myeloma with numerous intranuclear Russell bodies. Haematologica. 1999; 84 (2): 179–80.
30. Russell W. "An Address on a Characteristic Organism of Cancer". British Medical Journal. 1890; 2(1563): 1356–1360.
31. Mohan H. Textbook of pathology.6th ed. New Delhi:Jaypee Brothers ;2010 .
32. Cotran RS, Kumar V, Robbins SL: Robbins Pathologic Basis of Disease, 7th ed. Philadelphia, saunders- an \Imprint of Elsevier, 2004.
33. Grishman E, Churg J. Ultrastructure of hematoxylin bodies in systemic lupus erythmatous. Archieves of Pathology and Laboratory Medicine. 1979;103: 573-6.
34. Pranay T, Kumar AS, Chhabra S. Civatte Bodies: A Diagnostic Clue. Indian J Dermatol. 2013 Jul-Aug; 58(4): 327.
35. Shear M, Spieght P. Odontogenic Keratocyst. In Cysts of Oral and Maxillofacial regions. 4th ed. Oxford: Blackwell Munksgaard; 2007. p. 123-42.
36. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6202140.
37. Available from: https://ahdc.vet.cornell.edu/clinpath/modules/rbcmorph/bse.html.
38. Sears DA, Udden MM. "Pappenheimer bodies: a brief historical review". American Journal of Hematology. 2004; 75 (4): 249–50.
39. Howell W.H. The life-history of the formed elements of the blood, especially the red blood corpuscles. Journal of Morphology. 1890; 4(1): 57-116.
40. McPherson, Richard A, MR Pincus. Henry's clinical diagnosis and management by laboratory methods 22nd ed. Philadelphia, PA: Elsevier/Saunders; 2006.
41. Fessas P. Inclusions of Hemoglobin in erythroblasts and erythrocytes of thalassemia. Blood. 1963; 21:21-32.
42. Available from: http:// granuloma. homestead.com/inclusions.html.
43. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial Pathology.3rd ed Missouri: Saunders; 2009.

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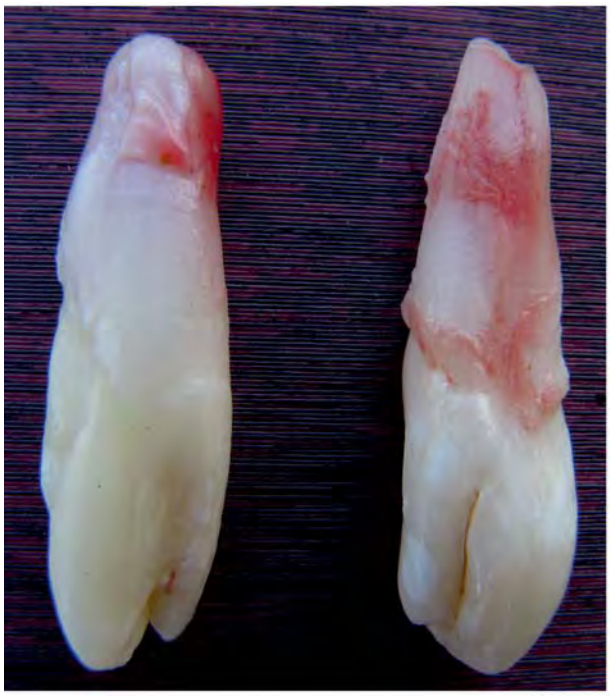

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