A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY,

CHENNAI - 600032

In Partial Fulfillment of the Regulations for the Award of the Degree of

M.S. IN OTO-RHINO-LARYNGOLOGY

DONE BY

DR KISHORE KUMAR P

REG NUMBER: 220420500502

DEPARTMENT OF OTORHINOLARYNGOLOGY

PSG INSTITUTE OF MEDICAL SCIENCE AND RESEARCH

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI, TAMILNADU

2020-2022

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M.S. IN OTO-RHINO-LARYNGOLOGY

GUIDE

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CHENNAI, TAMILNADU

2020-2022

CERTIFICATE

This is to certify that the thesis entitled "**A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE"** is a bonafide work of **Dr. KISHORE KUMAR P** done under the direct guidance and supervision of **Dr. S.PALANINATHAN M.S,** in the department of Otorhinolaryngology, PSG Institute of Medical Sciences and Research, Coimbatore in fulfilment of the regulations of **The Tamil Nadu Dr.MGR Medical University, Chennai** for the award of M.S. degree in ENT.

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DECLARATION BY THE CANDIDATE

I solemnly declare that this dissertation "**A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE"** was prepared by me at PSG Institute of Medical Sciences and Research, Coimbatore, under the guidance and supervision of **Dr.S.PALANINATHAN M.S,** Professor and HOD, Department of Otorhinolaryngology, PSG Institute of Medical Sciences and Research, Coimbatore. This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of M.S. Degree in ENT.

Dr. KISHORE KUMAR P

CERTIFICATE BY THE GUIDE

This is to certify that **"A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE"** is a bonafide work done by **Dr KISHORE KUMAR P,** Post graduate student, Department of Otorhinolaryngology, PSG Institute of Medical Science and Research, Coimbatore, under my guidance and supervision in partial fulfillment of rules and regulations of **The TamilNadu Dr. M.G.R Medical University, Chennai** for the award of M.S. Degree in ENT during the academic period from 2020 to 2022.

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This is to certify that this dissertation work entitled **"A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE"** of the candidate **Dr. KISHORE KUMAR P** with Registration Number **220420500502** for the award of **M.S** degree in the branch of **ENT**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **8 percentage** of plagiarism in this dissertation.

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Institutional Human Ethics Committee
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Recognized by The Strategic Initiative for Developing Capacity in Ethical Review (SIDCER, WHO)
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EC-CT-2018-0055

Ref. No.: PSG/IHEC/2021/Appr/Exp/049

March 22, 2021

To
Dr Kishore Kumar P
Postgraduate
Department of E N T
PSG IMS & R
Coimbatore
Guide: Dr Palaninathan S

Ref: Project No. **21/056**

Dear Dr Kishore Kumar,

Institutional Human Ethics Committee, PSG IMS&R reviewed and discussed your application dated 02.03.2021 to conduct the research study entitled "*A retrospective study on ENT complications in COVID-19 patients in a tertiary care center, Coimbatore"* during the IHEC meeting held on 12.03.2021.

The following documents were reviewed and approved:

1. Project submission form
2. Study protocol (Version 1 dated 02.03.2021)
3. Application for waiver of consent
4. Confidentiality statement
5. Data collection tool (Version 1 dated 02.03.2021)
6. Permission letter from Director – Research & Innovation
7. Current CVs of Principal investigator, Co-investigators
8. Budget

The following members of the Institutional Human Ethics Committee (IHEC) were present at the expedited review meeting held on 12.03.2021 between 10.00 am and 11.00 am:

Sl. No.	Name of the Member of IHEC	Qualification	Area of Expertise	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
1	Dr Rajani Sundar (Chairperson, IHEC)	MD, DA	Clinician	Female	No	Yes
2	Dr S Karthikeyan (Member – Secretary, IHEC)	MD	Epidemiologist, Ethicist	Male	Yes	Yes
3	Dr A Jayavardhana	MD	Clinician, Paediatrics	Male	Yes	Yes
4	Mrs M Nirmala (Alt. member-Secretary, IHEC)	M Sc	Nursing	Female	Yes	Yes

The study is approved in its presented form for the stated sample size. The decision was arrived at through consensus. Neither PI nor any of proposed study team members were present during the decision making of the IHEC. The IHEC functions in accordance with the New Drugs and Clinical Trials Rules, 2019. The approval is valid until one year from the date of sanction. You may make a written request for renewal / extension of the validity, along with the submission of status report as decided by the IHEC.

INSTITUTIONAL HUMAN ETHICS COMMITTEE
PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH
COIMBATORE-641004.

Following points must be noted:

1. IHEC should be informed of the date of initiation of the study
2. Status report of the study should be submitted to the IHEC every 12 months
3. PI and other investigators should co-operate fully with IHEC, who will monitor the trial from time to time
4. At the time of PI's retirement/intention to leave the institute, study responsibility should be transferred to a colleague after obtaining clearance from HOD, Status report, including accounts details should be submitted to IHEC and extramural sponsors
5. In case of any new information or any SAE, which could affect any study, must be informed to IHEC and sponsors. The PI should report SAEs occurred for IHEC approved studies within 7 days of the occurrence of the SAE. If the SAE is 'Death', the IHEC Secretariat will receive the SAE reporting form within 24 hours of the occurrence
6. In the event of any protocol amendments, IHEC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. Variation in the proposed sample size
   c. Alteration in the budgetary status should be clearly indicated and the revised budget form should be submitted
   d. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval
   e. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented
   f. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IHEC and only then can they be implemented
   g. Any deviation-Violation/waiver in the protocol must be informed to the IHEC within the stipulated period for review
7. Final report along with summary of findings and presentations/publications if any on closure of the study should be submitted to IHEC

Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

Thanking You,

Yours Sincerely,

Karthikeyan 22/3/2021

Dr S Karthikeyan
Member-Secretary
Institutional Human Ethics Committee

SECRETARY
PSG IMS&R
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INSTITUTIONAL HUMAN ETHICS COMMITTEE

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EC-CT-2018-0055

Ref.: PSG/IHEC/2021/Qry/Exp/035

March 13, 2021

Dr Kishore Kumar R
Postgraduate
Department of E N T
PSG IMS & R
Coimbatore

Dear Dr Kishore Kumar,

The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal and discussed your request for approval for the study proposal titled:

"A retrospective study on ENT complications in COVID-19 patients in a tertiary care center, Coimbatore"

The following are the suggestions / recommendations made by the reviewer:

1. Please provide justification for sample size
2. Kindly clarify how patients with ENT complications will be identified
3. Kindly submit flowchart of the study
4. Please clarify what are the benefits from the study

Decision: Approval pending minor modifications

Yours sincerely

13/3/2021

Dr S Karthikeyan
Member – Secretary
Institutional Human Ethics Committee

SECRETARY
INSTITUTIONAL HUMAN ETHICS COMMITTEE
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COIMBATORE-641004.

ACKNOWLEDGEMENT

I would like to express my deep sense of gratitude to my respected teacher and guide **Dr. S. PALANINATHAN M.S**., Professor and Head, Department of Otorhinolaryngology for his valuable advice and guidance. I am very much thankful for his constant inspiration, timely suggestions and structural support in carrying out this study without which this study would have not been completed.

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I also extend my sense of gratitude to all my colleague post graduates, my CRRIs and my friends for their constant help and cooperation during the study. I also extend my thanks to all the staff of Department of Otorhinolaryngology for their help in carrying out the study.

I would like to take this opportunity to show gratitude to my parents and my friends for their never ending support in completing this thesis.

Finally I whole heartedly thank all the patients involved in the study without which my study would not have been possible.

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INTRODUCTION

Severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2) is initially called as the 2019 novel Coronavirus (2019-nCoV). Coronaviruses belong to Coronaviridae family which are further divided into alpha (α-CoV), beta (β-CoV), gamma (γ-CoV), and delta (δ-CoV) coronaviruses. The alpha and beta coronaviruses commonly infect mammals .The β-CoVs are further subdivided into different lineages (A, B, C, and D lineages). SARS-CoV-2 is grouped in lineage B, which has nearly 200 published virus sequences [1]. SARS-CoV-2 (mainly beta-CoV of group 2B) is the causative agent of serious life-threatening disease called as coronavirus disease of 2019 (COVID-19) [2]

It was first discovered in Wuhan, China as the WHO reported the first case on December 31, 2019. [2]

However, some experts believe that the earliest case of COVID-19 was detected as early as November 17, 2019.[2] Since then COVID-19 has dramatic spread rapidly throughout the world and has reached pandemic proportions affecting all countries. WHO declared the outbreak a Public Health Emergency on January 30, 2020 and on March 11, 2020, COVID-19 was declared a global pandemic disease.[3]

It has been found that pulmonary epithelium is the primary target of the virus. The receptor-binding domain (RBD) S-protein of SARS-CoV-2, mainly determines the entry of the virus into human host cells through the Angiotensin converting enzyme 2 (ACE2) receptor. [4]

It is believed that SARS-CoV-2 transmission to humans occur by three methods

(a) Droplets in short range,

(b) Fomite (contact) transmission and

(c) Aerosol in long range. [5]

Droplets are produced when an infected person coughs, sneezes or talks loudly and can be infective to others in close contact up to a distance of 4 to 6 feet (1.8 m).[6] The virus also spreads by fomites when an infected person contaminates the object and gets picked up by a new person who gets infected. According to a study, SARS-CoV-2 remains viable in aerosols for minimum 3 hours, and stable on most objects like plastic (72 h), stainless steel (48 h), cardboard (24 h) and copper (4 h) for long durations.

Nearly 80% of COVID infections are mild to moderate that includes pneumonia and non-pneumonia cases, about 13.1% develop severe disease, while a further 6.1% develop serious disease requiring intensive hospital care support.[7]

The COVID-19 is presented mainly by lower respiratory tract related symptoms such as fever, cough, dyspnea and chest tightness that could progress rapidly to acute respiratory distress syndrome (ARDS).

However, COVID-19 also causes different upper respiratory tract related symptoms including nasal congestion, sore throat and smell dysfunction.

While the pathophysiology of the virus is still under investigation, new symptomatic manifestations and complications of the disease continue to be identified and described in medical literature.

This study aims to characterize long lasting symptoms of COVID-19 recovered patients presenting to our tertiary care hospital in Coimbatore.

Early identification of these highly sensitive conditions is key to allow for optimal treatment and improved outcomes, to avoid mortality and morbidity.

REVIEW OF LITERATURE

Menni et al.[8] found that loss of smell and taste were encountered in 59% of COVID-19 positive patients compared to 18% of those with COVID-19 negative test. They suggested that a combination of loss of smell and taste, fever, persistent cough, fatigue, GIT symptoms is a predictive of COVID-19 positive test.

Varia et al.[9] performed objective smell and taste tests on 72 patients with positive PCR for COVID-19 and with no previous history of smell and taste dysfunction. They found that 73.6% of patients had smell or taste dysfunction during the course of the COVID-19 with 14.4% had isolated olfactory dysfunctions.

Auditory complication due to coronavirus is little mentioned in the literature. In a previous report on other coronavirus infection [10], brainstem involvement was observed and the neuro-auditory problem is a possiblity.

While in the study of Mustafa [11], COVID-19 infection could have deleterious effects on cochlear hair cell functions despite being asymptomatic as reduction of high frequency pure-tone thresholds as well as the TEOAE amplitudes were detected.

According to Kim et al.[12] the duration of endotracheal intubation is independently associated with aspiration and the duration of intubation positively correlates with the degree of dysphagia, which may help in identifying patients requiring swallow examination.

Lanjuan Li academician and her colleagues who have accumulated experience with severe COVID-19 treatment, reminded clinicians to focus on fungal infections, especially in severely ill or immune compromised ones[13].

In addition to hyperglycemia and steroid use, COVID-19 infection with possible alteration in iron metabolism may have predisposed to mucormycosis (Lammaert et al., 2012, Kentaro et al., 2021).[14]

According to Lodigiani et al [15], A risk factor for epistaxis in covid population is the use of anticoagulant drugs. All patients who were receiving LMW heparin for therapy according to their individual risk factors and local policy are at risk of epistaxis.

According to Glass et al [16] and rees et all [17], In COVID-19 patients who were never intubated, one mechanism for vocal cord palsy would be the invasion of nerve fibers (peripheral or cranial nerves) by the virus using the same mechanism as seen in alveolar cells and destroying them .Another possible mechanism would be the inflammatory response of the host immune system against the peripheral and cranial nerves.

AIMS AND OBJECTIVES

1. To determine different Otorhinolaryngology complications reported in Covid 19 positive patients in our tertiary care centre, Coimbatore.
2. To estimate incidence, age and gender distribution, risk factors, treatment, follow up and mortality status of study participants.

JUSTIFICATION OF THE STUDY

The available data on the Ear Nose Throat (ENT) complications of COVID-19 patients is sparsely published. To the best of our knowledge, no previous review study to collect and describe the ENT complications in COVID-19 positive patients was reported.

The current study discusses the different Otorhinolaryngology (ORL) complications those were reported in COVID-19 positive patients who presented to our tertiary care centre, Coimbatore.

MATERIALS AND METHODS

STUDY TYPE: Retrospective Descriptive Study

STUDY PERIOD: August 2020 to June 2021

STUDY SETTINGS: Department of Otorhinolaryngology, COVID Review clinic, PSG Institute of Medical Sciences and Research, Coimbatore

STUDY POPULATION: Covid 19 confirmed treated patients attending our hospital with ENT COMPLICATIONS OR LONG LASTING ENT SYMPTOMS

SAMPLE SIZE: 100

INCLUSION CRITERIA:

- All COVID 19 confirmed patients with ENT COMPLICATIONS in the age of 18 years to 65 years.

EXCLUSION CRITERIA

- Infants toddler and adolescence upto age of 18 years and adults above 65 years.

METHODOLOGY

After obtaining ethical committee approval, this study will be carried out in total patients with laboratory confirmed SARS CoV 2 infection who attended review clinic and ENT OPD, PSG Institute of medical sciences and research, Coimbatore, from August 2020 to June 2021.

We identified 100 patients who developed spontaneous ENT complications like long lasting Anosmia, Septal abscess, Epistaxis, RhinoNasal Mucormycosis, Voice change for study.

After obtaining the patient information from MRD files and filling it systematically, careful history, examinations and investigations were studied regarding incidence, associated risk factors, age and gender distribution of ENT complications and same were recorded and analysed.

FLOWCHART OF THE STUDY

All Covid confirmed treated patients with ENT complications attending the Review clinic and ENT OPD in our hospital satisfying inclusion and exclusion criteria.

History, examination and investigations, findings as in variables recorded from MRD files and filling it systematically

Analysis of findings and Data will be recorded

VARIABLES RECORDED

NAME

AGE

GENDER

OCCUPATION

COMPLAINTS AND DURATION OF ILLNESS

PAST HISTORY

PERSONAL HISTORY

FAMILY HISTORY

GENERAL EXAMINATION

ENT EXAMINATION

- Complete head and neck examination
- Ear Nose Throat examination
- VII Cranial nerve examination
- Audiogram (if done)
- Imaging (MRI and HRCT if done)
- Complete blood counts, ESR, if Diabetic patient-Extended diabetic profile

BENEFITS FROM THE STUDY

This study aims to characterize long lasting symptoms of COVID-19 recovered patients presented to our tertiary care centre, Coimbatore. Early identification of these highly sensitive conditions is key to allow for optimal treatment and improved outcomes, to avoid mortality and morbidity.

EPISTAXIS

- Bleeding through the nose is called epistaxis.
- It is a common ENT Emergency and is seen in all age groups. Epistaxis is a sign and not a disease and attempt should be made to find the cause.

Blood Supply of Nose:

- Nasal septum and the lateral nasal walls are supplied by both external and internal carotid systems.

Nasal Septum

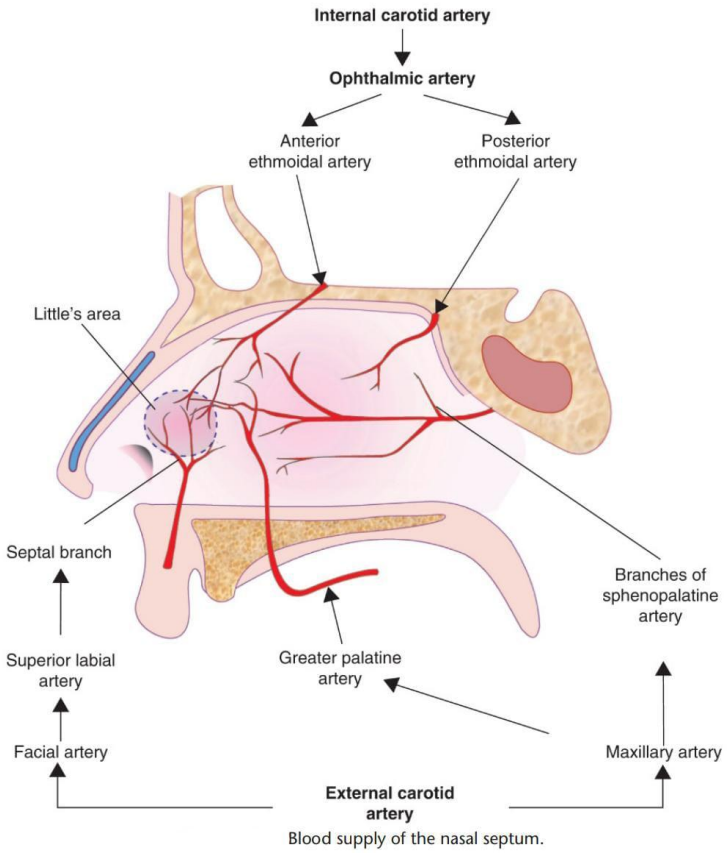


FIGURE 1

Internal Carotid System

1. Anterior ethmoidal artery
2. Posterior ethmoidal artery } Branches of ophthalmic artery

external carotid system

1. Sphenopalatine artery (branch of maxillary artery) is the most important arterial blood supply to the nasal cavity. It enters nasal cavity through the sphenopalatine foramen and divides into posterior septal and posterior lateral branches.[18]
2. Septal branch of greater palatine artery (branch of maxillary artery).
3. Septal branch of superior labial artery (branch of facial artery).

