# "A CLINICOPATHOLOGICAL STUDY OF FUNGAL DISEASES IN PATIENTS WITH CHRONIC RHINOSINUSITIS AND SINONASAL POLYPOSIS"

**A DISSERTATION SUBMITTED TO THE TAMILNADU**
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*In partial fulfilment of the Regulations*

*for the award of the Degree of*

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**DEPARTMENT OF E.N.T &HEAD AND NECK SURGERY**

**TIRUNELVELI MEDICAL COLLEGE**

**TIRUNELVELI**

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**THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED**

| | | |
|---|---|---|
| 1. | TIREC Application Form | ☐ |
| 2. | Study Protocol | ☐ |
| 3. | Department Research Committee Approval | ☐ |
| 4. | Patient Information Document and Consent Form in English and Vernacular Language | ☐ |
| 5. | Investigator's Brochure | ☐ |
| 6. | Proposed Methods for Patient Accrual Proposed | ☐ |
| 7. | Curriculum Vitae of The Principal Investigator | ☐ |
| 8. | Insurance /Compensation Policy | ☐ |
| 9. | Investigator's Agreement with Sponsor | ☐ |
| 10. | Investigator's Undertaking | ☐ |
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| 13. | Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) | ☐ |
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# INTRODUCTION

Chronic Rhino-Sinusitis (CRS) is a disease which involves persistent inflammation of the nose and the paranasal sinuses. The pathophysiology of CRS has been largely studied over the last 2 decades. But the exact etiology is ambiguous due to multiple host and environmental factors.The ubiquitous fungi are the most common causes resulting in many disorders including rhinosinusitis (1,2). Fungal sinusitis is observed in 5-10% of patients with chronic rhinosinusitis.

Fungal rhino-sinusitis (FRS) is divided into two groups: (A) Invasive and

(B) Non- invasive fungal rhino-sinusitis. Invasive diseases include: 1) acute invasive (fulminant) FRS; 2) granulomatous invasive FRS and; 3) chronic invasive FRS. The non- invasive diseases include:1) saprophytic fungal infestation 2) fungal ball and 3) fungus related eosinophilic FRS that includes allergic fungal rhino-sinusitis (AFRS).Patients groups belonging to various clinical settings are affected by both invasive and non-invasive .The Incidence of Allergic fungal sinusitis (AFS) among all rhinosinusitis requiring surgery is estimated to be 6-9%.

Findings of chronic rhino- sinusitis include central areas of increased contrast (hyper-attenuation) within abnormal paranasal sinuses can be seen in Sinus Computed Tomography . Patients with fungal rhino sinusitis often present with a history of chronic sinusitis cannot be treated by several courses of antibiotics.

FRS is being increasingly documented in people of all age groups.FRS creates an impact on both commercial socioeconomic status in the society. The patients who are affected with acute invasive FRS have high morbidity and have high mortality(3). Eventhough FRS is one of the major causes of Chronic rhino-sinusitis, it is often ignored and misdiagnosed mostly in developing countries such as India.

The existence of fungal hyphae in the sinus secretions can be demonstrated using fungal stains such as Gomori's methenamine silver (GMS) or 10% Potassium hydroxide (KOH) mount .The affirmation is done by culture on Sabouraud dextrose agar (SDA) with antibiotics. There fungi causing rhino-sinusitis exhibit demographic variation ;the dematiaceous fungi is the most common cause of rhino-sinusitis in the west, and Aspergillus is the most common cause of rhino-sinusitis in India. The organisms most commonly isolated are Aspergillus species like A. flavus or A. niger or demmatiaceous fungi such as Bipolaris spicifera or Curvularia lunata etc(4)

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Complete removal of allergic mucin which is done by Functional Endoscopic Sinus surgery is the gold standard treatment for Allergic Fungal Sinusitis. It facilitates permanent drainage and ventilation of the affected sinuses. FESS is an endoscopic surgery focused at restoring the function of the diseased paranasal sinus by re-establishing aeration and proper mucociliary clearance.

This present study is aimed at determining the prevalence of fungal sinusitis among patients undergoing FESS in this tertiary care hospital and also to know about the common fungi causing the disease in our region. This is done by subjecting the patients diagnosed with rhinosinusitis with or without polyposis for Functional Endoscopic Sinus Surgery and collecting the samples of the polypoidal mucosa from the affected sinuses per-operatively and they are tested for the presence of fungi by subjecting the samples to KOH mount and Fungal Culture

# REVIEW OF LITERATURE

## ANATOMY

To understand the pathophysiology and management of chronic sinusitis, a thorough Knowledge of the anatomy of the nose and paranasal sinuses is essential. Apart from providing pathway to the lower airway, its mucosa plays a key role in defense , air conditioning and humidification of the respiratory system

## NASAL CAVITY

It extends from external nares to choana. A pyramidal shaped Pyriform aperture constitutes the upper bony vault of the external nasal framework. The upper and the lower alar cartilages form the lower cartilaginous portion. The paired nasal cavity is divided into two by the septum with each half having a roof , floor , medial and lateral wall (5).

## NASAL SEPTUM

The nasal septum consists of a bony part, a cartilaginous part and small anterior membranous portion . The quadrilateral cartilage or the septal cartilage form the cartilaginous portion. It is 3-4 mm thick in the centre but antero-inferiorly the thickness increases to 4-8mm.The perpendicular plate of ethmoid and the vomer predominantly constitute the bony part which forms the anterior/superior part and the posterior/inferior part respectively. The anterior

membranous portion of the septum is attached to the medial crura of the lower lateral cartilages. Superiorly, the septal cartilage is connected to the upper lateral cartilages, just cranial to the domes of the lower lateral cartilages. It is also firmly attached to the nasal bones and to the perpendicular plate of ethmoid and vomer . Inferiorly , It sits on the crest of the maxilla. The perpendicular plate of ethmoid is continuous above with the cribriform plate and crista galli. The vomer articulates with the rostrum of sphenoid by two alae and inferiorly it articulates with the nasal crest of the palatine bone

