

Fig. 1 : CT Scan showing large cyst arising from Fig. 2 : Large cystic lesion involving left vallecula Fig. 3 : Large cystic lesion involving left lateral vallecula.

and lateral pharyngeal wall.

Recently a modified working classification of laryngeal cysts has been proposed by Ramesar et al<sup>7</sup> (1988), based on histology. However because this classification is based on histology it is largely retrospective and it diverts attention from the site, which is the primary factor in management.

Treatment of these cysts depends on their size and location. Tracheostomy is necessary if standard endotracheal intubation cannot be performed. Puncture of the cysts to allow decompression adds the risk of pulmonary aspiration; this procedure is not recommended prior to securing an airway. Many of these cysts may be treated by utilizing direct laryngoscopy<sup>9</sup>. incision with microsurgical instruments or removal with a nasal snare<sup>9</sup> or tonsillotome.<sup>1</sup> Giant obstructing cysts may be removed by the trans oral route, but may require a lateral pharyngotomy<sup>1</sup> or laryngofissure<sup>1,2</sup> for complete excision.

This case of vallecula cyst was marsupilized under general anesthesia without tracheostomy with nasotracheal intubation and follow up was uneventful.

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#### Address for correspondence

Dr. Naresh Nathani, Senior Resident, Department of ENT, SMS Medical College, Jaipur

**Short Communications** 

# **CANCRUM ORIS: A CASE REPORT**

#### Sudhakar Vaidya,\* Anjana Agrawal,\*\* VK Sharma\*\*\*

Abstract: Cancrum Oris is acute gangrenous stomatitis of oral and para-oral structures in age group between 2 to 16 years particularly in sub-Saharan Africa with high morbidity and mortality. Exact bacteriology is uncertain. Acute necrotising gingivitis and oral herpetic ulcers are considered to be antecedent lesions. The treatment of cancrum oris is penicillin, tetracycline, metronidazol and clindamycin, second step of management includes reconstructive surgery for the treatment of the functional and cosmetic problems. The authors are reporting a case of cancrum oris in a 1.5-year-old child.

Keywords: Cancrum Oris, Noma, Acute Gangrenous Stomatitis, Acute Necrotising Gingivitis.

#### **INTRODUCTION**

Cancrum oris (syn:NOMA, Gangrenous Stomatitis) is a rapidly spreading gangrenous condition affecting the soft and hard

tissue of the oral and para- oral structure. However it is very rare now, even in developing countries. It predominantly affects children between 2-16 Years of age. (Ref 1) It usually starts from

Dept. of Otorhinolaryngology, R D Gardi Medical College & Ujjain Charitable Hospital, Ujjain (MP), (India)

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the gingiva and spread to involve the mouth and face. Disabling and disfiguring complications such as ankylosis <sup>(2, 3)</sup> and partial loss of jaws, lips and cheeks are not rare. There is impairment of speech and mastication. The Physical and psychological disabilities of complications in a growing child are all too obvious. This disease has a high mortality rate but with the introduction of antibiotics as a from of therapy, a higher survival rate has been achieved.

Risk factors for cancrum oris are poverty, malnutrition, poor orodental hygiene, deplorable sanitation, substandard accommodation, close residential proximity to live stock and infectious disease like measles, scarlet fever, tuberculosis, malignancy or immunodeficiency.<sup>(4)</sup>

#### CASE REPORT

A one and half year female child belonging to poor economic group and living in sub-standard condition with poor sanitation and live stock presented with history of ulceration in gingiva and palate since 7 days in December 2004. It was a sudden onset, associated with fever and dysphagia. The palatal ulcer progressed to nose and cheek to the stage shown in the clinical photograph.(Fig:1)

#### **ON EXAMINATION**

There was bluish discoloration with black spots (gangrene) seen in both cheek. Size was about 3.5 cms into 4 cms in right cheek and 4 cms into 4 cm in left cheek. There was a necrotising ulcer of about 3 cms into 3 cms present in palate involving whole palate with gangrenous changes. It extended to maxilla and cheek. There was purulent discharge from nostrils with congestion and oedema of nasal mucosa. The child was anaemic, malnourished, febrile, had difficulty in deglutition, had signs of dehydration and had a toxic look.

Investigations done were haemogram, gram staining and culture and sensitivity test from the palatal lesion, HIV, VDRL and Australia antigen and plain radiograph of chest.

Hb:8gm	per 100cc
Total count	17000/cumm
P	70%
E	02%
В	01%
L	27%
ESR	by westergren method 22mm 1 hour

Urine routine & microscopic: no abnormality detected Gram staining and Culture & sensitivity tests from palatal lesion: Fusobacterium necrophorum and pseudomonas were isolated. HIV, VDRL and Australia antigen tests were normal. X-ray chest was normal.

Provisional diagnosis of cancrum oris was made which was confirmed by gram staining and bacteriological culture. Systemic antibiotic crystalline penicillin and metrogyl were started. Intravenous fluids, antipyretics, anti-inflammatory drugs supported the management and as patient was anaemic, blood



Fig 1: Clinical photograph

transfusion was given. In spite of all treatment and supportive management patient condition deteriorated on fifth day of admission and parents were not ready for further treatment and took the patient to home against the medical advice.