Lateral Wall

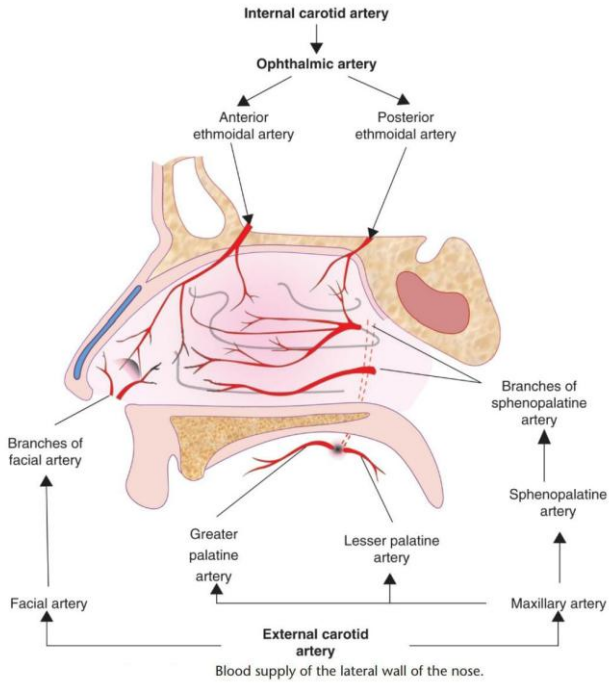


FIGURE 2

Internal Carotid System

1. Anterior ethmoidal
2. Posterior ethmoidal } Branches of ophthalmic artery

The posterior ethmoidal Branch of ophthalmic artery is smaller than the anterior ethmoidal artery and is present nearly in 80% of individuals. [19]

External Carotid System

1. Posterior lateral nasal branch → From sphenopalatine artery
2. Greater palatine artery → From maxillary artery
3. Nasal branch From infraorbital branch of maxillary artery
4. Branches from facial artery to nasal vestibule

Sites Of Epistaxis

Little's Area

On the anterior inferior part of septum an arterial plexus was identified as a common site of haemorrhage by James Little in 1879 and the same plexus was described one year later by Kiesselbach.[20] As a result of these descriptions, the area most frequently involved in epistaxis is known as Little's area or Kiesselbach's plexus.

Four arteries—anterior ethmoidal, septal branch of superior labial of facial artery, septal branch of sphenopalatine and the greater palatine, anastomose here to form a

vascular plexus. This area is exposed to the drying effect of inspiratory current and to finger nail trauma, and is the most common site for epistaxis in children and young adults.

Woodruff's Plexus

Woodruff (1949) described the plexus of prominent blood vessels lying under posterior end of the inferior turbinate.[21] It is a plexus of veins. It is a site of posterior epistaxis in adults.

Surgical anatomy of the sphenopalatine foramen

The sphenopalatine foramen is the transit point for the majority of arterial supply of the nasal cavity. Lateral to the foramen lies the pterygopalatine fossa. The foramen is formed by a U-shaped notch in the perpendicular plate of the palatine bone which is closed posterosuperiorly by the greater wing of sphenoid bone. The foramen transmits the sphenopalatine artery, vein and the naso palatine nerve (maxillary division of the trigeminal nerve). Clinically this foramen is the key area to the procedure of endonasal endoscopic sphenopalatine artery ligation (ESPAL). a small bony projection lies anterior to the foramen in 96% of cases, this landmark is called the crista ethmoidalis [22]. This bone is variable in size but consistent in the position anterior to the artery and so it is a important landmark for the endoscopic surgeon.

Causes of Epistaxis

A. Local Causes

Nose:

1. Trauma: digital trauma, intranasal surgery, fractures of middle third of face and base of skull, hard- nasal blowing, violent sneeze.
2. Infections
   (a) Acute: Viral rhinitis, sino nasal diphtheria, acute sinusitis.
   (b) Chronic: All crust-forming diseases, e.g.rhinitis sicca, atrophic rhinitis, granulomatous lesion of the nose, e.g., tuberculosis, syphilis, rhinosporidiosis, wegeners granulomatosis
3. Foreign bodies
   (a) Nonliving: Any neglected foreign body (stone, beads, paper), rhinolith.
   (b) Living: Maggots, leeches.
4. Neoplasms of nose and paranasal sinuses.
   (a) Benign: capillary Haemangioma, inverted papilloma.
   (b) Malignant: Carcinoma or sarcoma or lymphoma
5. Atmospheric changes: High altitudes, sudden decompression (Caisson disease).
6. Deviated nasal septum with spur

Nasopharynx:

1. Adenoiditis.
2. Juvenile nasopharyngeal angiofibroma.
3. Malignant tumours.

B. General Causes

1. Cardiovascular system: Hypertension, arteriosclerosis, mitral stenosis, pregnancy (due to hormonal changes).
2. Disorders of blood and blood vessels: Aplastic anaemia, leukaemia, thrombocytopenic purpura, Von Willebrand disease, haemophilia, Christmas disease, scurvy and hereditary haemorrhagic telangectasia.
3. Liver disease: Hepatic cirrhosis (deficiency of factor II, VII, IX and X) and vitamin K deficiency
4. Kidney disease: Chronic nephritis.
5. Drugs: Excessive use of analgesics like salicylates (as for joint pains or headaches), anticoagulant and antiplatelet therapy (for heart disease).
6. Mediastinal compression: Tumours of mediastinum (increased venous pressure in the nose).

7. Acute general infection: Influenza, measles, chickenpox, whooping cough, rheumatic fever, infective endocarditis, typhoid, pneumonia, malaria and dengue fever.

8. Vicarious menstruation (epistaxis occurring during the time of menstruation).

C. Idiopathic

Classification OF Epistaxis:

Adult or childhood Epistaxis

- There is a bimodal distribution in the age of onset of epistaxis. Childhood (less than 16 years) or adult (greater than 16 years).

Primary or secondary

- Between 70% to 80% of cases of epistaxis are idiopathic, spontaneous bleeding occurs without any proven precipitating or causal factor. This type of bleeding can be classified as primary epistaxis.[23]

- Epistaxis due to a clear and definite cause such as trauma, surgery or anti-coagulant overdose can be classified as secondary epistaxis

Anterior and Posterior Epistaxis:

In anterior epistaxis, blood flows out through the front of nose with the patient in sitting position.

In posterior epistaxis, the blood flows back into the throat. Patient may swallow it and later have a “coffee-coloured” vomitus. This may wrongly be misdiagnosed as haematemesis many times.

ADULT PRIMARY EPISTAXIS

Demography

The condition can occur at any part of age but is mainly a disease of the elderly population. Between 7% to 14% of adults have epistaxis at some time or other, but only 6% of cases are seen by ENT specialists.[24,25]

The peak presentation is in the sixth decade and there is a slight male predominance.[26,27] After head and neck cancer, epistaxis is the frequent cause of mortality in ENT patients.

AETIOLOGY

Chronobiology

The frequency of admission of epistaxis patients is greatest in the autumn and winter months.[28,29] This seasonal variation may be due to fluctuations in environmental temperature and humidity.[30] This chronobiological rhythm is also observed at the circadian level where onset of epistaxis and hospital admission show a biphasic pattern with peaks in the morning and late evening.[31,32]

Non-Steroidal Anti-Inflammatory Drugs (NSAID)

Adult type epistaxis is associated with the use of NSAID. NSAID used include prescribed and self administered medication especially aspirin. The action of non-steroidal anti-inflammatory drugs is mediated via anti-platelet aggregation effect due to altered platelet membrane structure.[33–35]

Alcohol

Epistaxis patients are more likely to have consumed alcohol within 24 hours of hospital admission. The use of alcohol by epistaxis patients is associated with a prolongation of the bleeding time and coagulation factor activity despite normal platelet counts. Effects of NSAID and alcohol mirror those reported in subarachnoid haemorrhage.[36, 37]

Hypertension

A number of studies have failed to show a causal relationship between hypertension and epistaxis.[38] Even if not causal, elevated blood pressure is observed in most of epistaxis patients. This apparent hypertension in acute admissions may be a result of anxiety and the invasive techniques used to control the bleeding.

Septal Abnormalities

1% and 80% of the population have a significant Septal abnormalities.[39] Therefore the perceived association between epistaxis and septal abnormalities can be coincidence. There is no clear evidence of an association between septal abnormalities and adult epistaxis

Management

First Aid

Majority of the time, bleeding occurs from the Little's area and can be easily managed by pinching the ala nasi (the Hippocratic technique) with thumb and index finger for about 5 min. This compresses the vessels of the Little's area.[40]

In Trotter's method patient is made to sit, leaning a little forward over a kidney tray or washbasin to spit any blood and breathe quietly from the mouth.

Cold compresses can be applied to the nasal dorsum to cause reflex vasoconstriction.

Direct or Indirect Therapies

Treatment may be divided into direct (bleeding point specific therapies) or indirect treatments.

Indirect therapies are those that do not require identification of the bleeding point.

Direct treatments are theoretically superior and, therefore, a true search for the bleeding vessel should always be undertaken.[41]

Direct Therapy

Cauterization:

This is very useful in anterior epistaxis when bleeding point has been identified. The area is first anaesthetized topically and the bleeding point cauterized with a silver nitrate or coagulated with electrocautery.

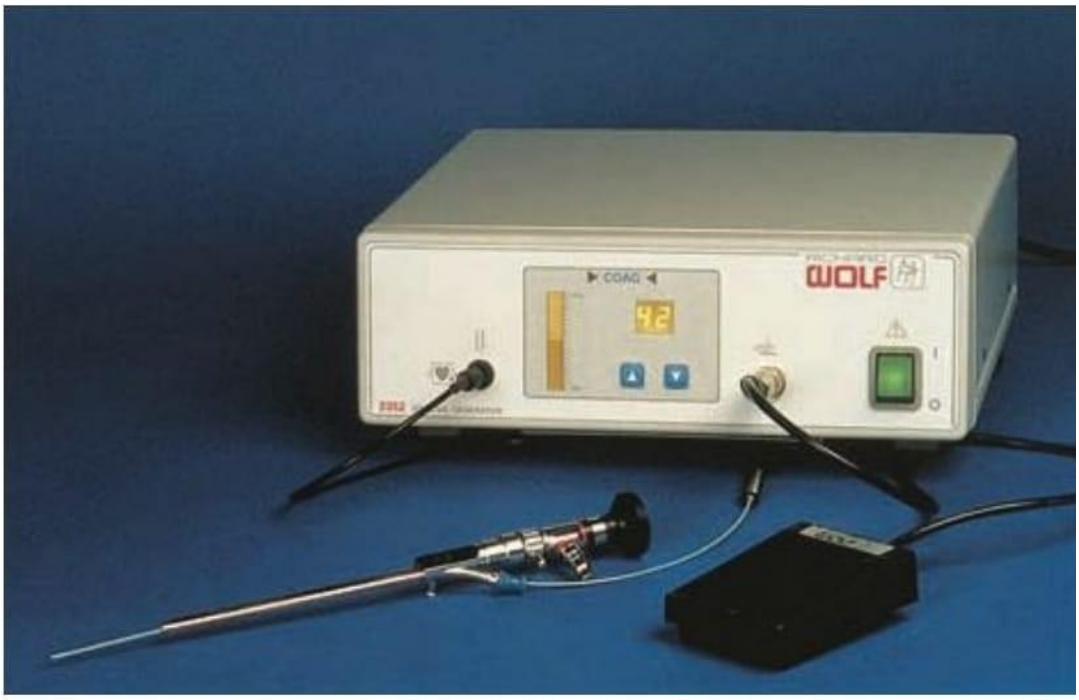


FIGURE 3

Single fibre bipolar unit for direct management of epistaxis

Endoscopy identifies the source of bleeding in posterior epistaxis in over 80% of patients and enables targeted haemostasis of the bleeding vessel using insulated hot wire cautery or bipolar electrodes.[42,43]

Monopolar diathermy should not be used in the nasal cavity as there are chances of blindness due to current propogation.[44]

Indirect Therapy

Anterior Nasal Packing:

If bleeding is profuse or the site of bleeding is difficult to localize, anterior packing can be done.

For this, ribbon gauze soaked with liquid paraffin can be used. About 1 m of gauze (2.5 cm wide in adults and 12 mm in children) is required for each nasal cavity. Few centimetres of gauze are folded upon itself initially and inserted along the floor and then the whole of nasal cavity is packed tightly by layering the gauze from floor to the roof and from before backwards.

Packing can also be done in vertical layers from back to the front.

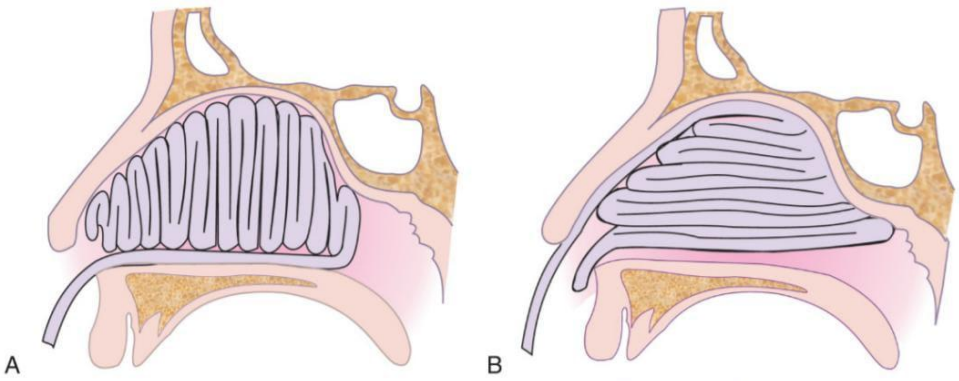
One or sometimes both cavities need to be packed. Once inserted, the packs are left inside undisturbed for between 24 to 72 hours. Re-bleeding with packs in situ is observed in nearly 40% of cases. Complications of packing include toxic shock syndrome, sinusitis, septal perforation, alar necrosis and hypoxia. Hence Packing is usually considered as an indication for antibiotic cover.[45,46]

Posterior Nasal Packing

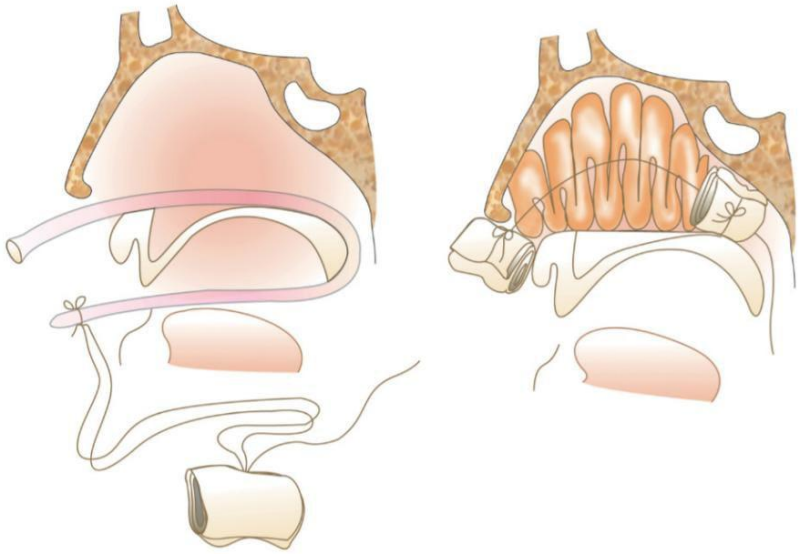
It is done for patients bleeding posteriorly into the throat. A postnasal pack is prepared by tying three silk ties to a piece of gauze rolled into the shape of a cone. A rubber catheter is passed through the nose and its end brought out through the mouth.

Ends of the silk threads are tied to cathether and catheter is withdrawn from nose. Pack, which follows the silk thread, is now packed into the nasopharynx . Anterior nasal cavity is now packed and silk threads tied over a dental roll. The third silk thread is cut short and allowed to hang in the oropharynx. It helps in easy removal of the pack . Patients requiring postnasal pack should be hospitalized.

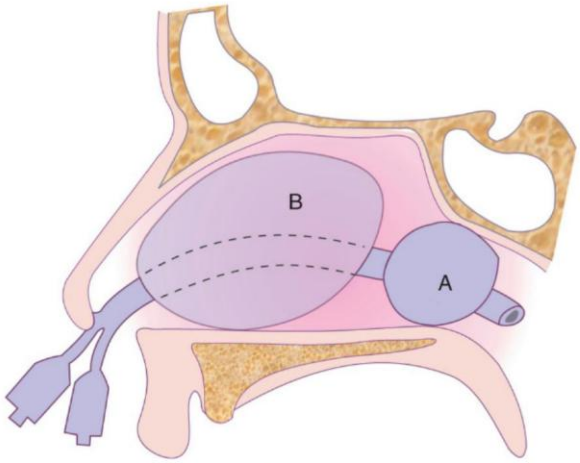
Instead of postnasal pack, a Foley's catheter of size 12–14 F can also be used. After insertion, balloon is inflated with 5–10 mL of normal saline. The bulb is inflated with saline and pulled forward so that choana is blocked with bulb and then an anterior nasal pack can be kept in the usual manner. These days nasal balloons are also available. A nasal balloon has two bulbs, one for the postnasal space and the other for nasal cavity.



Methods of anterior nasal packing. (A) Packing in vertical layers. (B) Packing in horizontal layers.



Technique of postnasal pack.



Epistaxis balloon for posterior epistaxis. Posterior balloon (A) is inflated with 10 mL and anterior balloon (B) with 30 mL. Catheter provides nasal airway.

FIGURE 4

Elevation of Mucoperichondrial Flap and Submucous Resection (SMR) Operation

In patients with persistent or recurrent bleeding from the septum, elevation of mucoperichondrial flap and then repositioning it back helps to cause fibrosis of tissues and constriction of blood vessels. Submucous resection operation can also be done to achieve the same results or remove any bony spur which may be sometimes the cause of epistaxis.[47]

Ligation of Vessels

1. External Carotid:

When conservative measures have failed and bleeding is from the external carotid system and the ligation of external carotid artery above the origin of superior thyroid artery can be done. It is avoided these days in favour of embolization or ligation of more peripheral branches of sphenopalatine artery. Complications include wound infection, haematoma and neurovascular damage. In a study, external carotid artery ligation achieved haemostasis in nearly 14 out of 15 patients.[48]

2. Maxillary artery:

Ligation of maxillary artery is done trans-antrally via anterior (sublabial) or combined anterior and medial (endoscopic) techniques. The mucosa of the posterior wall of the maxillary antrum is then elevated and a window is made through the pterygopalatine fossa. The branches of the internal maxillary artery are identified within the fat of the pterygopalatine fossa and carefully dissected out prior to clipping with

haemostatic clips. The proximal internal maxillary artery, descending palatine and sphenopalatine branches are all clipped and divided.

An endoscopic variation of this technique uses a wide middle meatal antrostomy as an instrument port with a 4 mm endoscope inserted through a small canine fossa antrostomy.[49] Trans-antral ligation achieves control of haemorrhage in 89% of cases and is comparable to embolization.[50]

3. Ethmoidal arteries:

In anterosuperior bleeding above the level of middle turbinate, which is not controlled by packing, anterior and posterior ethmoidal arteries, which supply this region, can be ligated. The vessels are exposed in the medial wall of the orbit by an external ethmoid (Lynch) incision.