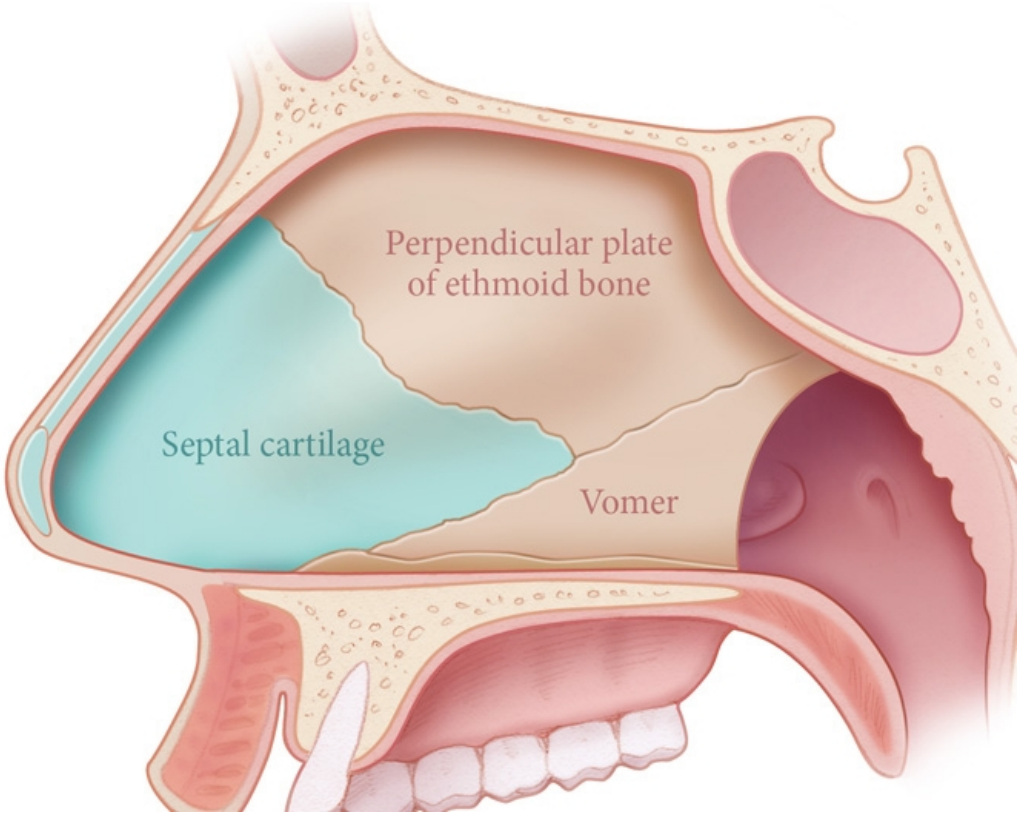


**Fig-1:Anatomy of nasal septum**

## LATERAL NASAL WALL

The lateral nasal wall comprises of three turbinates(inferior, middle and superior sometimes very rarely there is a supreme turbinate). The three turbinates divide the lateral wall into three meatus (inferior,middle and superior).

The paired inferior turbinates are curved and overhang from the lateral wall on either side. The inferior turbinate is composed of a medial mucosal layer(MML),a lateral mucosal layer(LML),and a central osseous layer inbetween[6]. The lining mucosa is similar to the rest of the nasal cavity– pseudostratified ciliated columnar epithelium and in addition to deeply situated basal cells and superficially ciliated and non-ciliated cells[6]**.** The lamina propria which forms the major portion of the IT lies between the epithelium and the periosteum of central osseus layer .It consists of a rich network of thin-walled venous sinusoids. The small-caliber venous sinusoids are located superficially .The inferior turbinate enlarges as a result of congestion of venous sinusoids and is a part of the normal physiological nasal cycle.The inferior turbinate is richly supplied by the branches of sphenopalatine artery.One to three large branches of the sphenopalatine artery run along the IT in a posterior–anterior direction and anteriorly anastomose with branches of facial artery[6].

The middle turbinate is complex structured. It is convoluted structure that bends

in three different planes. Depending on its attachment and its orientation in the three-dimensional space ,the middle turbinate is divided into three parts. The anterior one-third is in the sagittal plane and is attached above to the cribriform plate at the junction of the medial and lateral lamellae. It also has a small attachment anteriorly, to the frontonasal process of the maxilla. The middle one-third of the middle turbinate is oriented in the coronal plane and has its attachment laterally a to the lamina papyracea. This portion of the middle turbinate provides stability and hence it is called the ground lamella or the basal lamella. This ground/basal separates the anterior ethmoid air cells from the posterior ethmoid air cells[7].The posterior third lies in the horizontal plane and is attached to both the lamina papyracea and the perpendicular plate of the palatine bone extending upto the roof of the posterior choana.

Middle turbinate overlies the middle meatus. Within middle meatus anteriorly there is a sickle shaped curved ridge of bone called the uncinate process. Behind the uncinate process there is the well pneumatized and most constant anterior ethmoidal cell, namely the ethmoidal bulla. These structures are separated by a semilunar groove called the hiatus semilunaris. The hiatus semilunaris is a two-dimensional structure and it leads into the ethmoidal infundibulum, which is a three-dimensional space. The uncinate process, the bulla and the intervening infundibulum form the key area or the osteomeatal unit into which the frontal, the maxillary and anterior ethmoidal sinuses drain [8].

The term uncinate is derived from a Latin word, processus uncinatus, meaning a hooked outgrowth, and refers to a remnant of the descending portion of the first ethmoturbinal. The uncinate process is a thin plate of bone resembling a hook and it runs from anterosuperior to posteroinferior direction.It is oriented almost sagittally .The concave posterosuperior free margin of the uncinate is parallel to the anterior surface of the ethmoid bulla. It attaches to the perpendicular process (lamina perpendicularis) of the palatine bone and the ethmoid process of the inferior turbinate with bony spicules. The anterior margin of the uncinate which is convex ascends to the lacrimal bone, and sometimes to the skull base or lamina papyracea, remaining in contact with the bony lateral nasal wall. In some instances the uncinate process curves medially sometimes protruding out of the middle meatus.. In rare cases, the superior part of the uncinate process may attach with several "fingers" to the middle turbinate, the skull base, and the lateral nasal wall as well[9].

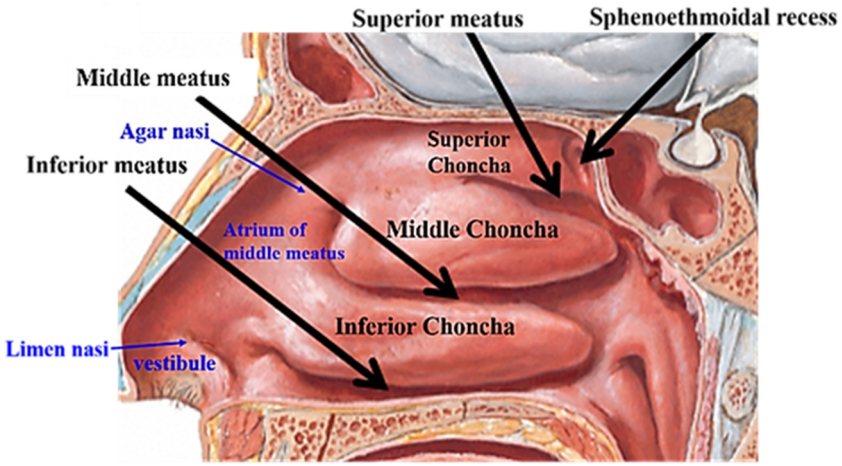


**Fig 2: Lateral wall of nasal cavity**

**Agger Nasi.**

The term is derived from the Latin term for nasal mound. It refers to the most superior remnant of the first ethmoturbinal, which persists as a crest or mound immediately anterior and superior to the insertion of the middle turbinate. An agger nasi cell results when this area of the lateral nasal wall under goes pneumatization. The Frontal recess which is close relation with the agger nasi air cell is narrowed depending upon its degree of pneumatization[10].

The ethmoidal bulla is usually a well pneumatized, most constant, anterior ethmoidal cell [11].In some rare instances, the bulla may be rudimentary or absent. A small recess called the Retrobullar recess seperates the bulla from the ground lamella. Sometimes the bulla does not extend superiorly upto the skull base and is separated from it by the suprabullar recess. The retrobullar and suprabullar recesses together form a semilunar space above and behind the bulla called the sinus lateralis of Grunwald[8].

The uncinate process hides the maxillary sinus which lies well within the ethmoidal infundibulum. The normal natural ostium of the maxillary sinus is usually ovoid and is related as follows. Inferiorly is the inferior turbinate,1 to 2 mm superiorly is the lamina papyracea and the orbit, posteriorly is the posterior fontanelle, 0.5 cm anteriorly lies the nasolacrimal duct.

The anterior fontanelle is double layer of mucosa without any underlying bone, found antero-inferior to the uncinate process. In a similar fashion, the posterior fontanelle is a mucosal layer that lies posterior and a little above the posterior attachment of the uncinate process. The mucosa in these fontanelles may sometimes be dehiscent to produce an accessory ostium of the maxillary sinus. The bulla has various patterns of drainage. It drains into the middle meatus, the hiatus semilunaris inferioris or into the sinus lateralis of Grunwald when present. Depending on the attachment of the uncinate process, the frontal sinus drains into the frontal recess either medial or lateral to the uncinate process. It can also drain into the suprabullar recess when it is present.