#### DISCUSSION

Cancrum oris is an infectious disease, which destroys the orofacial tissues and other neighboring structures in its fulminating course. The exact bacteriology is uncertain, although Vincent's spirochaete and fusiform bacillus in symbiosis have been considered to be the actual cause of the condition (Ref. 5). Acute necrotising gingivitis and oral herpetic ulcers are considered to be antecedent lesions and Fusobacterium necrophorum and Provotella intermedia, alpha- hemolytic streptococci and actinomyces species have been isolated from the lesions. Other organisms, which have been isolated from the lesions, are Peptostreptococcus micros, Veillonella parvula, Staphylococcus aureus, and Pseudomonas (Ref. 6, 7.) Additional to production of a growth-stimulating factor for Provotella intermedia, Fusobacterium necrophorum displays a classic endotoxin, a dermonecrotic toxin, a cytoplasmic toxin, and a hemolysin. (Ref.4). The disease usually affects children aged 2-16 years particularly, in Sub-Saharan Africa from 1 to 7 cases /1000 population. (Ref.4)

Without appropriate treatment the mortality rate from Noma is 70-90%. (Ref. 4) Nutritional deficiencies are said to be one of the causative factor. In a study of Nigerian children, significantly reduced plasma concentration of zinc (< 10.8 micro mol/L), retinol (< 1.05 micro mol/L), ascorbate (< 11 micro mol/L), and the essential amino acids, with prominently increased plasma and saliva levels of free cortisol compared with their healthy counterparts was documented. (Ref. 8).

Most of the strains of F.necrophorum and P. intermedia were found to be sensitive to clindamycin, tetracycline, metronidazol and penicillins except one strain of P intermedia, which showed resistance to penicillin. (Ref. 7) The morbidity rate is also high with functional disturbances and disfigurement. The treatment of noma is a two-step procedure. The first step includes treatment antibiotic treatment of the acute infection, correction of electrolyte and nutrition imbalance. Further attention has to be given to treatment of concomitant diseases such as malaria, typhoid, tuberculosis, HIV and other virus diseases. The second step includes reconstructive surgery for the treatment of the functional and cosmetic problems. Beside the defects of soft tissue and the facial skeleton, jaw ankylosis a severe problem is caused by the sequelae of noma. Reconstructive procedures are generally required at some subsequent stage and it is usually multi stage procedure .The surgical treatment of the sequelae in the patients affected by cancrum oris is possible, if the surgeon carefully evaluate each patient individually choosing simple, safe, sound and satisfactory technique. (Ref. 1.) There have been reports of one stage reconstruction of defects caused by Noma, which is both cost effective and safe. (Ref. 9).

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Address for correspondence: Dr. Sudhakar Vaidya MBBS, D.N.B. (ENT), D.L.O. Associate Professor Otorhinolaryngology R D Gardi Medical college & Ujjain Charitable Hospital Ujjain (MP), (India)

## **Short Communications**

# NASAL GLIOMA : A CASE REPORT

## J.K. Sharma,\* S.K. Pippal,\*\* Yatin Sethi,\*\*\* Shitij Arora,\*\*\* S K Raghuwanshi.\*\*\*

<u>Abstract:</u> Nasal glioma, also known as glial heterptopia or the occurrence of isolated non - teratomatous glial tissue is a rare and benign congenital defect. This condition is diagnosed usually at birth time and requires early treatment to prevent facial deformations. We report here a case of extranasal glioma that was diagnosed and treated at the department of ENT, Gandhi Medical College, Bhopal. We emphasize on the developmental theory proposed as the etiology and discuss the clinical aspects, treatment and follow up.

Key words: Congenital midline masses, Glial fibrillary acid protein.

## INTRODUCTION

Nasal gliomas are rare, benign congenital masses more accurately referred to as sequestered glial tissue. Habitually diagnosed right after birth may be detected in the neonatal period in 60% cases<sup>(1)</sup> These are one of the congenital midline masses, a category which also includes nasal dermoids and encephaloceles<sup>(2)</sup>. These disorders are clinically important because of their potential for connection to the central nervous system. Nasal gliomas may be extranasal or intranasal. Extranasal gliomas usually appear at the root of nose<sup>(17)</sup>

We describe here a case of an 11 month old female diagnosed as extranasal glioma at the tip of nose and subsequently treated in our service and to perform a literature review.

## CASE REPORT

This 11 month old female was brought for evaluation of a swelling at the tip of nose, just lateral to the midline on the left side.(figure 1). Detailed history yielded that the swelling was congenital, gradually increased in size to the present. The delivery was uneventful and the family history was unremarkable.

Physical examination revealed an external bulging about 1x1 cm in size, located on the nasal tip just left to the midline, firm, non pulsatile ,covered with normal healthy skin with no evidence of telengiectasias. There was no change in the size of swelling oncrying and the FURSTENBERG'S TEST was negative (no change in the size of the swelling with bilateral compression of the internal jugular veins). Rhinoscopy revealed an intranasal

\*Professor and Head, \*\*Associate professor, \*\*\*Residents, Department of ENT, Gandhi Medical college, Bhopal (India)