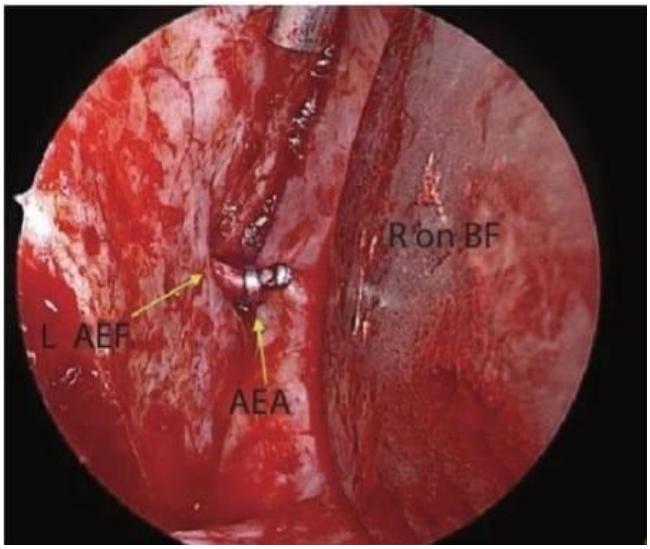


FIGURE 5

Operative field in ligation of anterior ethmoidal artery (left). LPEF: lamina papyracea and anterior ethmoidal foramen, AEA: Anterior Ethmoidal Artery with titanium clips applied.

Transnasal Endoscopic Sphenopalatine Artery Ligation (TESPAL)

An incision is made approximately 8mm anterior to and under cover of the posterior end of the middle turbinate, under general or local anaesthetic .This incision is carried down to the bone and a mucosal flap is elevated posteriorly until the fibroneurovascular bundle arising from the sphenopalatine foramen is identified. The foramen location is signalled by the bony landmark crista-ethmoidalis.[22]

Once the spheno palatine artery is identified, it can be ligated using haemostatic clips and divided or coagulated using bipolar diathermy. Success rates for this procedure in control of refractory posterior bleeding have reached 100% in many cases.[51] Complications including re-bleeding (anastomoses), infection and nasal adhesions are less common.

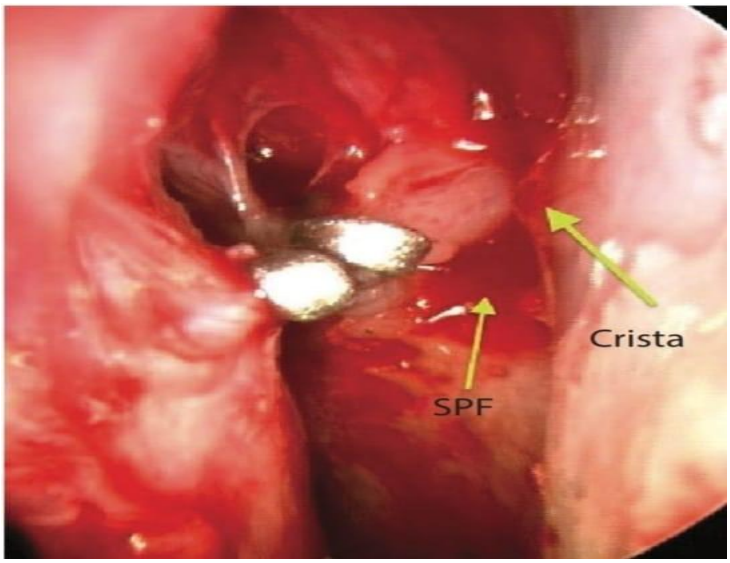


FIGURE 6

Haemostatic clips applied to main trunk of sphenopalatine artery (left) in procedure of ESPAL. Crista: Crista Ethmoidalis, SPF: Sphenopalatine Foramen.

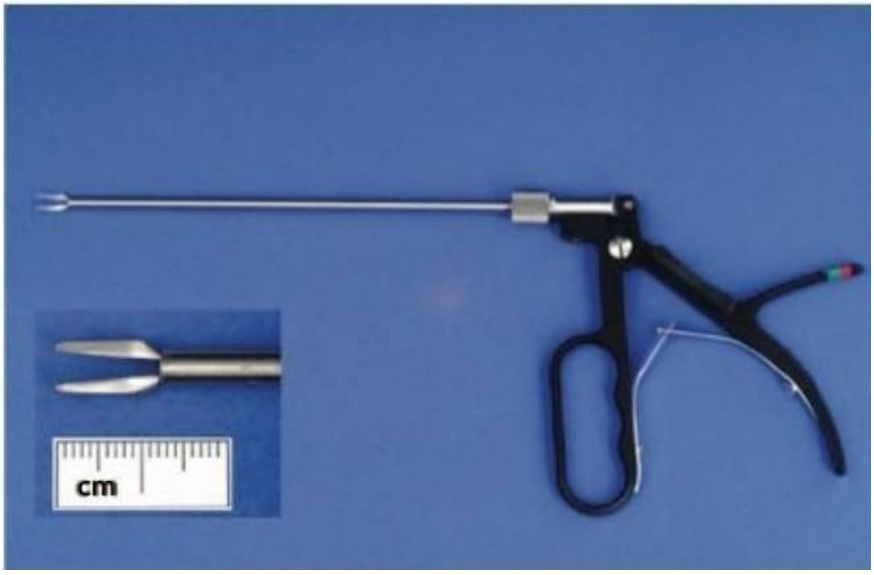


FIGURE 7

Rotatable ligation clip applicators suitable for ESPAL

Embolization

It is usually done by an interventional radiologist through femoral artery catheterization by seldinger technique Under local anaesthetic, to identify the bleeding point and display the nasal circulation.

Internal maxillary artery localized and the embolization is performed with absorbable gelfoam or non absorbable polyvinyl alcohol or coils. Ipsilateral or sometimes bilateral embolizations may be required for unilateral epistaxis because of cross circulation. Embolization is generally a safe procedure but may have potential risks like cerebral thromboembolism leading to stroke, vision loss , haematoma and tissue necrosis at local site. Ethmoidal arteries cannot be embolized.

Embolization under angiographic guidance has shown to control severe epistaxis in nearly 82% to 97% of cases.[52,53]

SEPTAL ABSCESS

Nasal Septal Abscess (NSA) is defined as collection of pus in the subperichondrial and subperiosteal planes of the septum.

NSA common in children. Males are at increased risk than females. This is due to the fact that mucoperichondrium and mucoperiosteum loosely adhere to the septum in children, and males are more involved in violent activities [54].

Etiology:

Trauma - major cause

Post septal surgery

Secondary to sinusitis,

Vestibulitis,

Dental pathologies.[55]

Presentation

- Nasal obstruction - most common symptom
- Pain,
- Fever,
- General tiredness,
- Headache,
- Purulent nasal discharge,

- Cosmetic deformity of the nose such as saddle nose
- Intracranial complications.[54]

Examination

On Anterior Rhinoscopy And Diagnostic Nasal Endoscopy:

septal bulge (bilateral >> unilateral).

The bilateral involvement can be due to septal cartilage resorption as a result of ischemia and necrosis.[56]

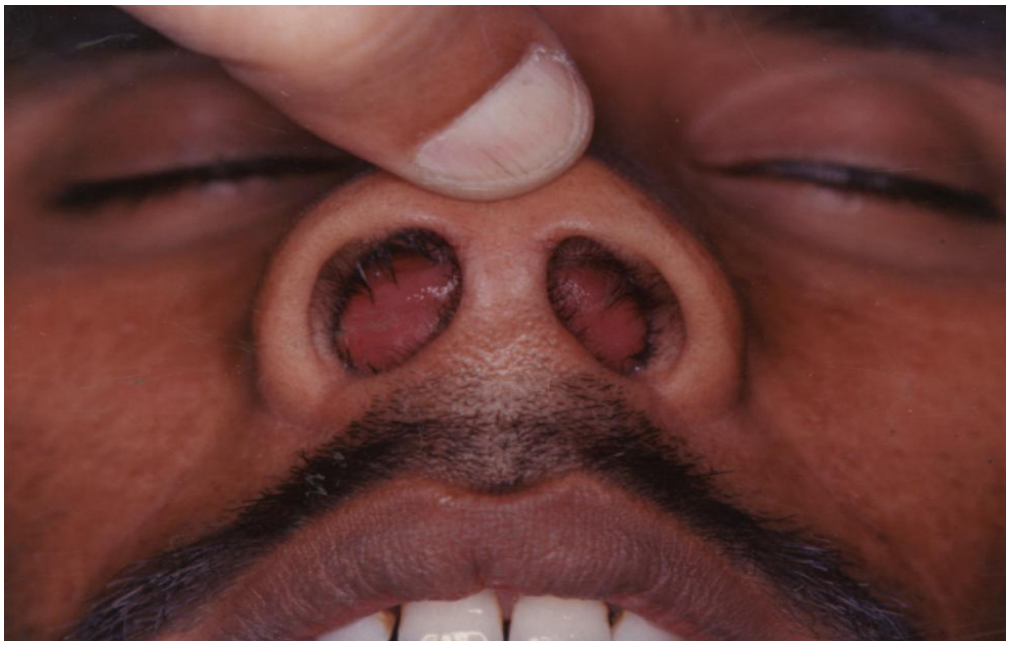


FIGURE 8

Septal Abscess

Organism

Aerobic bacteria – common;

Staphylococcus aureus in adults and

Haemophilus influenza in children.[55]

Complications

The infection can travel to and fro due to the valve-less nature of the facial venous system [57].

Complications of NSA are categorized into:

Local, Orbital, Cranial and Systemic [54].

Local complications

- Sinusitis,
- Vestibulitis,
- Facial cellulitis,

Orbital complication

- Pre-septal cellulitis,
- Orbital cellulitis

Cranial complications

- Meningitis
- Cavernous sinus thrombosis

The intracranial extension can be via the perineural sheath, lymphatics, and hematogenous route [58].

Investigation

CT PNS [59].

Typical appearance on CT examination as a cystic collection of fluid with rim enhancement involving the nasal septum [60].

MRI BRAIN with contrast

For cases of doubtful intracranial extension

Treatment

Early diagnosis proper incision and drainage along-with parenteral antibiotic treatment is the treatment of choice.

Simple needle aspiration has a role only for reducing pressure symptoms but is not curative and ideal.

Apart from Killian‘s incision, horizontal longitudinal or L shaped incision are other options [61].

The drainage of abscess bilaterally is required only if septal cartilage is intact, and pus is collected on both sides, but most of the times, partial destruction of cartilage is present to allow drainage by single incision [62]. Bilateral nasal packing is required postoperatively for a minimum of 48 hours, to prevent recollection.

VOCAL CORD PARALYSIS

Pathophysiology

Majority of laryngeal paralysis result from peripheral nerve damage. As the degree and pattern of neurological impairment varies, vocal cord paralysis is a heterogeneous condition with regards to symptoms, vocal cord position and electromyographic evidence of degree of nerve damage.[63, 64]

Larynx has a high propensity for re-innervation, which appears to be the rule rather than the exception. But, regeneration of the recurrent laryngeal nerve is more problematic than most of the peripheral nerves because it carries a mixed adductor and abductor fibres. Re-innervation is most often non functional and does not provide physiologic motion. [65,66]

The natural propensity for re-innervation accounts for general trend for voice to improve over time in unilateral vocal cord paralysis.

Nerve Supply of Larynx

Motor:

All the muscles of larynx (abductors, adductors or tensors) are supplied by the recurrent laryngeal nerve except the cricothyroid muscle. The cricothyroid receives its innervation from the external branch of superior laryngeal nerve.[67]

Sensory:

Above the level of glottis , larynx is supplied by internal laryngeal nerve—a branch of superior laryngeal, and below the glottis by recurrent laryngeal nerve.

Recurrent Laryngeal Nerve:

Right recurrent laryngeal nerve arises from vagus nerve at the level of subclavian artery, hooks around the artery and then ascends between the trachea and oesophagus in tracheo esophgeal groove.

The left recurrent laryngeal nerve arises from vagus nerve in the superior mediastinum at the level of arch of aorta, loops around it and then ascends into the neck in the tracheo-oesophageal groove. Thus, left recurrent laryngeal nerve has a much longer course which makes it more prone to paralysis compared to the right one.

Superior Laryngeal Nerve:

It arises from inferior ganglion of the vagus nerve below jugular foramen, descends behind internal carotid artery and at the level of greater horn of hyoid bone, divides into external and internal branches.

The external laryngeal nerve supplies cricothyroid muscle while the internal laryngeal nerve pierces the thyrohyoid membrane and supplies sensory innervation to the larynx and hypopharynx.[68]

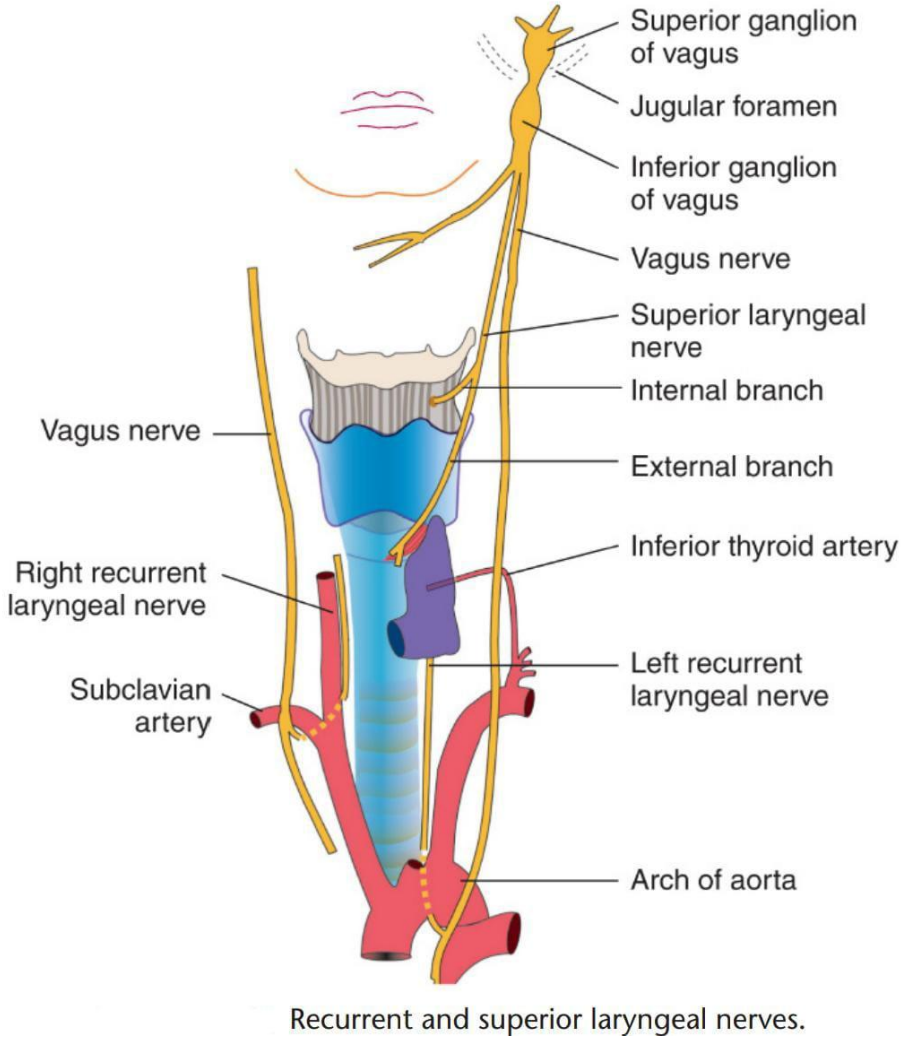


FIGURE 9

Classification of laryngeal paralysis

Laryngeal paralysis can be unilateral or bilateral, and may involve:

1. Recurrent laryngeal nerve.
2. Superior laryngeal nerve.
3. Both recurrent and superior laryngeal nerves (combined or complete paralysis)

Causes of Laryngeal Paralysis

- MECHANICAL - due to surgery or other trauma;
- FUNCTIONAL - due to a range of medical conditions; and
- IDIOPATHIC - In about 30% of cases, cause remains obscure

Laryngeal paralysis affects men more often than women, probably due to the underlying gender distribution of thoracic malignancy.

Left vocal fold is more commonly affected than the right, due to the lengthy course and more profound descent into the thorax of the left recurrent laryngeal nerve, and its consequent increased vulnerability to disease and surgery.

Surgeries and procedures that place laryngeal nerves at risk: [69]

Cervical surgery	Thoracic procedures	Other surgery	Other medical procedures
• Thyroidectomy/parathyroidectomy • Anterior approach to the cervical spine • Carotid endarterectomy • Implantation of vagal nerve stimulator • Cricopharyngeal myotomy/repair of Zenker diverticulum	• Pneumonectomy and pulmonary lobectomy • Repair of thoracic aortic aneurysm • Coronary artery bypass graft • Aortic valve replacement • Esophageal surgery • Tracheal surgery • Mediastinoscopy • Thymectomy • Ligation of persistent ductus arteriosus • Cardiac and pulmonary transplant	• Skull base surgery • Brainstem surgery, or neurosurgery that requires brainstem retraction	• Central venous catheterization • Endotracheal intubation

A number of neurologic conditions remain rare causes of laryngeal paralysis through a central mechanism.

These include

- Lateral medullary infarct (Wallenberg syndrome)
- Arnold-Chiari malformation
- Charcot–Marie–Tooth disease and its variants
- Postpolio syndrome
- parkinsonism

Theories of Vocal Cord Paralysis

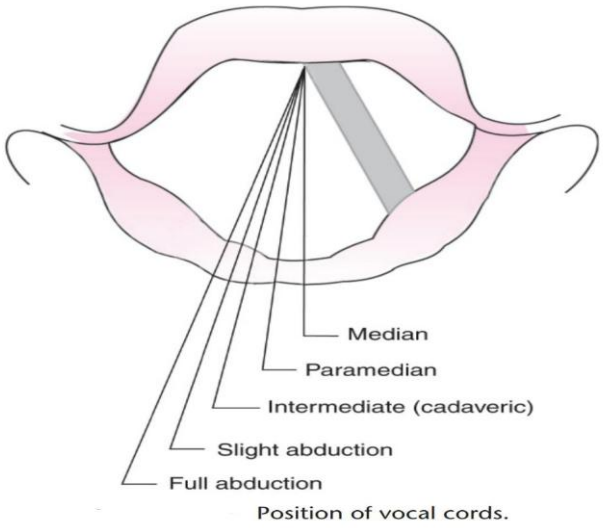
Semon's Law [70,71]

Which states that, in all progressive organic lesions, abductor fibres of the nerve, which are phylogenetically newer, are more susceptible and thus the first to be paralyzed compared to adductor fibres.

Wagner And Grossman Hypothesis [72,73]

Which states that cricothyroid muscle which receives innervation from superior laryngeal nerve keeps the cord in paramedian position due to its adductor function

Position of the cord	Location of the cord from midline	Situation in	
		Health	Disease
Median	Midline	Phonation	RLN paralysis
Paramedian	1.5 mm	Strong whisper	RLN paralysis
Intermediate (cadaveric)	3.5 mm. This is neutral position of cricoarytenoid joint. Abduction and adduction take place from this position	—	Paralysis of both recurrent and superior laryngeal nerves
Gentle abduction	7 mm	Quiet respiration	Paralysis of adductors
Full abduction	9.5 mm	Deep inspiration	—



Position of vocal cords.

Unilateral Laryngeal Nerve Paralysis

Clinical evaluation

History:

Patients with unilateral laryngeal paralysis typically present with complaints of hoarseness and hypophonia.

Dysphagia-related complaints are more common in patients with 'high' vagal injury, affecting both superior and recurrent laryngeal nerves, which leads to hemilaryngeal anaesthesia, pharyngeal constrictor muscle atony and cricopharyngeal muscle hyperfunction and glottic insufficiency from immobile vocal fold.