The maxillary sinus shows a constant drainage pattern and always drains into the infundibulum. The sphenoid sinus drains into the sphenoethmoidal recess.

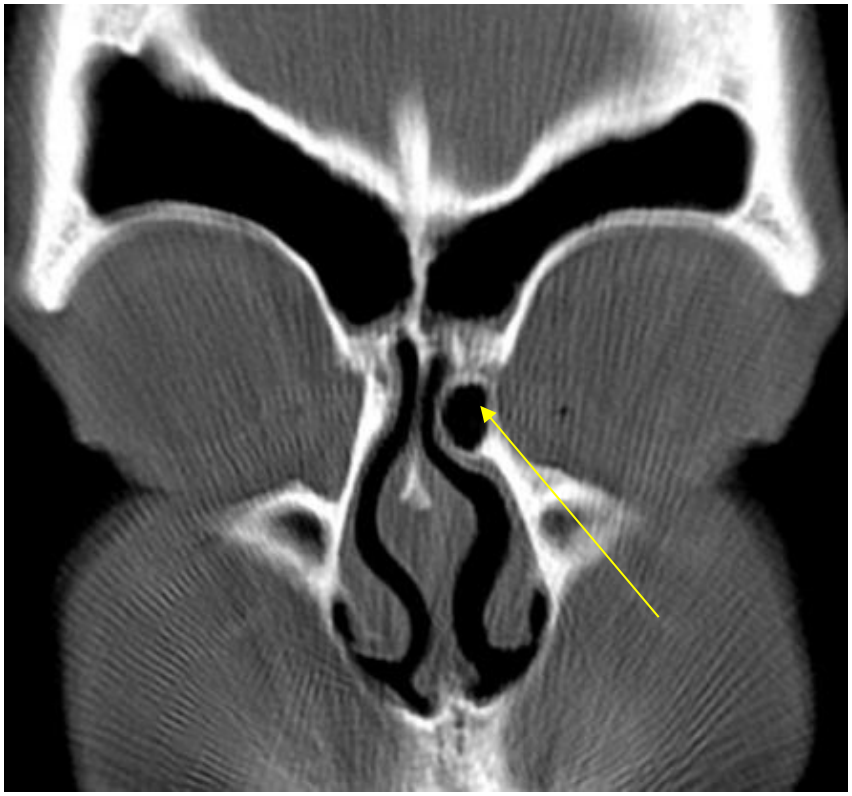

**Fig 3 :CT image showing the agger nasi air cell (arrow)**

**Frontal Recess**.

The frontal recess is perhaps the most complicated structure in the anterior ethmoid complex. It communicates with the frontal sinus superiorly. The middle turbinate forms the medial wall of the frontal recess mainly its most anterior and superior part. The lamina papyracea forms the lateral wall of the frontal recess. A discrete posterior margin exists when the basal lamella of the bulla reaches the skull base . It forms a discrete posterior margin, separating the frontal recess from the suprabullar recess. When the lamella of the bulla inserts far anteriorly and/or the bulla is well pneumatized, the frontal recess becomes narrowed. The frontal recess has the shape of an inverted funnel in sagittal section. While the

frontal recess and the frontal infundibulum together resembles the shape of an hourglass., with the natural ostium of the frontal sinus being the constricted portion [8].

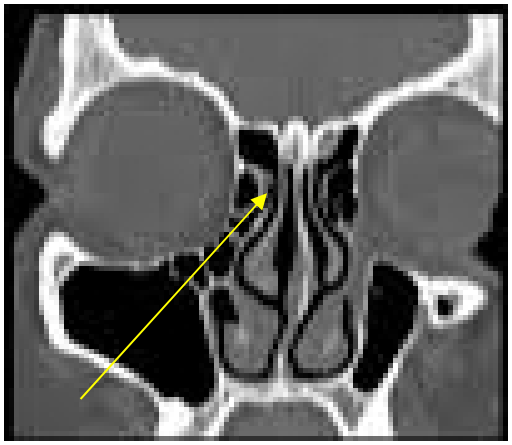

**Fig 4:CT image showing Frontal Recess(arrow)**

## PARANASAL SINUSES

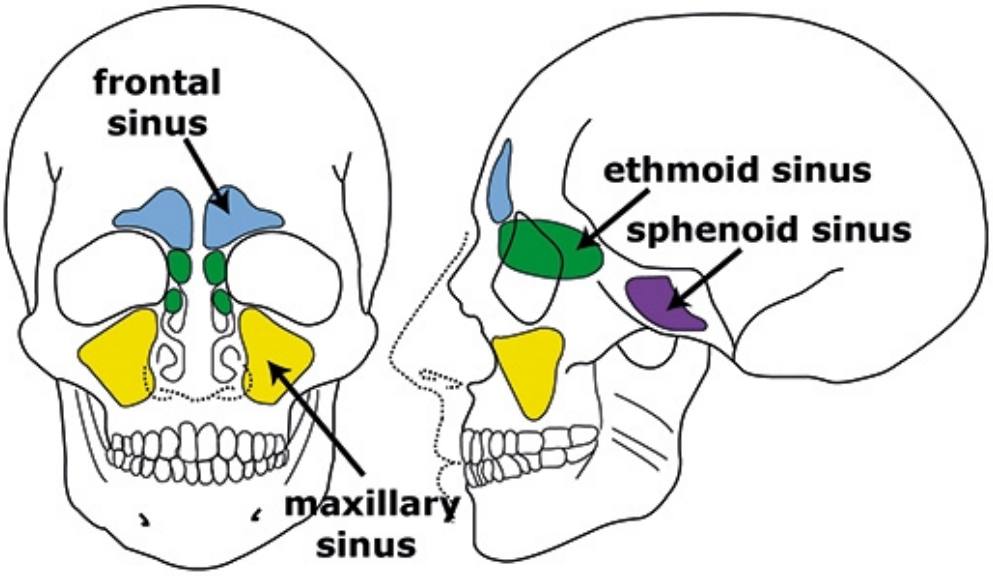


**Fig 5: Paranasal sinuses**

## MAXILLARY SINUS

The maxillary sinus is the largest and most constant of all the paranasal sinuses. It is the first sinus to develop in utero. After birth, the sinus passes through two periods of rapid growth, between birth and 3 years of life, then between ages 7 and 18 years. The maxillary sinus has the shape of a pyramid with an anterior wall corresponding to the facial surface of the maxilla. Its posterior bony wall of the maxillary sinus separates it from the pterygomaxillary fossa medially and from the infratemporal fossa laterally. Its medial wall is formed by the middle meatus mucosa, a layer of connective tissue and the sinus mucosa. It contains no bone. The floor of the maxillary sinus is formed by the alveolar process of the maxilla and the hard palate. The roof of the maxillary sinus corresponds to

the floor of the orbit. The presence of the infraorbital ethmoid cell, or Haller cell is the most common anatomical variation in the maxillary sinus .It is the pneumatized ethmoid air cell that project along the floor of the orbit, arising most often from the anterior ethmoids[10,11].

## THE ETHMOID LABRYNTH

The ethmoid sinus is the most compartmentalized paranasal sinus located between the lateral nasal wall and the medial orbital wall, lateral to the olfactory cleft and fossa. Initially only a few cells are pneumatized at birth, but during the course of adulthood their number can go beyond 15 cells. The ethmoid air cells have a honeycomb-like appearance with intricate passageways and blind alleys. Because of the complexity b of its anatomy it is referred to as the ethmoid labyrinth . The frontal bone in its posterior extension covers the roof of the ethmoid sinus-fovea ethmoidales. Along the roof of the ethmoid from lateral to medial runs The anterior and posterior ethmoid arteries, terminal branches of the internal carotid artery via the ophthalmic artery.

The ground or basal lamella of the middle turbinate, apart from defining the anatomical separation between the anterior and the posterior ethmoid cells, it also creates a bony septation that dictates the drainage pattern of the ethmoid cells. The anterior ethmoid air cells has its drainage pathway into the middle meatus while the posterior ethmoid air cells drains into the superior and

supreme meati. It thus represents the surgical posterior limit for an anterior ethmoidectomy[3,10].

## SPHENOID SINUS

The sphenoid sinuses are located at the base of the skull at the junction of the anterior and middle cranial fossae. Their growth starts as an invagination of the nasal mucosa into the posterior portion of the cartilaginous nasal capsulebetween the third and fourth months of fetal development. The sphenoid is primarily a pit in the sphenoethmoid recessbetween birth and 3 years of age.At age of three, Pneumatization of the sphenoid bone starts and it extends towards the sella turcica by seven years of age, and reaches its final form in the second decade.The two sinuses generally develop in an asymmetrical fashion, separated by the bony intersinus septum. Pneumatization of the sphenoid sinus can sometimes invade the anterior and the posterior clinoid processes, the vomer and also the posterior part of the nasal septum. The sphenoid sinus drains into the spheno-ethmoid recess through a single ostium: this ostium is classically situated 7 cm from the base of the columella at an angle of 30°with the floor of the nose in a parasagittal plane.

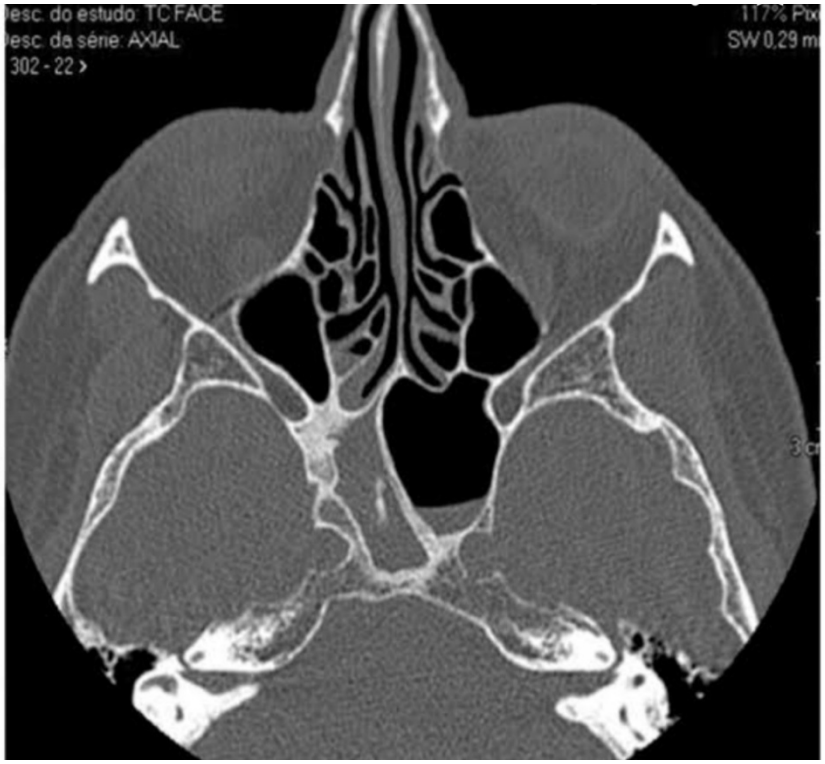


**Fig 6:CT Image (axial section) showing posterior ethmoid air cells & sphenoid sinus**

Depending on the extent of pneumatization, the sphenoid sinus can be classified into three types[8]**:**

1. Conchal: the area below the sella is a solid block of bone without pneumatization.
2. Presellar: the sphenoid is pneumatized to the level of the frontal plane of the sella and not beyond.
3. Sellar: the most common type, where pneumatization extends into the body of the sphenoid beyond the floor of the sella, reaching sometimes the clivus.

The lateral wall of the sphenoid sinus shows various prominences. Among them two most important prominence are, one for the carotid canal and the other for the optic canal: the internal carotid artery is the most medial structure in the cavernous sinus, and rests against the lateral surface of the sphenoid bone.The optic canal is found in the postero-superior angle between the lateral, posterior and superior walls of the sinus, horizontally crossing the carotid canal from lateral to medial [(12)].

**THE FRONTAL SINUS**

The frontal sinus is closely related to the anterior ethmoid in both its embryology and its anatomy. Usually at birth the frontal sinus, is a small blind pouch often indistinguishable from the anterior ethmoid cells. At 2 years of age, pneumatization of the frontal sinus begins and it becomes significant in early adolescence, and complete in the late teens. The right and left frontal sinuses are asymmetrical and they develop independently. The frontal sinus enclosed within the frontal bone between a thick anterior table and a relatively thin posterior table. The posterior table separates the sinus from the frontal lobe of the brain. It has the shape of a pyramid: its medial wall corresponds to a bony intersinus septum;while the floor of the frontal sinus corresponds to the anterior roof of the orbit. The frontal sinus opens in the nasal cavity at the level of the frontal recess , the drainage pathway resembling an hourglass shape.

## PHYSIOLOGY

## NASAL MUCOSA

The nose and paranasal sinus is a natural pathway for respiration and it also has pivotal physiological roles like olfaction, nasal resistance, conditioning of the inspired air, protection of the lower airway, vocal resonance, ventilation and drainage of the sinuses.

The mucosa of nasal cavity is lined mostly by the respiratory mucosa except for the anterior nasal vestibule which is lined by stratified squamous epithelium. Posterior to this the nasal cavity is lined by psuedostratified columnar epithelium which is about 120 $cm^2$ in area and around 0.3-0.5mm in thickness[13]**.** Columnar cells having around 300–400 microvilli on their surface forms up to 70% of the epithelium[13]. These microvilli play an important role in increasing the surface area and also help to retain moisture. Each cell has about 200-500 cilia on the surface, each cilia being 5 to 10 μm long and 250 nm thick[13].

The movement of cilia occurs by sliding filament mechanism which generates ATP.

Cilia and peri-ciliary spaces are covered by layer of mucus which is 10–15 μm thick. About 2L/ day of mucus is produced by nasal mucosa. The endonasal mucus is secreted by goblet cells and adjacent submucosal glands which form 5-

15 % of respiratory mucosa [13]. The cleaning of upper and lower airway by interaction of nasal mucus and ciliary beating is the main mechanism of mucociliary clearance. The physical, biochemical and chemical properties of the mucus along with the number, structure, co-ordinated stroke of the cilia are important parameters. The mucus is slightly acidic in nature with a pH-value of 5.5–6.5. The endonasal mucus has two layers. The outer viscous gel layer is composed of glycosylated macromolecules of very high molecular weight which forms a network of tangled polymers which is ideal for trapping inhaled debris. The deeper periciliary layer is lower in viscosity and is composed of mainly water and electrolytes. This deeper layer is the "Sol phase". Within the deeper periciliary layer there are mucins forming an apical glycocalyx extending 500-1500 nm from the epithelial cell surface. The sol phase is crucial for proper mucociliary transport and separating the mucus from the epithelial cell wall and membrane. If the sol phase is too short, the glycocalyx of the cell membrane will interact with the gel phase thus impairing the clearance of the mucus blanket[14].