Surgeries affecting pulmonary reserve, which includes most of the thoracic procedures, appear to carry a higher risk of aspiration, and age may be a independent risk factor. Some patients may present with characteristically 'wet' vocal quality due to pooled secretions.[74]

Physical Examination:

The examination includes palpation of the neck for enlarged lymph nodes or thyroid enlargement. All the cranial nerves should be evaluated, with special attention to the spinal accessory and glossopharyngeal nerves, which share the jugular foramen with the vagus nerve. Presence of ipsilateral tongue deviation, palatal palsy or Horner syndrome should raise the suspicion of a cranial base lesion. Flexible nasolaryngoscopy provides a more accurate information of laryngeal function than rigid techniques.

Pitch-raising phenomenon using intact crico thyroid muscle function generates a characteristic voice described as 'paralytic falsetto'. The presence of supraglottic hyperfunction during phonatory effort indicate the possibility of glottic insufficiency. The ventricular folds may serve as an accessory phonatory mechanism that tends to be acting without much effort when the vocal folds proper do not approximate effectively.[75]

Cartilage on the paralyzed side of vocal cord fall forward into the laryngeal introitus (i.e. a ‘prolapsed arytenoid’), is a commonfinding in laryngeal paralysis. This is due to profound denervation of nerve supply with loss of muscular support for the cartilage.

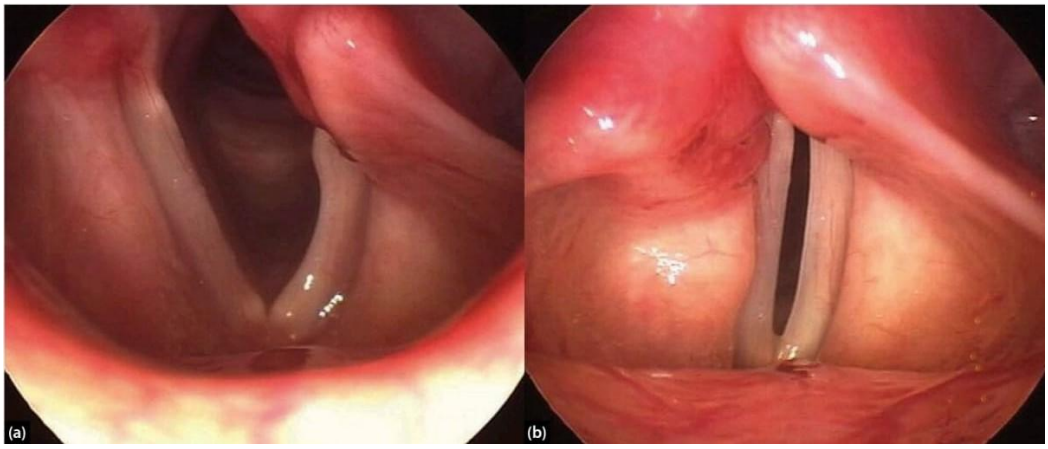


FIGURE 10

The incidence of cricoarytenoid dislocation as a cause of vocal fold immobility is rare, as the cricoarytenoid joint is strikingly robust and resistant to injury. A problem-free intubation is unlikely to cause a cricoarytenoid joint injury, which usually follows obvious trauma, be it from intubation or external sources. The presence of arytenoid erythema, oedema and the absence of a ‘jostle sign’ are suggestive of cricoarytenoid joint injury. [76,77,78]

The Voice Handicap Index (VHI) and the voice-related quality of life (V-RQOL) are patient-completed self-rating scales that measure various aspects of vocal disability.

Investigations:

Computed tomography (CT) from base of skull to the level of the arch of aorta or right subclavian artery is the minimum recommended radiological study for laryngeal paralysis. In patients where a 'high vagal' paralysis is suspected, magnetic resonance imaging (MRI) provides a more reliable means of imaging the skull base or central nervous system.

Available literature suggests that Laryngeal electromyography (LEMG) may be a more useful predictor of poor outcome , mainly because electromyographic signs such as fibrillations and positive sharp waves, provide evidence of an absence of re-innervation. [79]

Treatment:

Observation:

Factors favouring observation include:

- no evidence of visible aspiration
- injured laryngeal nerve(s) is (are) structurally intact and potential for recovery of function remains
- minimal vocal cord disability and/or minimal vocal demand
- comorbidities that prevent further intervention.

Injection Laryngoplasty

It is done in patients who opt for temporary relief of their symptoms, despite when eventual recovery is expected. This is achieved by injection of a absorbable bulking substance into the paralyzed vocal fold to improve the glottic insufficiency. Such substances include hyaluronic acid preparations, calcium hydroxylapatite paste, carboxymethylcellulose-glycerine gel, micronized human dermis and autologous fat.

Factors favouring injection augmentation include:

- difficulty in swallowing
- high degree of vocal disability or patient has high vocal demand
- good or indeterminate functional prognosis
- small glottic gap (2mm to 3mm)
- no posterior glottic gap
- Injection laryngoplasty can be performed perorally via direct laryngoscopy or transcutaneously under topical anaesthetic agent in the office, provided the patient is cooperative.

Laryngeal Framework Surgery

Laryngeal Framework surgery is generally done for treatment of glottic insufficiency from unilateral vocal fold paralysis that is not expected to improve.

Favourable factors are:

- difficulty in swallowing
- high degree of vocal disability or patient has high vocal demand
- poor functional prognosis
- large glottic gap
- posterior glottic gap
- shortened life expectancy.

Framework surgery consists of medialization thyroplasty, most commonly done procedure. It involves the surgical insertion of an implant, made of silicone, expanded polytetrafluoroethylene , calcium hydroxylapatite or other biologically inert material, into the paraglottic space to displace the paralysed cord medially.

Complications of the procedure include airway obstruction and perforation into the laryngeal lumen. Medialization thyroplasty narrows the laryngeal airway and in combination with post-operative oedema and haematoma, it can cause airway obstruction. Rarely, patients undergoing medialization thyroplasty may require intubation or tracheotomy in the immediate post-operative period.[80]

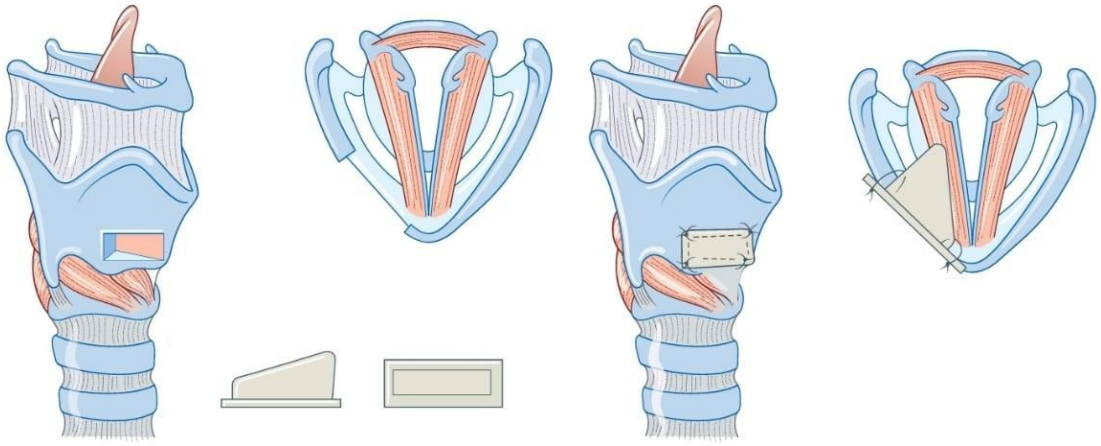


FIGURE 11

In medialization laryngoplasty, a medialization shim of biologically inert material is inserted into the paraglottic space through a thyroid cartilage window to displace the vocal fold towards the midline.

In patients with a poorly supported arytenoid cartilage or a posterior gap, Arytenoid repositioning procedures may be added to medialization thyroplasty as this configuration is difficult to remedy with thyroplasty alone. Arytenoid repositioning surgery is designed to suspend the arytenoid cartilage or internally rotate it in physiologic phonatory position. In majority, the muscular process of the arytenoid cartilage is approached through the inferior constrictor muscle and back of the thyroid lamina. A non-absorbable suture is then passed through this structure and secured to the thyroid lamina to exert anterolateral traction on the muscular process and rotate the vocal process medially and slightly caudally. This is known as arytenoid adduction.[81]

Adduction arytenoidopexy, is a less commonly used approach, which involves opening the cricoarytenoid joint capsule and suturing the arytenoid in optimal position to the crest of the cricoid.[82]

Re-Innervation

Re-Innervation using both the ansa cervicalis and the hypoglossal nerves would seem to be a logical approach to vocal fold paralysis. Due to complex innervation of the vocal fold muscles, re-innervation generally tends to improve the bulk and tone of vocal fold muscle but will not restore normal physiologic motion. In cases of nerve section recognized during surgery, immediate re-anastomosis or re-innervation if tension-free anastomosis of nerve is not possible, is the treatment of choice.[83]

Bilateral Vocal Fold Paralysis

Etiology

Thyroidectomy remains the primary surgical cause despite both nerves are also at risk at esophagectomy, tracheal resection, thymectomy and other mediastinal procedures. Similarly, tracheal, esophageal and thyroid malignancies may compromise both nerves. Neurologic disorders tend to cause diffuse involvement of the peripheral nervous system. Amyotrophic lateral sclerosis, post-polio syndrome, Charcot–Marie–Tooth neuropathy, Arnold Chiari malformation and GuillainBarré syndrome have been documented causes of bilateral paralysis. Some bilateral laryngeal paralyses are idiopathic, attributed to the same infectious agents as unilateral paralysis.[84]

Clinical Evaluation

Patients with bilateral vocal cord paralysis typically present with dyspnea, noisy breathing and exercise intolerance. The severity of respiratory symptoms is inversely proportional to the size of glottic chink between two immobile vocal folds. Respiratory noise is more on inspiration, as negative pressure pulls the paralysed vocal folds into closer approximation. Voice and swallowing is usually not affected.

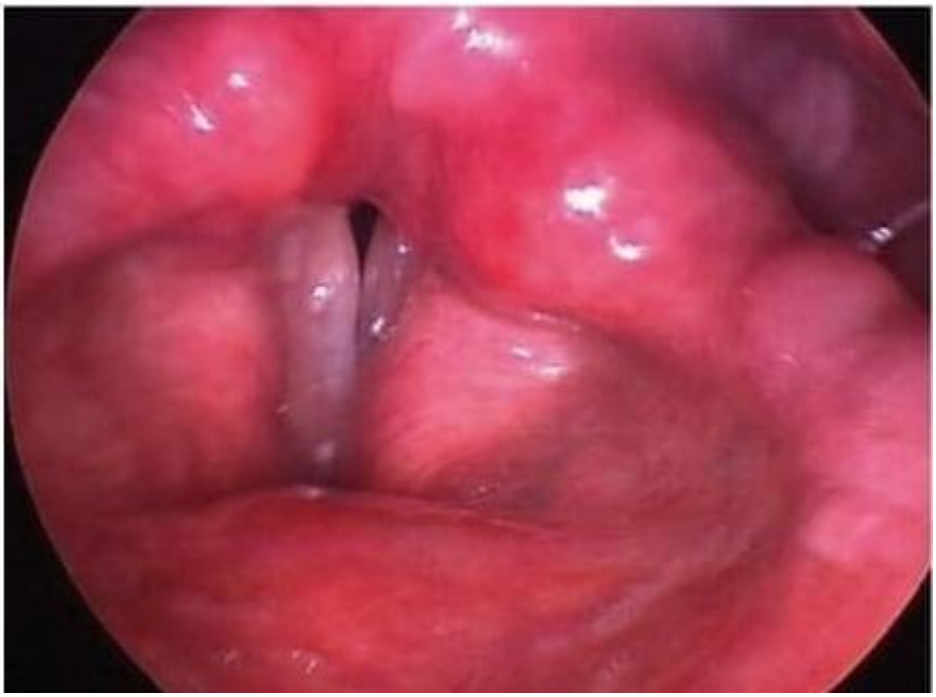


FIGURE 12

INVESTIGATION

Radiologic imaging similar to unilateral vocal fold paralysis, with special attention to mediastinum and infra-laryngeal neck, as well as the central nervous system should be taken. The degree of airway obstruction may be quantified using a flow-volume loop.

Bilateral vocal cord paralysis typically shows variable (rather than fixed) extra-thoracic obstruction, as the vocal folds adduct passively during inspiration and abduct with positive expiratory air pressure.

Treatment

It is Guided by the degree of airway limitation.

Tracheostomy should be performed emergently and may be considered a reasonable treatment option for the long term , as it guarantees adequate airway with minimal impact on phonation and deglutition.

Other treatment options include lateralization of the vocal cord or removal of vocal fold tissue or arytenoid to enlarge the glottic space. Effort is made to preserve the membranous part of vocal fold in these procedures, but voice and sometimes swallowing may be affected. Some of these procedures are irreversible, so the ENT surgeon must be sure that any possibility of spontaneous improvement has been evaluated before they are considered. Laryngeal pacemakers are currently being evaluated for vocal cord paralysis in human trials.[85]

ANOSMIA

Classification of Olfactory disorders:

- Anosmia refers to absence of smell sensation or function.
- Partial anosmia refers to ability to perceive some, but not all odours.
- Hyposmia or microsmia refers to decreased perception or sensitivity to odours.
- Hyperosmia refers to increased sensitivity to common odours.
- Dysosmia (also called cacosmia or parosmia) refers to distorted or perverted smell sensation

The Nose: Structure in relation to smell

The olfactory neuroepithelium is present in a small region of nasal mucosa in the upper recesses of the nasal chambers lining the cribriform plate and part of the superior turbinate and septum.[86]

The olfactory cleft, refers to region approximately 1mm wide and located 7cm deep in the nasal cavity , contains the majority of the olfactory neuroepithelium.[87]

While most of the inspired air coming into the nose pass through the conduits around the inferior and middle turbinates and along the septal wall, only 10–15% of the inspired air reaches the olfactory neuroepithelium.[88] Thus, minor changes in nasal anatomy and airflow can result in substantial air-flow blockage to olfactory cleft without much impairment in nasal respiration.[89,90]

Four Neural Systems within the Human Nose:

- The main olfactory system (Cranial Nerve I or CN I).
- The accessory olfactory system (i.e., the vomeronasal system)
- The trigeminal somatosensory system (CN V)
- The nervus terminalis or terminal nerve (CN 0).[91,92]

OLFACTORY NEUROEPITHELIUM AND NEURAL TRANSDUCTION

In the adult, six distinct classes of cells seen within the neuroepithelium:

- The bipolar sensory receptor neuron is of CNS origin and extends odourant receptor-containing cilia into the mucus.
- The supporting or sustentacular cell surrounds the bipolar receptor cells , regulates mucus production, transports molecules across the epithelium, and detoxifies odourants.
- The duct cell of Bowman's glands produce most of the mucus within the olfactory receptor region.
- The microvillar cell is located at the surface of the epithelium sends tufts of microvilli into the nasal mucus.
- The horizontal (dark) basal cells, refers to class of stem cells within the basement membrane of the epithelium.
- The globose (light) basal cells, a multipotent basal cell that can give rise to neurons and non-neuronal cells, including the horizontal basal cells.[91]

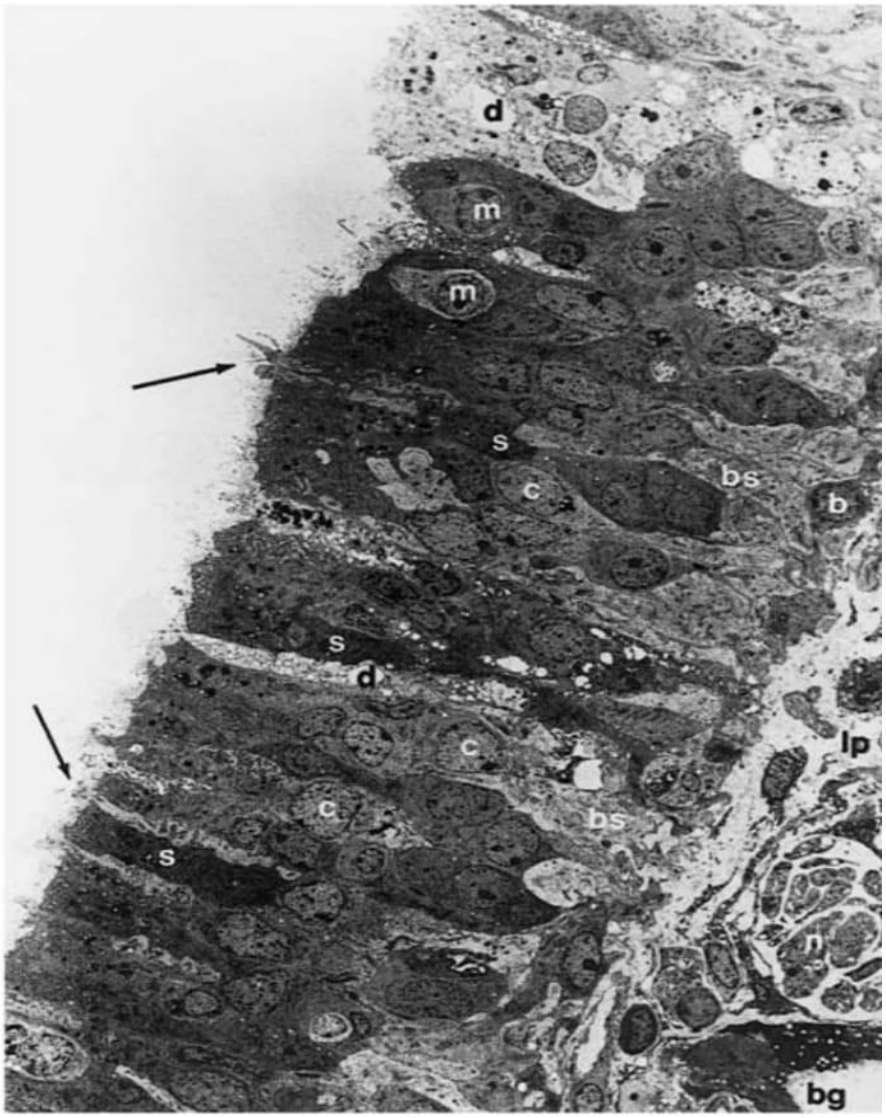


FIGURE 13

Low-power electron micrograph of a longitudinal section through a biopsy specimen of human olfactory mucosa taken from the nasal septum. Four cell types are indicated: ciliated olfactory receptors (c). microvillar cells (m), supporting cells (s) and basal cells (b). The arrows point to ciliated olfactory knobs of the bipolar receptor cells. d=degenerating cells; bs=base of the supporting cells; lp=lamina propria; n=nerve bundle; bg=Bowman's gland.