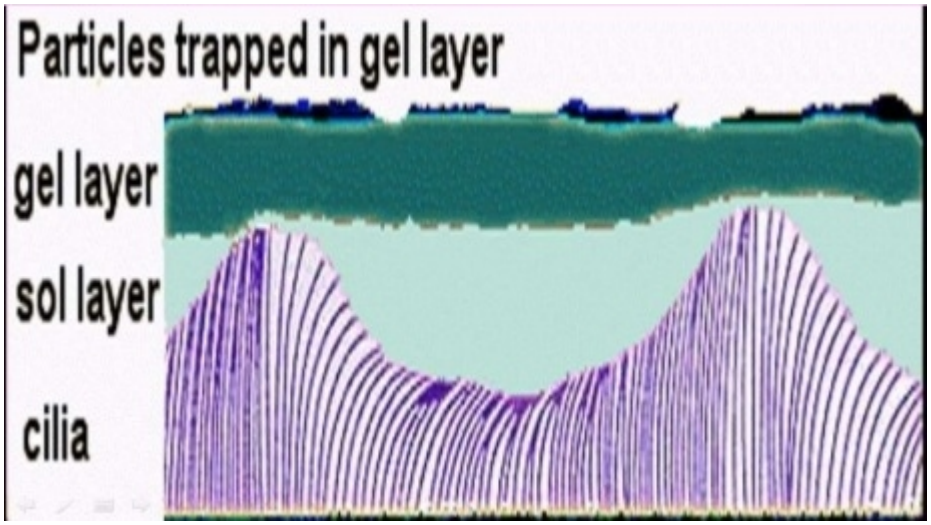


**Fig 7: Nasal mucosa with two layers**

## NASAL CYCLE

The nasal cycle is an alternation of nasal congestion and decongestion in humans which is autonomically mediated. This occurs via vasodilatation and vasoconstriction respectively once every 30 minutes to 6 hours [(15)]. When one nasal cavity is congested other is decongested, and is often asymmetrical but the total nasal airway resistance remains constant. This reciprocal is caused by alternate congestion and decongestion of the venous sinusoids that lines the nasal mucosa. These sinusoids are like erectile tissue and are particularly well developed in the anterior end of nasal septum and inferior turbinate **(6).** This mechanism is under the control of autonomic nervous system, while the sympathetic system controls the decongestive phase, parasympathetic system controls the congestive phase [(16)]**.**

## MUCOCILIARY CLEARANCE MECHANISM

Mucociliary clearance is very essential for modifying the physical condition of inspired air.

This includes humidification, clearing of impurities in the inspired air, filtering the air from noxious materials and protecting itself from organic and in organic substances.[15]

The cilia moves the mucus layer at a velocity of 2–25 mm/sec. The optimal conditions at which proper mucociliary clearance is usually achieved are a temperature of 37° C with 100% relative humidity. The mucociliary clearance mechanism occurs in a proper coordinated fashion from within the paranasal sinuses through the natural ostium into the nasal cavity beating posteriorly ultimately reaching the nasopharynx. A smooth uninterrupted mucociliary clearance depends on the viscosity of the mucous, integrity of the cilia and the absence of any obstructive cause in the outflow pattern of the mucociliary clearance pathway [14].

The mucociliary clearance mechanism provides an important line of defense by the surface fluids that contain macrophages, basophils and mast cells, eosinophils, interferons and lysozymes in addition to the physical removal of the inhaled debris

They discourage microbial colonization and enhance intrinsic protection[17].

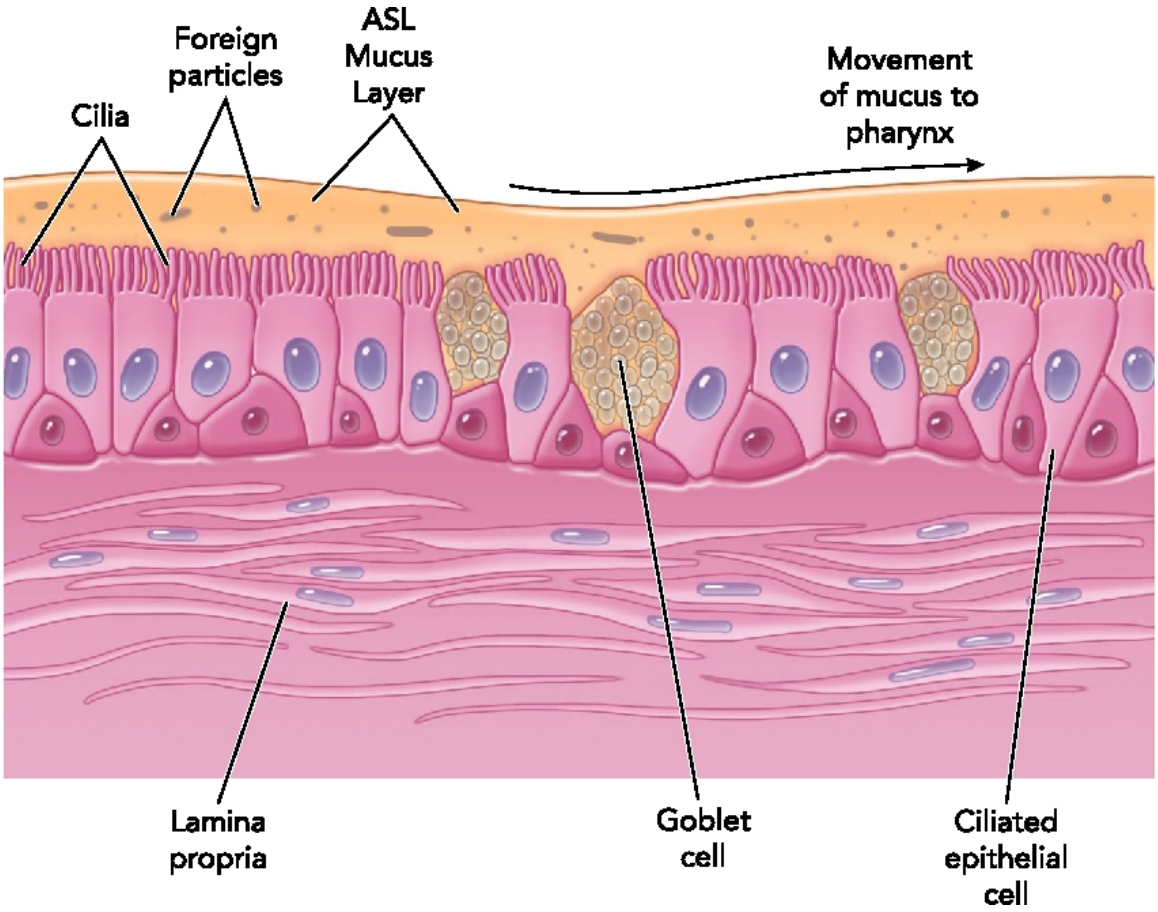


**Fig 8: Cilia beating towards the nasopharynx**

**Sinusitis**

Chronic rhinosinusitisis the presence of two or more symptoms one of which should be any of the symptoms like nasal blockage/ obstruction/ congestion or nasal discharge either anterior or posterior nasal drip with or without facial

pain/pressure and with or without cough for more than 12 weeks[18]. another definition put forth in 1996 by the Rhinosinusitis Task Force (RSTF) is that , the term CRS includes all inflammatory disorders of the nose and paranasal sinuses with a minimum duration of 12 weeks. Inspite of its increased prevalence and significant health impact, the exact etiology of chronic rhinosinusitis (CRS) still unknown whereas pathophysiology of acute bacterial sinusitis is well defined. CRS is a heterogeneous condition which is characterized broadly by persistent inflammation of the sinonasal mucosa.

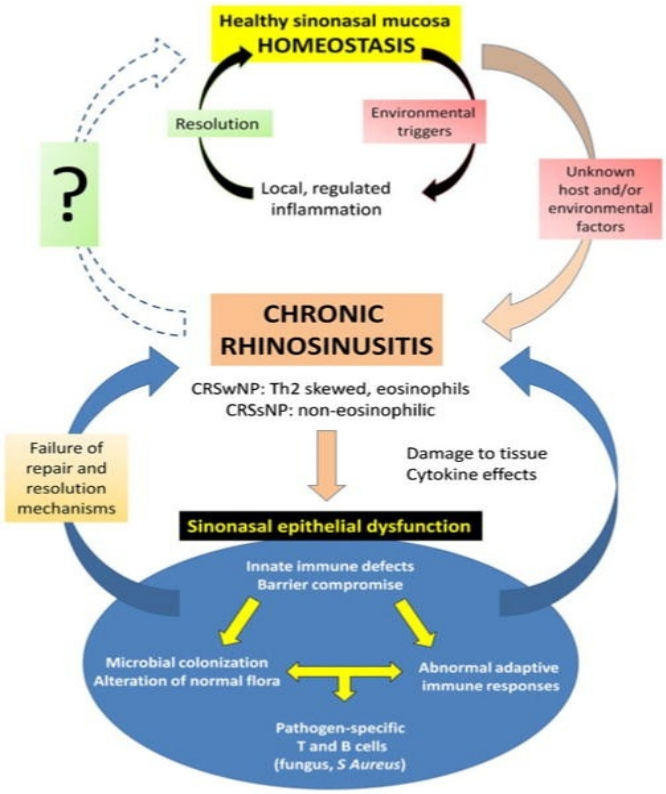


**Fig 9: Pathophysiology of Chronic rhinosinusitis**

Inflammation of the nasal and paranasal sinus mucosa for a long term is the hallmark of CRS(Benninger et al 2003). Eventhough it is called as 'chronic sinusitis', the term 'chronic rhinosinusitis' is more commonly being now used more frequently, because of the entire nasal and sinus passage involvement (Benninger et al 2003). Along with causing physical suffering, CRS also affects the physiological aspects of life. which was established by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) (Benninger et al 2003). But this definition did not mention a specific etiology for the eosinophilic inflammatory process.

Mucostasis and microbial colonization can be caused by inflammation of any origin disrupting the normal epithelial function. Infection will in turn stimulate further inflammation and aggravate the chronic disease process.

**CRS: Cellular Characteristics**

CRS is characterized by poor mucociliary function, with entrapment of thickened or purulent secretions within the sinus cavities. There are two types of CRS. One is Chronic Rhinosinusitis with nasal polyposis(CRSsNP) and the other is Chronic Rhinosinusitis without polyposis(CRSwNP). In many cases, medical or surgical restoration of sinus outflow in CRSsNP improves or even reverses mucosal disease, suggesting that functional or anatomic obstruction can serve as a primary inflammatory stimulus [10]. In contrast, enlargement of sinus ostia does not typically improve eosinophilic inflammation in CRSwNP,

although intensive medical therapy with systemic corticosteroids can dramatically, albeit transiently, reduce polyps[21,22]. The persistent effectiveness of steroids in CRS highlights the central importance of inflammation in the pathophysiology of the disease.

**Etiology and pathogenesis**

CRS has many proposed causes. It seems to mirror the increasing trends seen in allergic rhinitis and often results in a significant health burden for the individual and the society(18). It is most often associated with bacterial infection. Meltzer et al in 2004 cultured a variety of aerobic (staphylococcus spp., Gram-negative rods) and anaerobic (prevotella, fusobacterium, peptostreptococcus) bacteria from patients with CRS However, it is still unknown if bacteria are causing infection, or the inflammatory response is caused as a result of exposing the host to super antigens or the bacteria are able to colonize the pre-existing diseased sinus mucosa (Meltzer et al 2004).

The primary pathology is the disturbance in ostial function, especially in patients with nasal polyposis and this obstruction in the osteomeatal complex can be primary or secondary to other factors(19). Colonization of fungi in the paranasal sinus is a newly developed concept in the pathogenesis of CRS. Fungus is a common finding in the upper respiratory tract and it is due to the presence of fungal spores in the air, even of healthy individuals.. Allergic fungal rhinosinusitis (AFRS) is a type of CRS in which patients develop an allergic

response to the fungus colonizing the sinonasal cavities and thereby producing mucin. In contrast, fungus balls are caused by overgrowth of fungus in the nose and paranasal sinuses, without an inflammatory reaction (Meltzer et al 2004). The inflammatory reaction in response to a fungus ball is more of an irritative inflammation, like a foreign body reaction, i.e., giant cells, and not an eosinophilic inflammation, which is present in CRS.

Recent studies show a much broader role to fungi in CRS. It is proposed that in certain individuals, colonizing fungi stimulate a hypersensitivity reaction in the sinonasal mucosa that is non- immunoglobulin E (IgE)-mediated. Instead of an allergic response the fungi provoke a local inflammatory response with eosinophilic infiltration in some individuals. This condition has been termed Eosinophilic fungal rhinosinusitis (EFRS)

The mucin of the affected patients also contains fungal hyphae. In a study by Ponikau and colleagues (1999) ,they collected specimens from 210 patients with CRS (with or without polyposis) and found that 96% were culture positive for multiple fungi, the most common species being Alternaria (44%). Other recent studies using fungal DNA detection with PCR (Gosepath et al 2004) also shows similar results. Therefore fungi are present in patients with CRS and in healthy controls.In sensitized individuals alone, the fungi initiates an eosinophilic reaction, that targets the fungi present in the mucus and allows the degranulation of the eosinophils.

## Etiology

The etiologic studies of sinusitis are more concentrated on ostiomeatal obstruction, allergies, polyps, occult and subtle immunodeficiency states, and dental diseases. Microorganisms are often considered as secondary invaders. Any disease process or toxin that affects cilia has a negative effect on CRS.

### *Bacterial involvement*

The bacteria that are proposed to be the cause of CRS differ from those involved in acute rhinosinusitis. The following bacteria have been reported in samples obtained through endoscopy or sinus puncture in chronic sinusitis patients.

- Staphylococcus aureus (both methicillin-susceptible S.aureus [MSSA] and methicillin-resistant S.aureus [MRSA] strains)[20]
- Coagulase-negative staphylococci
- H.influenzae
- M.catarrhalis
- S.pneumoniae
- Streptococcus intermedius
- Pseudomonas aeruginosa
- Nocardia species

- Anaerobic bacteria (Peptostreptococcus, Prevotella, Porphyromonas, Bacteroides, Fusobacterium species [21] [22]

Unlike the microbes which are proposed to cause acute sinusitis, the exact roles of the microbes involved in chronic sinusitis remains unknown. There are many controversies among researchers regarding the etiology of chronic sinusitis . Much of the disagreement may be explained by methodology. Studies using suitable methods for recovery of anaerobes have proved their importance in chronic sinusitis but the studies which did not use such methods did not recover the anaerobes. So anaerobic bacteria can be recovered in 50 – 70 % of specimens when proper techniques are used. This variable presence of microbes in the specimens could be due to the prior usage of various broad spectrum antibiotics by the patients included in the study.