Approximately 6 million receptor cell axons finally unite to form 30–50 fascicles, termed the olfactory fila, which pierce the cribriform plate and pia mater to synapse with second-order neurons within the glomeruli of the olfactory bulb. The olfactory receptor neurons predominantly use the neurotransmitter glutamate to excite Olfactory Bulb neurons.[93]

The olfactory bulb and central projection

The olfactory bulbs are complex structures located on the ventral surface of the frontal lobes.

The main afferent second-order neurons are termed mitral and tufted cells.

Higher order brain regions targeted by the mitral and tufted cells of the bulb include (from rostral to caudal):

- The piriform cortex.
- The entorhinal area.
- The amygdaloid cortex (a region contiguous with the underlying amygdala).
- The corticomedial nuclear group of the amygdala.

Clinical evaluation of smell function

- A detailed clinical history
- Objective quantitative olfactory testing
- A thorough physical examination
- appropriate brain and rhinosinus imaging.

Quantitative olfactory testing

A number of brief self-administered tests range from the 3-item Pocket Smell Test to the 40-item University of Pennsylvania Smell Identification Test or UPSIT.[94]

The UPSIT known as the Smell Identification Test commercially is the most widely used olfactory test. [95] The UPSIT can be self-administered and scored in less than one minute by non-medical personnel.

This test consists of four booklets each containing 10 microencapsulated (‘scratch and sniff’) odourants. Test results are in the form of a percentile score of a patient’s performance relative to age- and sex-matched controls.

Based on the scores olfactory function can be classified into one of six categories: normosmia, mild microsmia, moderate microsmia, severe microsmia, anosmia, and malingering. Very low UPSIT scores reflect avoidance allowing for determination of malingering. The reliability of this test is very high.

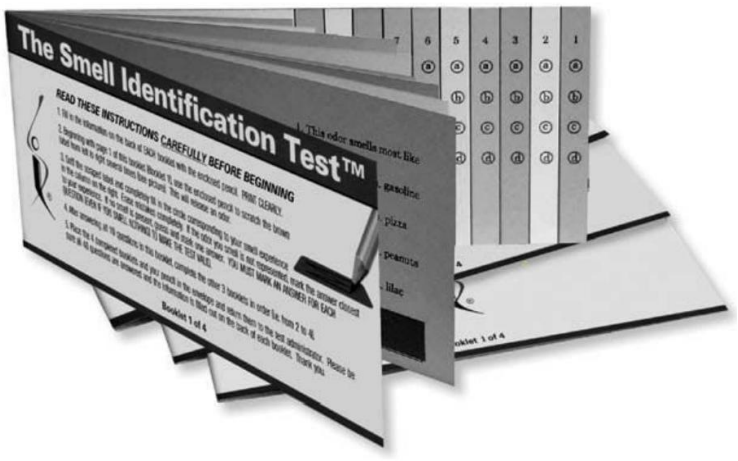


FIGURE 14

Types of olfactory dysfunction after viral infection

Transient or short-term dysfunction

a. Conductive (obstructive) or mechanical losses (eg, congestion) result from blockage of inspired air due to local inflammation and oedema of mucosal tissue in the olfactory cleft and upper nasal cavity.

b. Sensorineural (olfactory epithelium and olfactory nerve) dysfunction further subdivided into two types:

    - Altered quantity or function of odorant-binding receptor molecules
    - Neuropraxia or dysfunction of olfactory sensory neurons

c. Central (olfactory bulb and brain) dysfunction further subdivided into:

    - Pathology isolated to the olfactory bulbs
    - Pathology isolated to higher-order brain regions such as the piriform cortex and orbitofrontal cortex.

Chronic or permanent dysfunction

- Loss of olfactory epithelium (due to death of neural stem cells)
- Disruption of central olfactory processing region or network.[96,97]

Mechanism of olfactory dysfunction after Covid infection

The spread of virus or subviral ribonucleoprotein unit may occur through cribriform plate into the olfactory bulb of the CNS via a paracellular route or a transcellular route, although evidence is very minimal.

The cytokine storm hypothesis states that post-viral neurological disease can be due to unchecked, exaggerated, and sterile immunopathology, with active viral replication playing an initiating and secondary role. [98]

Another proposed mechanism for viral invasion involves direct targeting of non-neuronal receptor sustentacular support cells, which express the ACE2 receptor and TMPRSS2 (transmembrane protease serine 2) by the virus. Once infected and impaired, these cells might disrupt the electrophysiological and biochemical homeostasis of adjacent olfactory sensory neurons, and the resultant resource-restricted environment might silence the olfactory receptor similar to transient neuropraxia. [99]

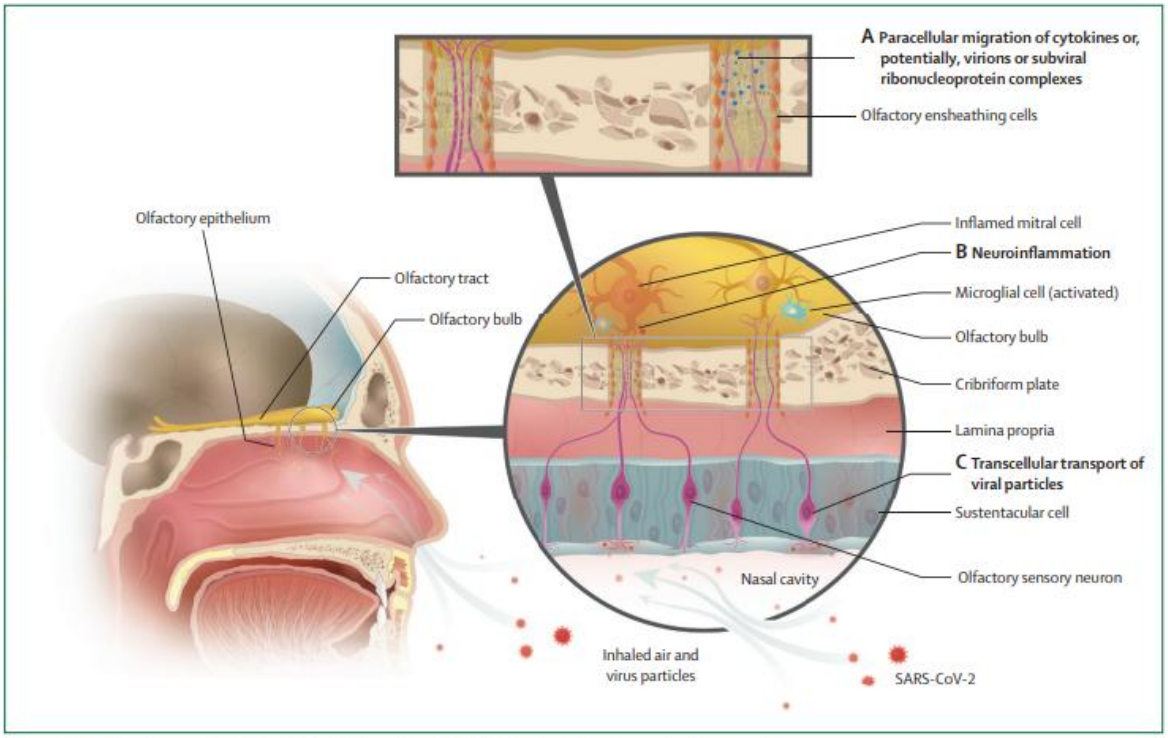


Figure: Potential pathways by which SARS-CoV-2 can infect the olfactory bulbs and generate inflammation

FIGURE 15

Imaging

MRI is the non invasive investigation of choice

Thickening and clumping of olfactory fibres is the common finding reported,[100] suggesting postviral inflammatory neuropathy.

Treatment

Oral steroids, rather than topical steroids, has shown to improve olfactory function in a group of patients.[101]

Effective treatments are available for conductive anosmia, where there is an obstruction of airflow to the olfactory neuroepithelium. Topical nasal steroids are ineffective in returning smell function because the steroid fails to reach the affected regions ie the olfactory cleft. Increased efficacy occurs when the nasal sprays are administered in the head-down Moffett's position. [102]

Pre and post-intervention olfactory testing should be performed to establish intervention efficacy.

Sensorineural olfaction impairment is difficult to manage, and the prognosis for patients is poor. The majority of patients who recover smell function following trauma do so within 12 weeks of injury.[103]

Alpha-lipoic acid has shown to improve the results of objective tests of olfactory function in an uncontrolled study,[104] but its use may be associated with neurological side effects, including headache, dizziness and confusion, which may be difficult to interpret among Covid-19 manifestations.

Omega-3 supplements found to be protective against olfactory loss during the recovery period after skull base surgery and therefore may have beneficial role in aiding recovery after post-viral olfactory loss,[105] although this has not been formally tested in Covid-19 patients.

Intranasal vitamin A can also be used to improve olfactory function,[106] but it is locally irritant to the nose.

MUCORMYCOSIS

Mucormycosis is an aggressive, opportunistic devastating fatal fungal infection commonly involving Nasal, Orbital and Cerebral regions. This condition is characterized by a time course of less than 4 weeks duration.[107]

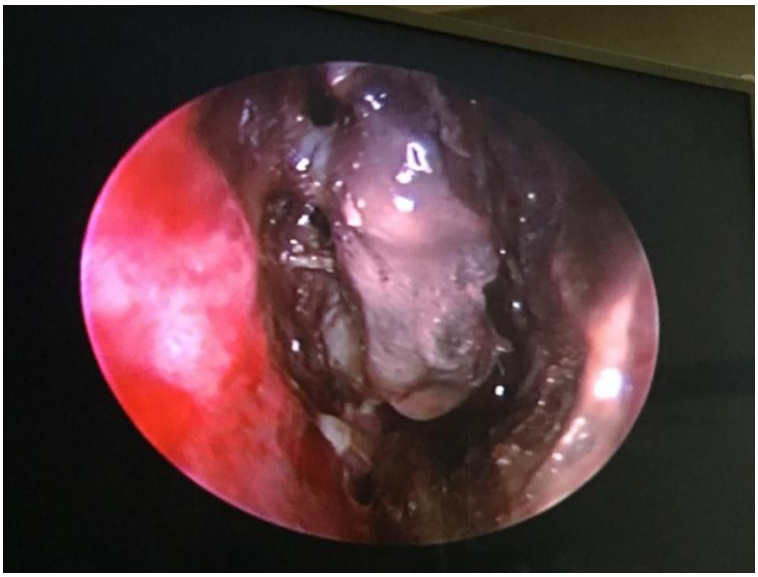


FIGURE 16

PATHOGENESIS:

It is acquired primarily via inhalation of spores through nasal route.

The fungus is angio-invasive causing occlusion of blood vessels, leading to tissue necrosis.[108,109,110]

↓

From the nasal cavity it enters the middle turbinate

↓

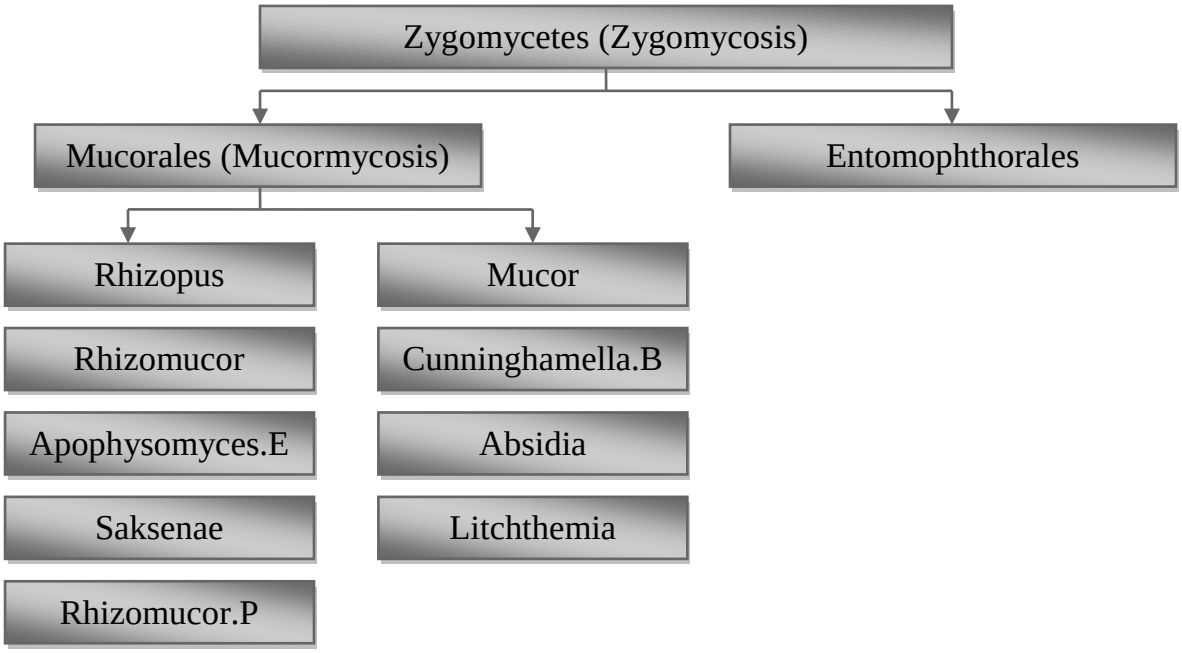
reach maxillary sinus

↓

erodes the floor of the orbit to reach the eye and further to CNS.

Species:

Fungi in the order of mucorale (e.g. Rhizopus, Rhizomucor and Mucor) and Aspergillus species are the most common causative species.[111]



Types:

- Sinonasal (8%)
- Rhino orbital (41%)
- Rhino orbito cerebral (27%)
- Others (10%)
    - Pulmonary mucormycosis
    - Cutaneous,
    - Gastrointestinal,
    - bone & joint infection and
    - disseminated Mucormycosis. [112]

Predisposing Factors

- Concurrent / Recently treated COVID -19[113]
- Uncontrolled Diabetes Mellitus [114]
- Inappropriate use of Steroids o High doses of steroids o Using for prolonged periods [115,116]
- Immunocompromised individuals o Malignancy o Transplant recipients [117,118]
- Prolonged use of broad-spectrum antibiotics
- People under long standing Oxygen therapy [119]
- Prolonged ICU stay[120]
- People under mechanical ventilation[121]
- Nosocomial (health care associated mucormycosis) [122]
- ICU gadgets - if not properly sterilized.

CLINICAL PRESENTATION

I. Generalized Symptoms:

i. Headache

ii. Low Grade Fever [123]

iii. Malaise & Lethargy

II. Nasal Symptoms:

i. Nasal Obstruction

ii. Nasal Discharge often bloody, brownish, or blackish. [124]

III. Ocular Manifestations: [125]

i. Pain and redness around eyes, watering

ii. Periorbital Swelling / Discolouration, Proptosis

iii. Diplopia / Diminution of vision

iv. Ptosis

v. Ophthalmoplegia

vi. Facial Swelling & pain, paresthesia, Numbness in the infra orbital region

IV. Oral Manifestations:

i. Tooth ache /Loosening of teeth

ii. Blackish discolouration of oral mucosa

iii. Loss of sensation/ numbness over palatal region

V. Chest pain, haemoptysis in case of **pulmonary** involvement

VI. Altered sensorium in case of **cerebral** involvement [123]

Diagnosis

Diagnostic Nasal endoscopy: Ulceration / Blackish necrotic eschar[124]

Oral Examination: Black eschar on the Palate

Ocular Examination:[125]

- Redness, watering
- Proptosis, Periorbital edema, ecchymosis
- Restricted eye movement, Ophthalmoplegia
- Decreased Corneal Sensation
- Fundus examination: cherry red spot or disc edema or both.

Microbiology:

I. KOH smear

Specimen:

- Nasal mucosal scraping in normal saline or Tissue biopsy from suspected mucosa in normal saline

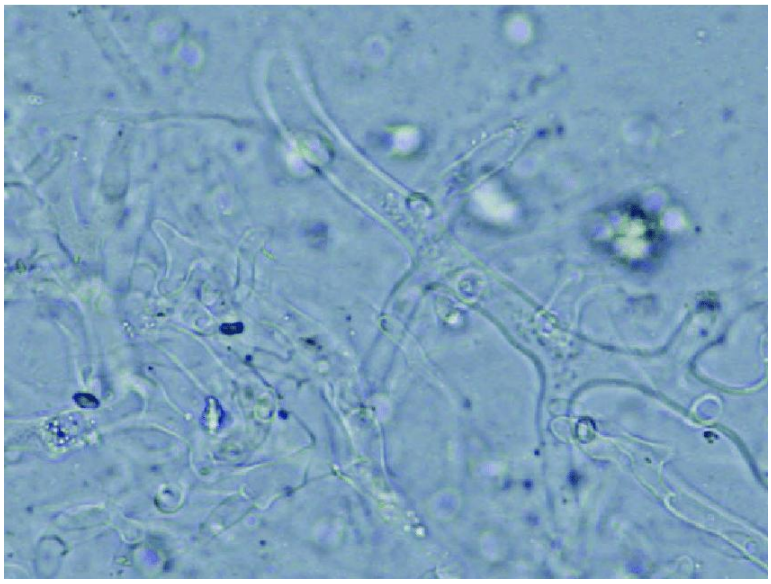


FIGURE 17

- Presence of broad aseptate hyaline hyphae (Ribbon Like) with irregular branching is indicative of Mucormycosis.[126,127]

II. Fungal culture on Saboraud's dextrose agar or Potato dextrose agar in a test tube at 30℃ & 37℃.

- Any growth suggestive of fungi should be examined by making Lactophenol cotton blue mount on a slide and examined under microscope for identification of the fungus.[126,127]

Pathology:

Specimen:

- Debrided tissue for HPE to be sent to the Laboratory immediately in 10% Formalin.

- Histopathological processing and staining done with routine H&E and special stains like PAS & GMS.[126,127]

- Diagnosis is by visualizing the fungal infiltration of blood vessels, vasculitis with thrombosis, tissue infarction ,acute neutrophilic infiltrate and fungal hyphae based on morphology in the tissue.