***Fungal involvement***

The following fungi have been reported in samples obtained with endoscopy or sinus puncture in patients with chronic sinusitis [23] :

- Aspergillus species
- Cryptococcus neoformans
- Candida species
- Sporothrix schenckii
- Alternaria species

**Risk factors**

The conditions and risk factors which predispose the patients to the develop chronic sinusitis are as follows:

- Anatomic abnormalities of the ostiomeatal complex (eg, septal deviation, concha bullosa, deviation of uncinate process, Haller cells)
- Allergic rhinitis
- Aspirin sensitivity
- Asthma
- Nasal polyps
- Non-allergic rhinitis (eg, vasomotor rhinitis, rhinitis medicamentosa, cocaine abuse)
- Defects in mucociliary clearance
- Naso-tracheal intubation
- Naso-gastric intubation
- Hormonal (eg, puberty, pregnancy, oral contraception)
- Obstruction by tumor
- Immunologic disorders (eg, common variable immunodeficiency, immunoglobulin A [IgA] deficiency, IgG subclass deficiency,AIDS)
- Cystic fibrosis
- Primary ciliary dyskinesia, Kartagener syndrome
- Wegener granulomatosis

- Repeated viral upper respiratory tract infections
- Smoking
- Environmental irritants and pollutants
- Gastroesophageal reflux disease (GERD). The reflux of gastric contents may play a contributing role in some cases of CRS; this relationship still needs to be better defined
- Periodontitis/significant dental disease

Systemic diseases (ie, granulomatosis with polyangiitis (Wegeners granulomatosis), Churg-Strauss vasculitis, Sarcoidosis

**Inflammation Induced by Microbial and Environmental Factors**

The sinonasal epithelium acts as the first line of defense mechanism against the inhaled pathogens and particulates as it in persistent contact with the outside environment. But the occasional presence of microorganisms and particulates is not necessarily pathologic. Although pre-existing inflammation with impairment of mucociliary clearance is responsible for retention of microbial and environmental particulates in CRS or these factors actually stimulate inflammation is still unclear.

**Mucociliary Dysfunction**

The ciliated columnar epithelium lines the paranasal sinuses and protects the paranasal sinuses by the continuously flowing mucus blanket which is made up of a complex network of immunoglobulins, carbohydrates, enzymes,

glycoproteins, electrolytes, and water. Any derangement in the mucociliary clearance may contribute to mucus stasis, infection, and inflammation. CRS is commonin systemic diseases such as primary ciliary dyskinesia (dysfunction of ciliary movement) or cystic fibrosis (dysfunction of mucus secretion),[24,25]. Cytokines and other inflammatory mediators present in CRS (eg, IL-8 and IL-13) may impact ciliary function [4].Those pathogens and particulates present in the respiratory tract also have been shown to affect the ciliary function and may attribute to pathologic inflammation [26].

Glycoproteins present in the mucus are important components of the mucus to determine the viscosity and their expression is altered in CRS.In both CRSwNP and CRSsNP, the genes MUC5AC and MUC5B are up-regulated, contributing to secretory cell hyperplasia and metaplasia. The Significantly increased concentration of the glycoprotein galactose β 1,3 GalNAc are illustrated in CRS sinonasal mucosa, perhaps contributing to the high viscosity of mucus and acting as potential innate receptors for pathogenic bacteria [6].

**Sinonasal Epithelial Cell Innate Immune Function**

The most important function of sinonasal mucosa is to give protection against infection. Innate and adaptive immune mechanisms act together to identify and remove the infectious threats from the sinonasal tract (Fig. 2). Innate mechanisms do not need prior antigenic exposure, nor do they rely on combinatorial re-arrangement of receptors.

The mucus blanket serves as the primary innate defense of the sinonasal tract. Lysozyme, lactoferrin, β-defensins, cathelicidins, and surfactant proteins which are secreted by the sinonasal mucosa inhibit microbial growth in the mucus, and may be up-regulated in response to activation of receptors by bacterial, fungal, or viral proteins. In recent years, there has been an increased focus on the potential role of the sinonasal innate immune system in the pathogenesis of CRS. In theory, innate immune system defects may predispose to infection and increased antigenic exposure or may stimulate inflammation directly via interaction with adaptive immune cells. Once inflammation occurs, innate mechanisms fail to promote resolution and repair which inturn may lead to persistence of the disease.

As like T and B cells, dendritic cells in addition are important components of the adaptive immune system within the sinonasal mucosa where they are likely play a critical role in local T- and B-cell differentiation. B-cell proliferation and antigen-specific IgE are present in increased concentrations in polyps. Increased B-cell activators and proliferation factors are usually described in patients with CRSwNP in comparison to those with CRSsNP and controls [23]. Increased proliferation and maturation of B cells promotes immunoglobulin isotype switch recombination, potentially exacerbating eosinophilic inflammation in CRSwNP. A relative deficit of myeloid dendritic cell subsets in CRSwNP has been thought to favor priming of T cells to a Th2

phenotype contributing to persistent inflammation [27] The epithelium communicates with dendritic cells via thymic stromal lymphopoetin and other inflammatory cytokines and signaling molecules.

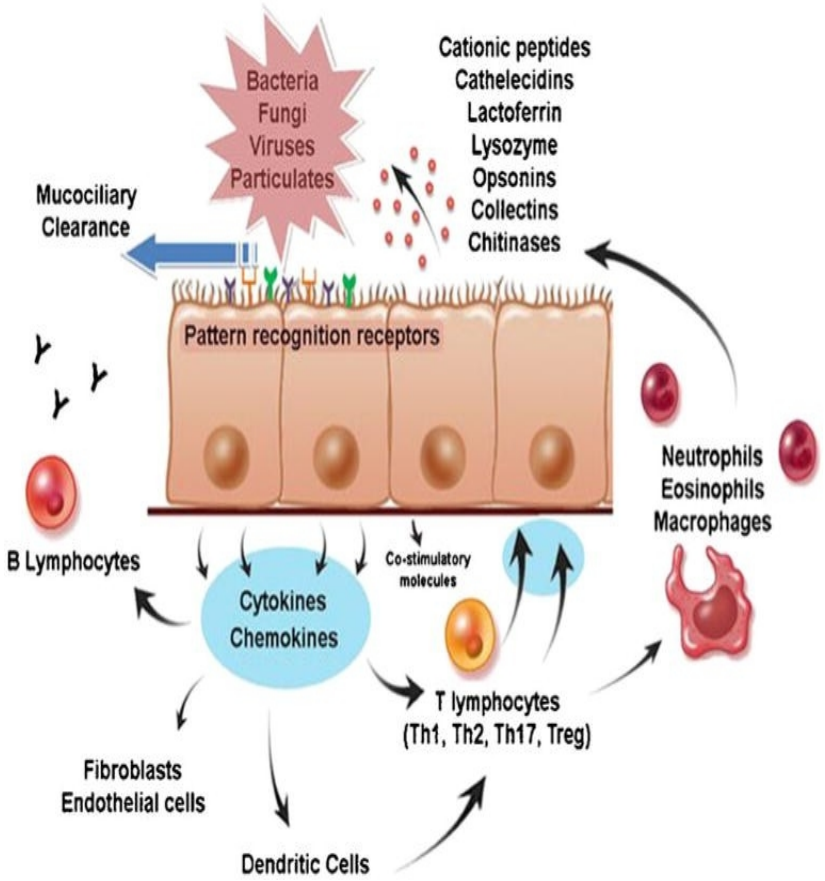


Fig 10: Sinonasal Epithelial Cell Innate Immune Function

**Diagnosis of Chronic Rhinosinusitis**

Nasal Endoscopy

Nasal endoscopy is the most preferred method to demonstrate any pathology at the level of the ostiomeatal complex, that are not visualized by anterior and posterior rhinoscopy. Nasal endoscopy might reveal pathologies in the nasal mucosa like swelling , secretions, and/or NPs at the ostiomeatal complex or spheno-ethmoidal recess.