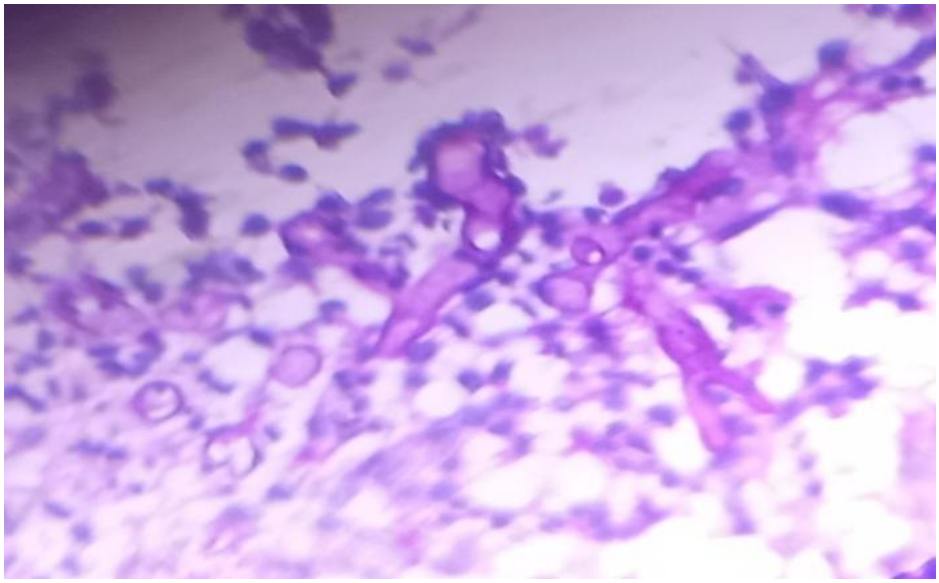


FIGURE 18

Radiology:

- HRCT Scan of Orbit, Paranasal sinuses & Brain, with contrast if renal status permits.[128]
- MRI Paranasal sinuses and orbits (optional).[126,127]

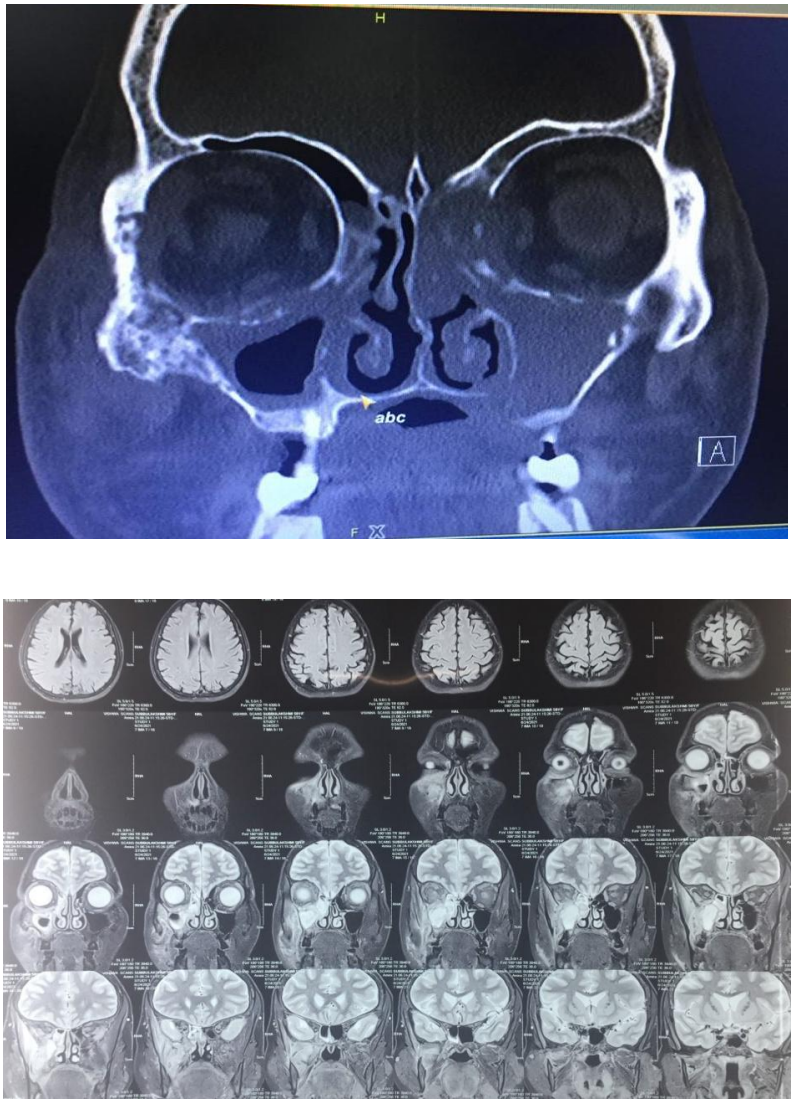


FIGURE 19

General Investigations:

a. CBC

b. Fasting and Post Prandial Blood Sugar Monitoring

c. HbA1C

d. LFT

e. KFT with electrolytes

Treatment

- multi-disciplinary approach.[126,127]
- involving General Physician/Infectious disease specialist, ENT surgeon, Ophthalmologist, Neurologist, Neurosurgeon and Faciomaxillary surgeon depending on the extent of involvement.
- Treatment should be early and aggressive.

Medical

Drugs Recommended - Dose - Duration [129]

Inj.Amphotericin B – Deoxycholate 0.75 -1mg/kg/day

up to total daily maximum of 50mg

(OR) Liposomal Amphotericin B 5mg/kg/day (nasal & orbital)

8 – 10mg/kg/day (Cerebral)

(OR) Inj.Amphotericin B – Lipid Complex 3 - 5mg/kg/day

4 to 6 weeks depending on severity

Followed By

Posaconazole 300mg once a day orally 2 to 4 weeks depending on severity

Salvage therapy:

In patients who cannot tolerate Amphotericin B due to severe renal impairment / allergy / selected cases for cerebral mucormycosis.[118,129]

Inj. Posaconazole: 300mg IV BD / oral /Day 1 - followed by 300mg OD X 4 – 6 weeks

(or)

Inj. Isavuconazole: 200mg IV TID /oral X 2 days - followed by 200mg IV OD /oral X 4 – 6 weeks

Staging:[130]

Proposed Staging of Rhino-Orbito-Cerebral Mucormycosis (ROCM)

Staging of Rhino-Orbito-Cerebral Mucormycosis	Symptoms	Signs	Primary Assessment	Confirmation of Diagnosis
Stage 1: Involvement of the nasal mucosa 1a: Limited to the middle turbinate 1b: Involvement of the inferior turbinate or ostium of the nasolacrimal duct 1c: Involvement of the nasal septum 1d: Bilateral nasal mucosal involvement	Nasal stuffiness, nasal discharge, foul smell, epistaxis	Foul-smelling sticky mucoid or black-tinged, or granular or haemorrhagic nasal discharge, nasal mucosal inflammation, erythema, violaceous or blue discoloration, pale ulcer, anaesthesia, ischemia, eschar	Diagnostic nasal endoscopy, Contrast-enhanced MRI (preferred) or CT-scan	Deep nasal swab or endoscopy-guided nasal swab or nasal mucosal biopsy for direct microscopy, culture and molecular diagnostics; nasal mucosal biopsy for rapid histopathology with special stains
Stage 2: Involvement of paranasal sinuses 2a: One sinus 2b: Two ipsilateral sinuses 2c: > Two ipsilateral sinuses and/or palate/oral cavity 2d: Bilateral paranasal sinus involvement or involvement of the zygoma or mandible	Symptoms in Stage 1 + facial pain, facial edema, dental pain, systemic symptoms (malaise, fever)	Signs in Stage 1 + unilateral or bilateral, localized or diffuse facial edema, edema localized over the sinuses, localized sinus tenderness	Diagnostic nasal endoscopy, Contrast-enhanced MRI (preferred) or CT-scan	Same as Stage 1 + sinus biopsy for direct microscopy, culture and molecular diagnostics and rapid histopathology
Stage 3: Involvement of the orbit 3a: Nasolacrimal duct, medial orbit, vision unaffected 3b: Diffuse orbital involvement (>1 quadrant or >2 structures), vision unaffected 3c: Central retinal artery or ophthalmic artery occlusion or superior ophthalmic vein thrombosis; involvement of the superior orbital fissure, inferior orbital fissure, orbital apex, loss of vision 3d: Bilateral orbital involvement	Symptoms in Stage 1 and 2 + pain in the eye, proptosis, ptosis, diplopia, loss of vision, infraorbital and facial V1 V2 nerve anesthesia	Signs in Stage 1 and 2 + conjunctival chemoses, isolated ocular motility restriction, ptosis, proptosis, infraorbital nerve anesthesia, central retinal artery occlusion, features of ophthalmic artery occlusion and superior ophthalmic vein thrombosis. V1 and V2 nerve anesthesia, and features of III, IV and VI nerve palsy indicating orbital apex/superior orbital fissure involvement.	Diagnostic nasal endoscopy, Contrast-enhanced MRI (preferred) or CT-scan	Same as Stage 2 + orbital biopsy if indicated and if feasible (if the disease is predominantly orbital) for direct microscopy, culture and molecular diagnostics and rapid histopathology
Stage 4: Involvement of the CNS 4a: Focal or partial cavernous sinus involvement and/or involvement of the cribriform plate 4b: Diffuse cavernous sinus involvement and/or cavernous sinus thrombosis 4c: Involvement beyond the cavernous sinus, involvement of the skull base, internal carotid artery occlusion, brain infarction 4d: Multifocal or diffuse CNS disease	Symptoms in Stage 1 to 3 + bilateral proptosis, paralysis, altered consciousness, focal seizures	Signs in Stage 1-3 (some features overlap with Stage 3) + V1 and V2 nerve anesthesia, ptosis, and features of III, IV and VI nerve palsy indicate cavernous sinus involvement. Bilaterality of these signs with contralateral orbital edema with no clinico-radiological evidence of paranasal sinus or orbital involvement on the contralateral side indicate cavernous sinus thrombosis. Hemiparesis, altered consciousness and focal seizures indicate brain invasion and infarction.	Diagnostic endoscopy, Contrast-enhanced CT Scan, MRI (preferred)	Same as Stage 3

Surgical

ENT:

Aim

Surgical Debridement of the Dead Necrotic Tissue to prevent the further progression and spread of lesion.

Nasal & Sinus involvement (Without bony involvement)

Endoscopic sinus surgery debridement of infected and necrotic tissue, with drainage of infected paranasal sinuses performed. It minimizes the fungal load in the tissue.[131]

End point of surgery:

- Bleeding from normal Tissue after removal of Necrotic Tissue.
- Patient complaints of pain if the surgery is done under LA.

Nasal & Sinus involvement (With bony involvement)

Endoscopic sinus surgery debridement with removal of devitalized bone. [132]

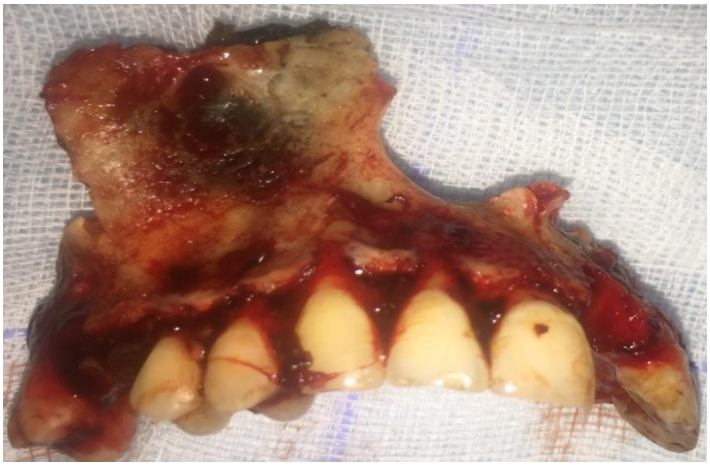


FIGURE 20

Sinus involvement with early loss of vision:

Endoscopic sinus surgery + Trans cutaneous Retro Bulbar Amphotericin B 1ml of 3.5 mg per ml.

FIGURE 21

Ophthalmology:

Stage 3a, 3b –

Endoscopic orbital decompression with sampling of retro orbital fat for HPE.

Stage 3c –

Exenteration [133]

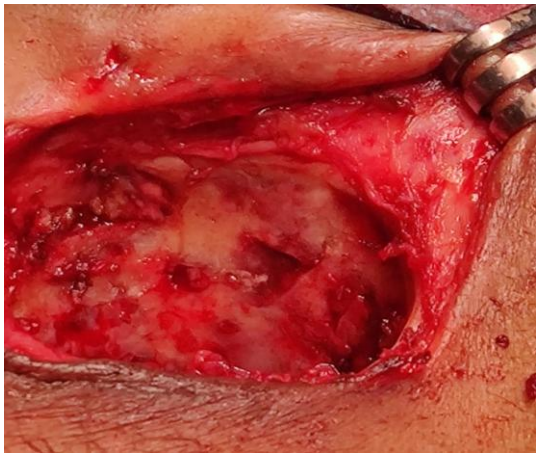


FIGURE 22

Cerebral:

Anterior Table: Debridement

Posterior Table: Cranialization Debridement of osteomyelitic skull bone and involved cerebral parenchyma (Safe Maximum Resection). Early commencement of treatment has better outcomes. Those presenting late with signs of intracranial involvement have a poorer outcome.[134]

Follow up after surgery:

- Relook nasal endoscopy of weekly intervals for 6 weeks to assess epithelization of nasal cavity and to remove any residual necrotic bone.
- Daily nasal douching with diluted Amphotericin B solution (50mg vial in 500ml of normal saline)

Preventive measures: [130]

1. Personal Hygiene

    a. Good Oral Hygiene

    b. Periodical change of Bed Linen

2. Medical Management

    a. Judicious use of steroids at right time, in right dose and for right period.

    b. Strict diabetes control (110-180mg/dl) with Insulin and Oral Hypoglycemic Agents

3. Hospital / Institutional level

    a. Use clean, sterile water for humidifiers during oxygen therapy

    b. Disinfecting all gadgets in ICU regularly

c. NOT TO REUSE disposable oxygen delivery devices like Nasal prongs, Face masks etc.

4. Advice to the Patient and care-giver at the time of discharge:

a. Monitor blood glucose level in diabetics

b. Inform the patients about early symptoms & signs of Mucormycosis.

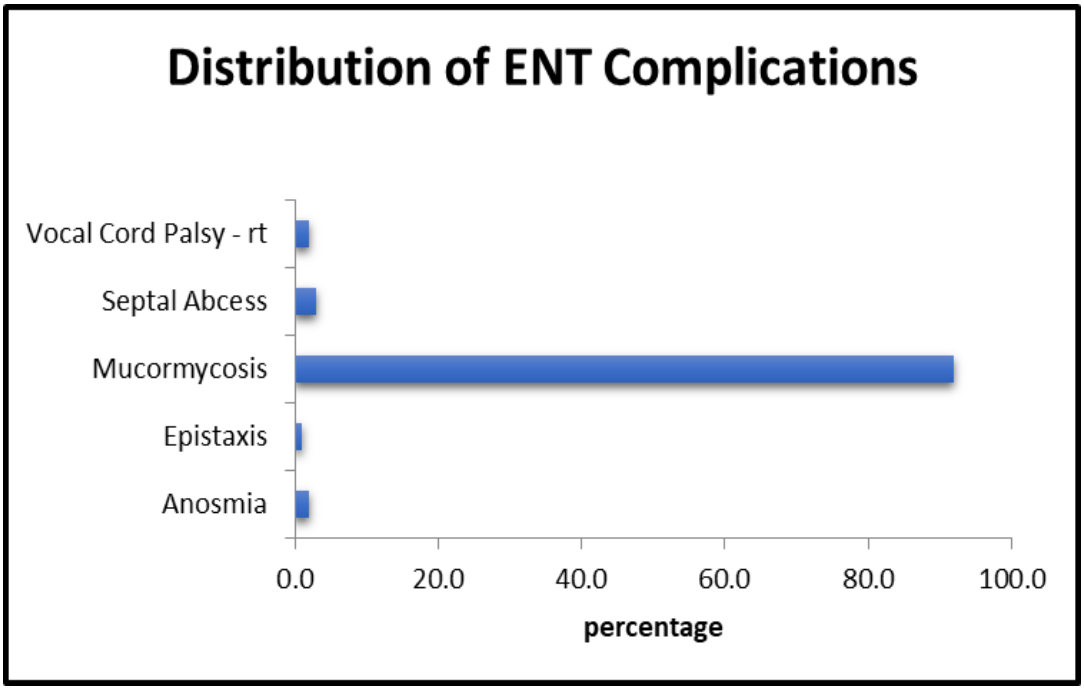
RESULTS

STATISTICAL ANALYSIS

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0.(Armonk, NY: IBM Corp).To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.

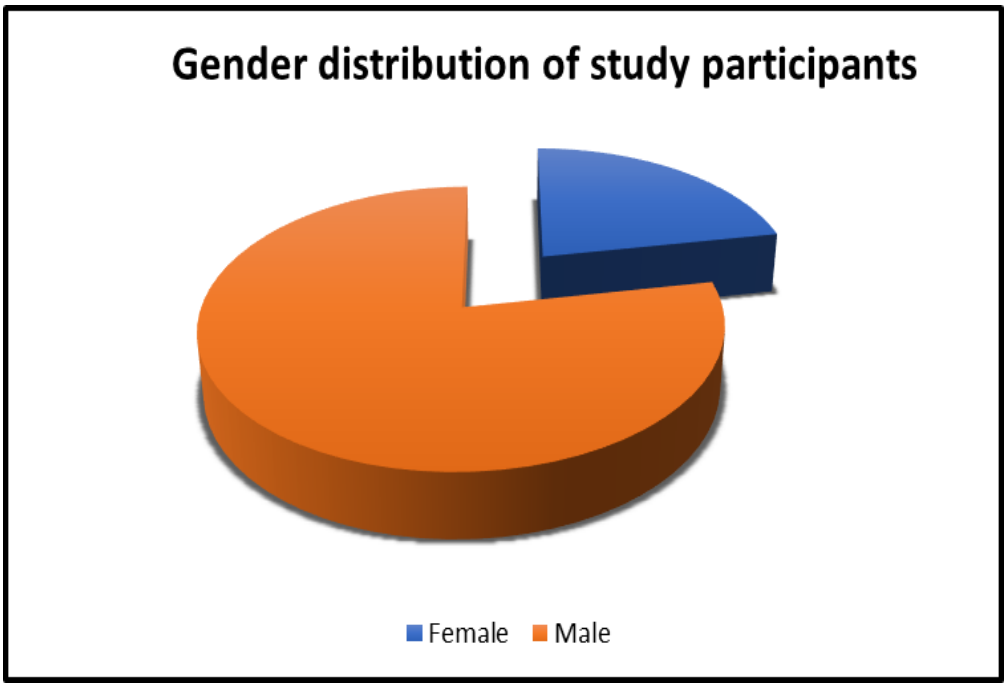
Distribution of ENT Complications among study participants:

Distribution of ENT Complications		
	Frequency	**Percent**
Anosmia	2	2.0
Epistaxis	1	1.0
Mucormycosis	92	92.0
Septal Abcess	3	3.0
Vocal Cord Palsy (Unilateral-Right)	2	2.0
Total	100	100.0



In our study of 100 patients, 92% patients presented with mucormycosis, 3% patients presented with septal abscess, 2% patients presented with anosmia, 2% patients presented with unilateral vocal cord palsy and 1% patient presented with epistaxis.

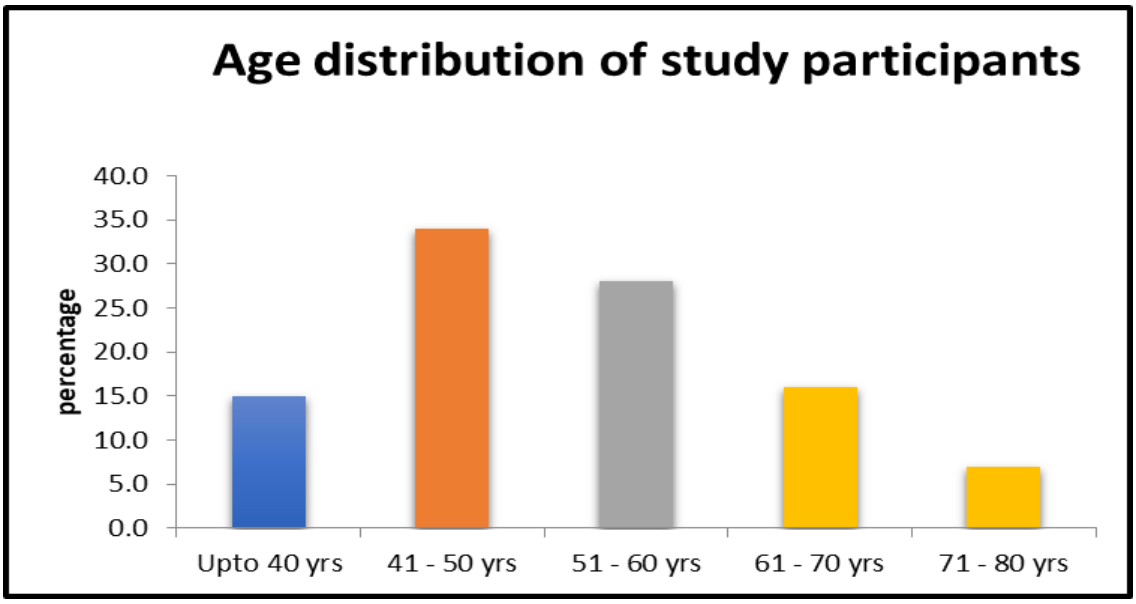
Gender distribution of study participants		
	Frequency	**Percent**
Female	22	22.0
Male	78	78.0
Total	100	100.0



Of the study population, who presented with ENT complications 78% were males and 22% were females.

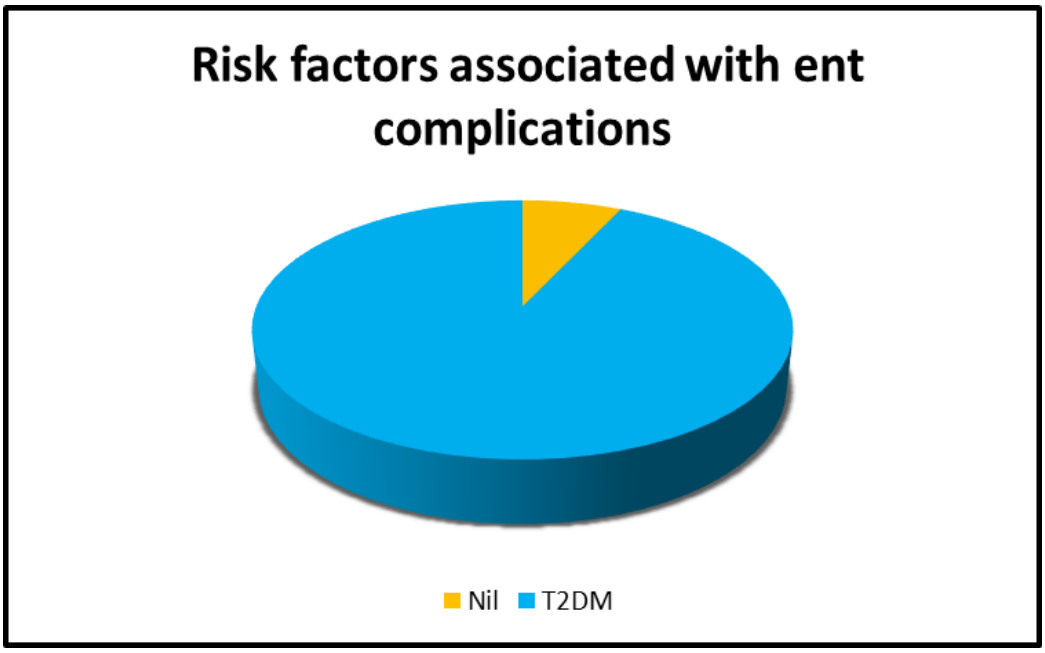
The mean age of the study participant was 52 ± 12 yrs.

Age distribution of study participants		
	Frequency	**Percent**
Upto 40 yrs	15	15.0
41 - 50 yrs	34	34.0
51 - 60 yrs	28	28.0
61 - 70 yrs	16	16.0
71 - 80 yrs	7	7.0
Total	100	100.0
Mean ± SD = 52 ± 12 yrs		



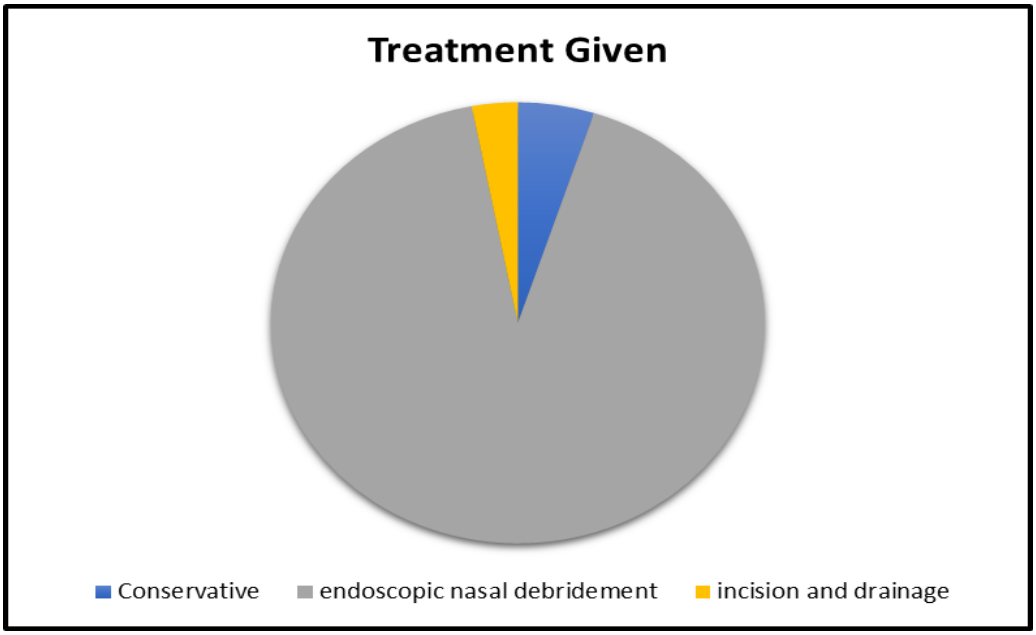
Based on age of the participants, patients who presented with complications, 34% cases more commonly belong to age group 41- 50 years. In the remaining, 28% belong to age group 51 – 60 years, 16% belong to age group 61-70 years, 15% belong to age group less than 40 years and 7% belong to age group 71- 80 years.

Risk factors associated with ent complications		
	Frequency	**Percent**
Nil	7	7.0
T2DM	93	93.0
Total	100	100.0



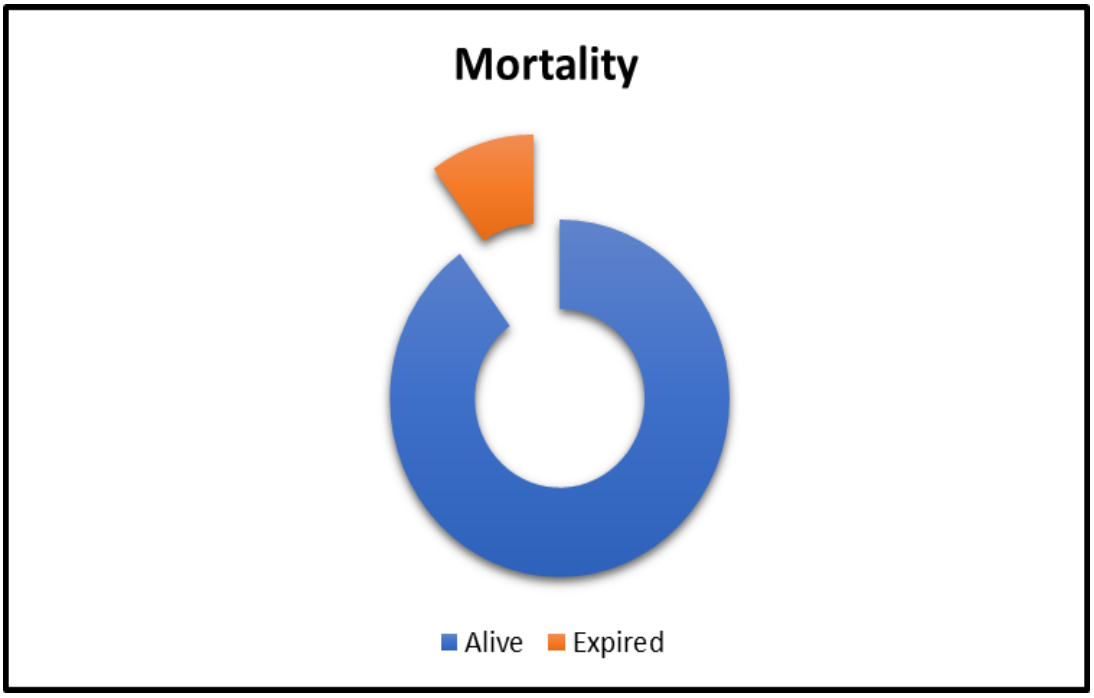
In our study on 100 cases, type 2 diabetes mellitus was the predominant risk factor and it was present in 93% of cases. About 7% individuals had no associated risk factors.

Treatment Given		
	Frequency	**Percent**
Conservative	5	5.0
Endoscopic nasal debridement	92	92.0
Incision and Drainage	3	3.0
Total	100	100.0



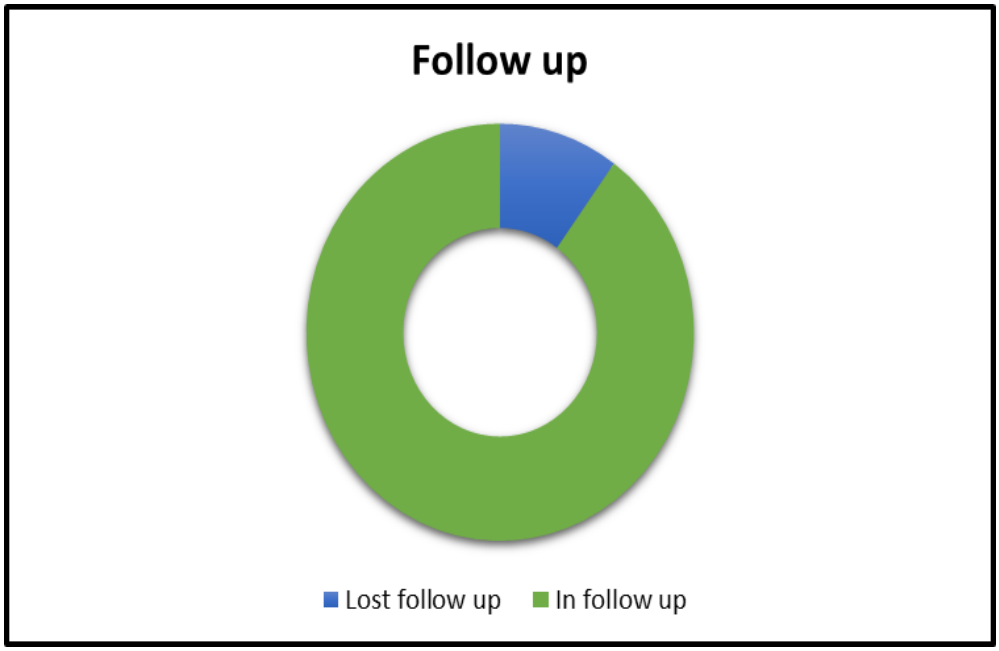
In a total of 100 cases, entire 92% mucormycosis patients underwent Endoscopic nasal debridement, incision and drainage done in all 3% septal abscess cases, rest of the 5% cases who presented with anosmia, epistaxis and unilateral vocal cord palsy was managed conservatively.

Mortality		
	Frequency	**Percent**
Alive	90	90.0
Expired	10	10.0
Total	100	100.0



Among the 100 study participants, about 10% cases expired mainly due to post Covid complications (respiratory failure, pneumonia…)

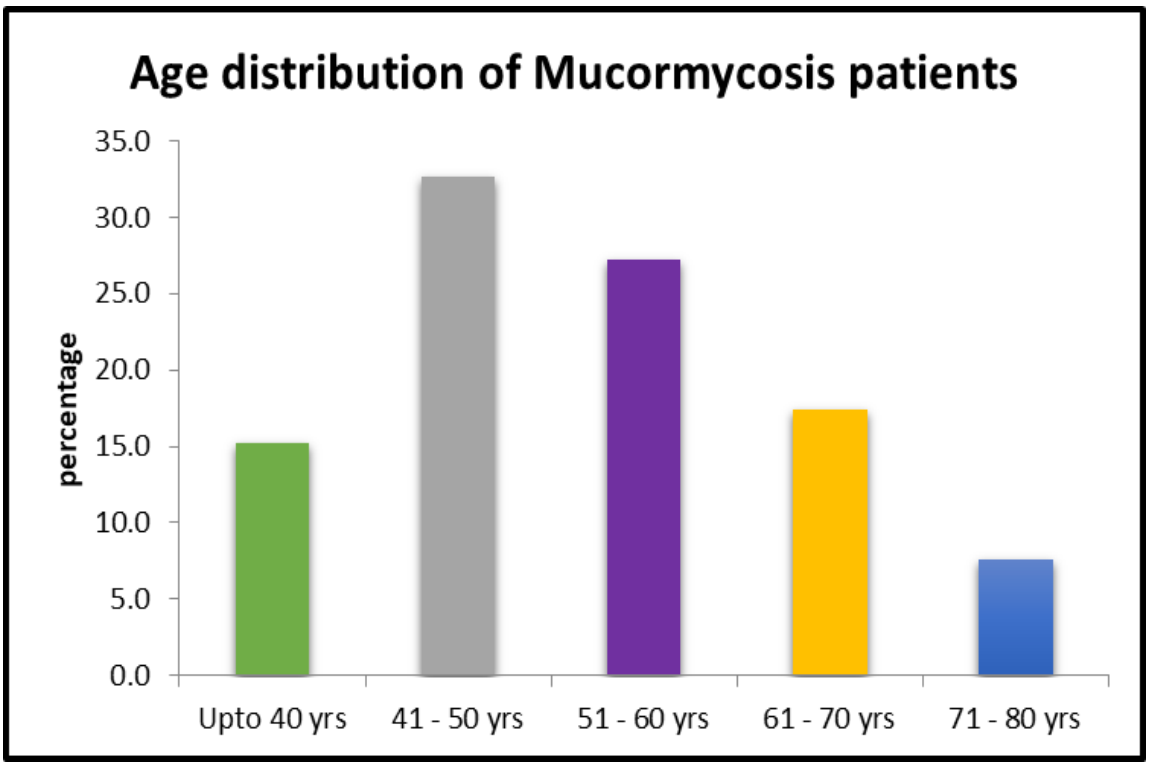
Follow up		
	Frequency	**Percent**
Lost follow up	10	10.0
In follow up	90	90.0
Total	100	100.0



Post treatment nearly 90% patients are in regular follow up.

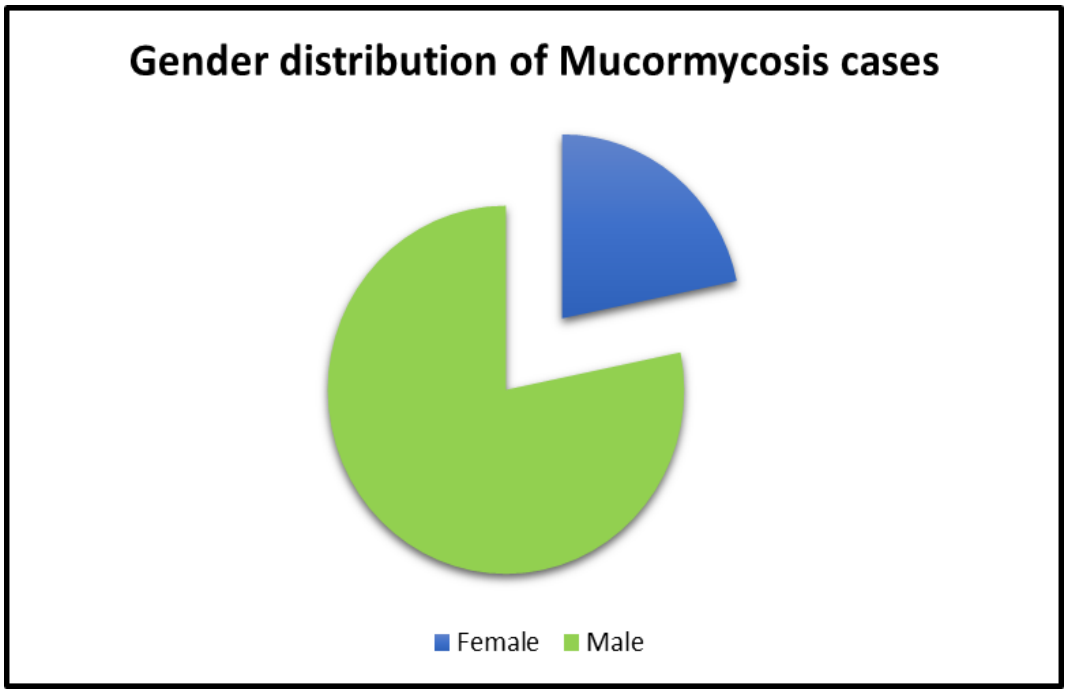
The mean age of the study participant was 53 ± 12 yrs.

Age distribution of Mucormycosis patients		
	Frequency	**Percent**
Upto 40 yrs	14	15.2
41 - 50 yrs	30	32.6
51 - 60 yrs	25	27.2
61 - 70 yrs	16	17.4
71 - 80 yrs	7	7.6
Total	92	100.0
Mean ± SD = 53 ± 12 yrs		



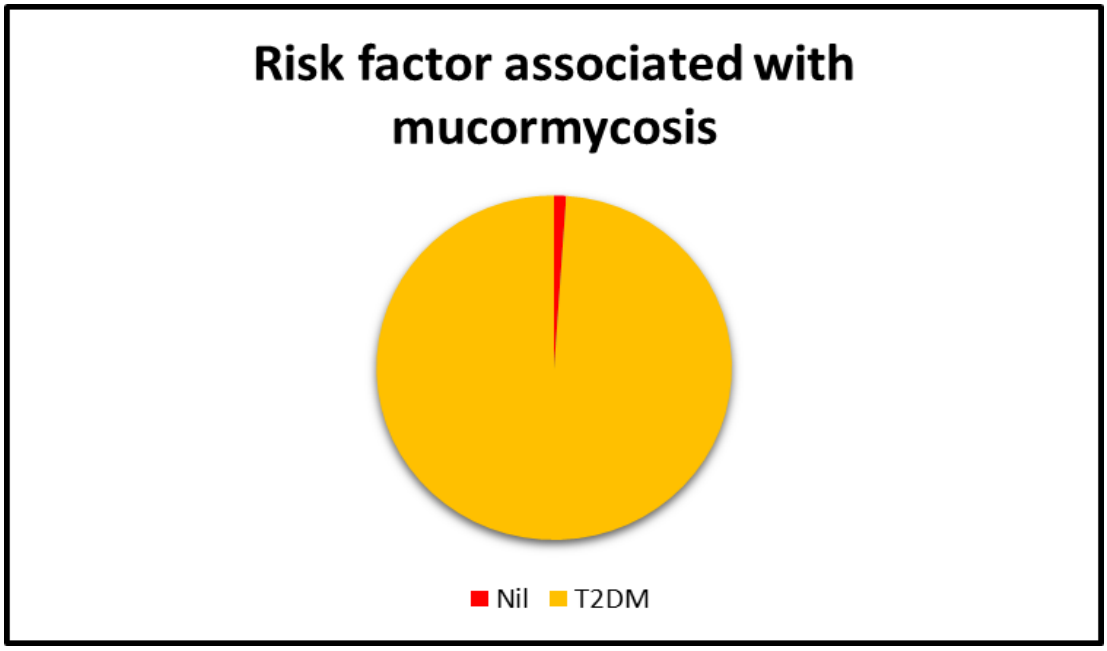
Among patients who presented with mucormycosis, the predominant post Covid ENT complication, about 32.6% study participants more commonly belong to age group 41- 50 years. In the remaining, 27.2% belong to age group 51 – 60 years, 17.4% belong to age group 61-70 years, 15.2% belong to age group less than 40 years and 7.6% belong to age group 71- 80 years.

Gender distribution of mucormycosis cases		
	Frequency	**Percent**
Female	20	21.7
Male	72	78.3
Total	92	100.0



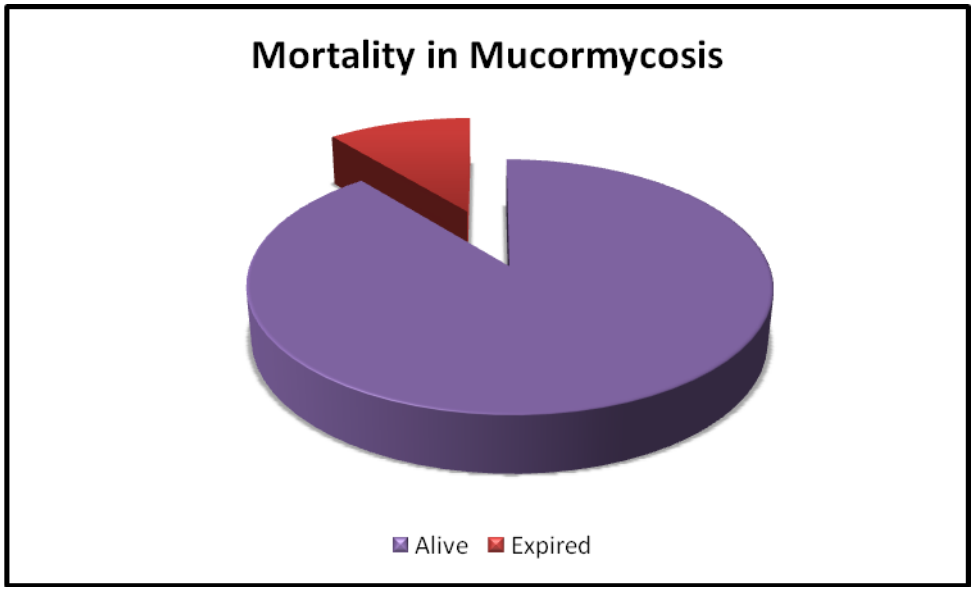
Of the study population, who presented with mucormycosis, 78.3% were males and 21.7% were females.

Risk factor associated with mucormycosis		
	Frequency	**Percent**
Nil	1	1.1
T2DM	91	98.9
Total	92	100.0



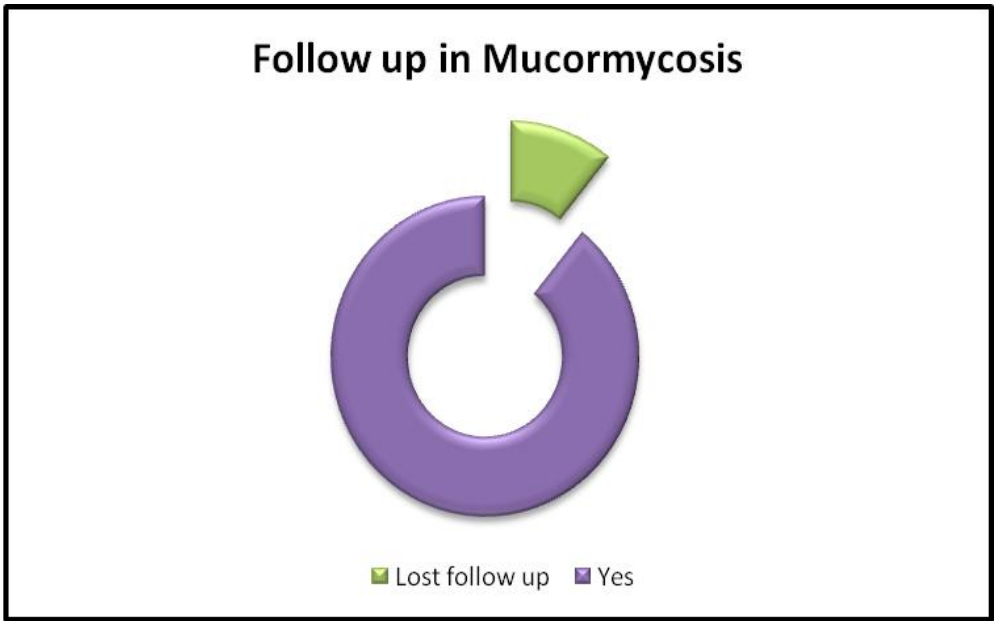
Among the 92 mucormycosis cases, type 2 diabetes mellitus was the predominant risk factor and it was present in 98.9% cases.1.1% study participant had no associated risk factors.

Mortality in mucormycosis cases		
	Frequency	**Percent**
Alive	82	89.1
Expired	10	10.9
Total	92	100.0



In a total of 92 mucormycosis cases, post treatment about 10.9% cases expired mainly due to post Covid complications - respiratory failure & pneumonia

Follow up in mucormycosis cases		
	Frequency	**Percent**
Lost follow up	10	10.9
In follow up	82	89.1
Total	92	100.0



Post treatment, 89.1% mucormycosis patients are in regular follow up.

DISCUSSION

This study carried out in total patients with laboratory confirmed SARS Cov 2 infection who attended review clinic and ENT OPD, in our tertiary care centre, Coimbatore, from august 2020 to June 2021.

We identified 100 patients who developed spontaneous ENT complication like long lasting Anosmia, Septal abscess, Epistaxis, Rhinonasal Mucormycosis, Voice change for study.

After obtaining the patient information from MRD files and filling it systematically, history, examinations and investigations done was studied regarding incidence, associated risk factors, age and gender distribution of ENT complications and same was recorded and analysed.

Distribution of ent complications among study participants:

In our study on post Covid ENT complications among 100 cases, mucormycosis was the predominant post Covid ENT complication which accounts for 92% (92/100) of cases.

And in study conducted by Muthu V, Rudramurthy SM, Chakrabarti A, Agarwal R et al [135] among 233 post covid patients,Overall, rhino-orbital mucormycosis was the most frequent presentation and accounted for 89% (206/233) of cases in India.

Age:

The overall mean age of our study participants with post Covid ENT complications was 52 ± 12 yrs.

In study conducted by Kundakarla Bhanuprasad et al [136] among 132 post Covid participants, The mean age of study participants who presented with ENT complication was 50.52 ± 11.66 .

And in studies on post covid vocal cord palsy by Tin S, Foo F, Breitling M, Saverimuttu J et al [137], epistaxis by Dell'Era et al [138] and anosmia by Vaira et al [139] the mean age of participants was 58 years, 71.5 years and 51.2 years respectively.

Gender:

In our study, Of the study population, who presented with ENT complications 78 % were males and 22 % were females .

In study conducted by Kundakarla Bhanuprasad et al [136], which included 132 participants, who presented with mucormycosis 78% were males and 12% were females.

Similarly in studies on epistaxis by Dell'Era et al [138] which included 30 cases 77% (23) were males.

Risk factor:

In our study Most patients (93%) had underlying diabetes mellitus as a risk factor which is higher than that (two-thirds of patients) found in a multicentric COVID-Mucor study by Patel et al [140] from India during the Covid-19 pandemic.

And in studies on post covid vocal cord palsy by Tin S, Foo F, Breitling M, Saverimuttu J et al [137] and anosmia by Vaira et al [139] majority patients had type 2 diabetes as underlying risk factor.

Treatment:

In our study of 100 cases, all the 92 mucormycosis patients underwent Endoscopic nasal debridement, incision and drainage done in all 3 septal abscess, rest of the 5 cases who presented with anosmia, epistaxis and vocal cord palsy was managed conservatively.

In a study by Muthu V, Rudramurthy SM, Chakrabarti A, Agarwal R et al [135] on post covid mucormycosis surgical treatment is done in about 168/233 (72.1%).

In a study on septal abscess in Covid case by Maan AS, Kaur G, Arora R et al [141] patient is managed by incision and drainage.

And in studies on post covid vocal cord palsy by Tin S, Foo F, Breitling M, Saverimuttu J et al [137], epistaxis by Dell'Era et al [138] and anosmia by klein et al [142] patients were managed conservatively.

Mortality and follow up:

In our study Among the 100 study participants, about 10 mucormycosis cases expired mainly due to post Covid complications. Remaining 90% of cases are in regular follow up.

In Garg D, Muthu V, Sehgal I, Ramachandran R, Kaur H, Bhalla A, et al study ofCOVID-19-associated mucormycosis, the overall reported mortality was 16.3%. [143]

CONCLUSION

- Mean age of participants in the study is 52 ± 12 yrs.
- Mucormycosis was the predominant post Covid complication among study population.(92%)
- Post covid ent complications more common in males with male to female ratio of 3.5:1
- Type 2 diabetes mellitus is the central risk factor for Covid associated complications.
- Among the study population, 95 % cases were managed by surgical intervention and 5 % of cases were managed conservatively.
- Post treatment 90 % of cases are in regular follow up.
- The mortality in treated patients is 10 % and is mainly due to Covid respiratory complications.
- Hence in conclusion early identification of these highly sensitive post Covid ENT complications is key to allow for optimal treatment and improved outcomes, to avoid mortality and morbidity among vulnerable populations.

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ANNEXURE

PROFORMA

VARIABLES RECORDED

NAME

AGE

GENDER

OCCUPATION

COMPLAINTS AND DURATION OF ILLNESS

PAST HISTORY

PERSONAL HISTORY

FAMILY HISTORY

GENERAL EXAMINATION

ENT EXAMINATION

- Complete head and neck examination
- Ear Nose Throat examination
- VII Cranial nerve examination

- Audiogram (if done)
- Imaging (MRI and HRCT if done)
- Complete blood counts, ESR, if Diabetic patient-Extended diabetic profile

From

Dr. KISHORE KUMAR P.,
MS ENT PG 2020 BATCH,
PSGIMSR

To

DIRECTOR - RESEARCH & INNOVATION
PROFESSOR & HOD, COM. MED.

PSGIMSR,

08.03.21

Respected mam,

I, Dr. Kishore Kumar P, am undertaking a study titled, "A Retrospective Study on ENT Complications in COVID-19 patients in a Teertiary Care Centre, Coimbatore".

I kindly request you to permit me to access Medical Record Department to obtain details of COVID patients for my thesis patient.

Thanking you,

Date: 2/3/2021

Yours faithfully,

P. Kishore

From,

DR.KISHORE KUMAR.P,

MS ENT PG 2020 BATCH,

PSGIMSR,

To whomsoever it may concern

I, DR.KISHORE KUMAR.P, am undertaking a study titled **'A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE '.**

I hereby assure that the data taken will be used only for the approved study and the subject information and subject identity will not be revealed to anybody under any circumstances.

Thanking you,

Yours sincerely,

Signature of PI

Signature of Co-investigator/s:

Date: 02-03-2021

SOP 15-V 1.2 / ANX 01-V1.0

Application form for requesting Waiver of Written Informed Consent/ waiver of consent

(To be filled by PI)

1. Proposal Number :
2. Principal Investigator's name: Dr.Kishore Kumar P
3. Department: Department of Otorhinolaryngology
4. Title of project:

A RETROSPECTIVE STUDY ON ENT COMPLICATIONS IN COVID-19 PATIENTS IN A TERTIARY CARE CENTER, COIMBATORE.

REASONS FOR REQUESTING WAIVER OF INFORMED CONSENT
Please check the reason(s) for requesting waiver

1. ✔ Research involves 'less than minimal risk'
2. ✔ There is no direct contact between the researcher and participant

I hereby assure that the rights of the participants will not be violated.

Following are the measures described in the Protocol for protecting confidentiality of data and privacy of research participant

1. **Never shall the patient's name/origin/photograph be used during the research process, it shall always remain confidential.**

Undertaking: I hereby declare that contents of the soft and hard copies of this document submitted to the IHEC are the same.

Principal Investigator's signature with date: P. Kishore

27/2/2021

LIST OF ABBREVIATIONS

SARS CoV 2	-	Severe Acute Respiratory Syndrome
ORL	-	Oto Rhino Laryngology
MRD	-	Medical Record Department
TESPAL	-	Transnasal Endoscopic Spheno Palatine Artery Ligation
SPF	-	Spheno Palatine Foramin
NSA	-	Nasal Septal Abscess
PAS	-	Periodic Acid Schiff
GMS	-	Gomori Methenamine Silver
HxE	-	Hematoxylin x Eosin Stain

ANNEXURE

ENT COMPLICATIONS IN POST COVID PATIENTS

S NO	IP/OP NO	NAME	AGE	SEX	COMORBIDITY	COVID +VE	ENT COMPLICATIONS	RX GIVEN	MORTALITY	FOLLOW UP
1	I21024332	Krishna Kumar	50	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
2	I21015255	Selvi Devi	53	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
3	I21005974	Kamala	56	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
4	I21016091	Venkataswamy	52	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
5	I21014690	Natarajan	62	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
6	I21016259	Jayanthinathan	50	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
7	O21026354	Selvakumar	42	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
8	I21016761	Dilip Kumar	32	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
9	I21015044	Tirumalaisamy	51	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
10	I21014894	Balasubramanian	48	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
11	I21016591	Sathish	37	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
12	I21017155	Muruganandham	48	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
13	I21017094	Yuvraj	38	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
14	I21017242	Murugan	56	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
15	I21017084	Ummar	60	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
16	I21017092	Periasamy	43	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
17	I21016956	Kokilavan	58	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
18	I21016945	Manoharan	49	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
19	I21017025	Selvi	55	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
20	I21016736	Senthilmani	41	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
21	I21017588	Bagiyaraj	40	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
22	I21017799	Ashok Kumar	41	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
23	I21017936	Vellingiri	57	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
24	I21017938	Srinivasan	46	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
25	I21017819	Sudalaimuthu	61	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
26	I21018032	Maariyappan	55	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
27	I21018157	Tirumalaisami	54	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
28	I21018449	Arjunan	34	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
29	I21018379	Yogeshwaran	45	M	Nil	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
30	I21018433	Muniyapasamy	46	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
31	I21018413	Kanagaraj	54	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
32	I21018722	Saraswathi	50	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
33	I21018881	Palanisamy	68	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
34	I21018948	Velumani	57	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
35	I21018942	Palanisamy	46	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
36	I21018763	Ravikumar	36	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
37	I21018980	Semmalaiyappan	58	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
38	I21019176	Moorthi	50	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
39	I21019272	Sasikala	60	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
40	I21019079	Sriram	18	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
41	I21018083	Murugavel	40	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
42	I21019401	Balachandran	54	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
43	I21017905	Yuvraj	42	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
44	I21019358	Periyasamy	44	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
45	I21017781	Gurupasupathy	42	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
46	I21019355	Saratha	57	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
47	I21019632	Chinnaraj	70	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
48	I21019542	Bagyam	66	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
49	I21019657	Nirmala	80	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
50	I21019768	Senthilkumar	46	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
51	I21019917	Selvaraj	60	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
52	I21020020	Rangathan	80	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
53	I21019369	Thirumoorthy	63	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
54	I21019971	Muthukumar	52	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
55	I21020202	Madhu	60	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
56	I21020507	Mohanraj D	63	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
57	I21029906	Arulkumar	40	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
58	I21020302	Padma	38	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
59	I21020651	Sachithanantham	58	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
60	I21020969	Subbalakshmi	57	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
61	I21019535	Ramesh	37	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
62	I21020583	Lakshmi	67	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
63	I21020412	Ilayaraj	44	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
64	I21027276	Moorthyrajan	37	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
65	I21021398	Ponnuvel	45	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
66	I21021489	Tamilselvi	47	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
67	I21021390	Abdul Sardar	37	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
68	I21021499	Ramasamy	75	m	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
69	I21022059	Sulochana	43	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
70	I21021832	Varadaraj	50	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
71	I21022408	Kalaichelian	68	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
72	I21021907	Chandrasekar	37	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
73	I21022728	Thulasiammal	68	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
74	I21022206	Krishnasamy	42	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
75	I21023129	Ramasamy	75	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
76	I21023976	Vasantha	56	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
77	I21027503	Sengutuvan	68	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
78	I21028113	Vadivel	43	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
79	I21006720	Balan	68	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
80	I21007195	Jeyachandran	67	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
81	I21008890	Chandran	42	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
82	I21014008	Mohanraj V	68	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
83	I21015350	Vishwanathan	63	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
84	I21014728	Jeyakrishnan	64	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
85	I21014953	Deivanai	72	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
86	I21014271	Velusamy	72	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
87	I21013614	Latha Prasad	55	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement	Expired	
88	I21015874	Dhanalkshmi	44	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
89	I21013483	Deivakarunai	75	F	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
90	I21016617	Balasubramanian T	54	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
91	I21016541	Arumugam	50	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
92	I210017481	Madan	41	M	T2DM	Positive	MUCORMYCOSIS	Endoscopic nasal debridement		+
93	I21010296	Muthukumar	55	M	T2DM	Positive	Septal Abcess	Incision and Drainage		+
94	I21015139	Sudhakar	47	M	Nil	Positive	Septal Abcess	Incision and Drainage		+
95	I21016098	Murali	59	M	Nil	Positive	Septal Abcess	Incision and Drainage		+
96	I21024232	Jaganathan	34	M	Nil	Positive	Epistaxis	Conservative		+
97	I21025792	Susheela	56	F	T2DM	Positive	Anosmia	Conservative		+
98	I21028749	Sultan Basha	42	M	Nil	Positive	Anosmia	Conservative		+
99	I21032751	Rangarajan	46	M	Nil	Positive	Vocal Cord Palsy - rt	Conservative		+
100	I21035395	Sulakshmi	42	F	Nil	Positive	Vocal Cord Palsy - rt	Conservative		+