Microbiological samples like swabs, aspirates, lavages and biopsies can also be obtained via nasal endoscopy .

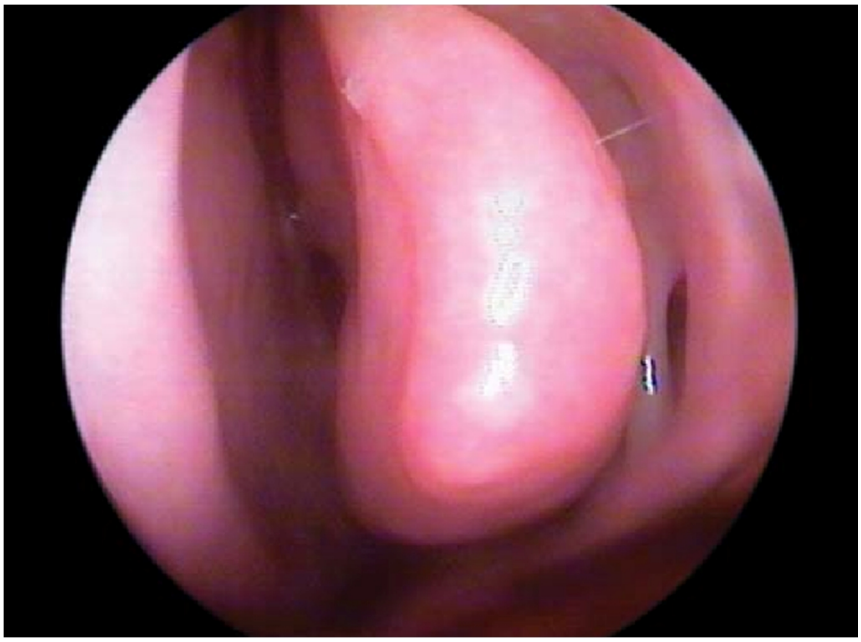

Fig 11: Diagnostic nasal endoscopic picture of the Left nasal cavity

Imaging in CRS

For quantifying disease severity and assessment of medical or surgical therapeutic response nasal endoscopy findings are often inadequate with respect to quantifying the extent of the disease in the adjacent sinuses and surrounding soft tissues, hence there is big significance of imaging in CRS.

X-rays of the paranasal sinuses were widely used before but now they offer a limited role as compared to Computed Tomographic (CT) images. CT scans of the paranasal sinuses, in addition to providing a diagnosis demonstrate the regional anatomy of the sinuses and provide a roadmap for the operating surgeon. Imaging is also vital in diagnosing the complications of CRS. Unlike ARS the complications in CRS are less dramatic. They mostly include mucocele formation, osteitis, bone erosion and expansion, metaplastic bone formation and optic neuropathy.

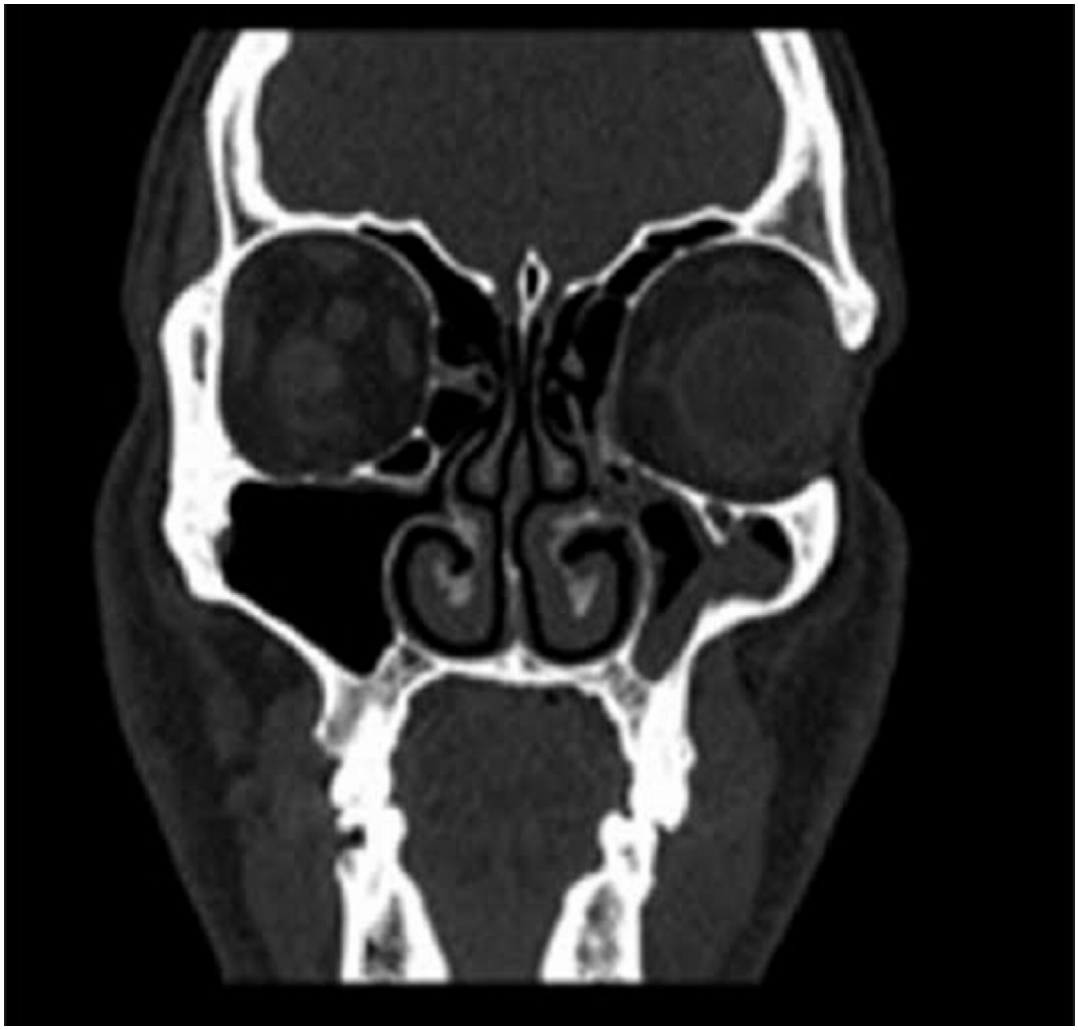

**Fig 12:CT image showing Frontal,Anterior ethmoid and maxillary sinus with left maxillary sinusitis**

## SURGICAL MANAGEMENT OF CRS

### FESS

For patients refractory to medical management, Endoscopic sinus surgery is the standard treatment . The surgery is aimed at removal of tissue obstructing the ostiometal complex (OMC) and the facilitation of ventilation and drainage preserving the normal non-obstructing anatomy and mucous membrane for mucosal regeneration. This procedure has evolved into a comprehensive procedure to address all sinuses. Thus the widespread removal of polypoid disease, osteitic bone, and removal of bony partitions has also become a part of FESS .

Maxillary sinus is best addressed using a 0° and 30° Endoscope. In The middle meatus ,the uncinate process is first removed. The uncinate process can be fractured anteriorly with a ball-tipped probe and a backbiting through-cutting instrument used to make an osteotomy along the junction of the superior two thirds and inferior one third of the uncinate process taking care to avoid injuring nasolacrimal duct. The superior and inferior portions are removed with a through-cutting instrument. The same can be achieved by using a microdebrider. The removal of the uncinate process reveals the infundibulum and the maxillary sinus ostium and the next procedure done is antrostomy.

The surgical technique of clearing ethmoid sinus begins with identification of ethmoid bulla . It is safest to start from medial and removed until the lamina papyracea is identified. The lamina papyracea mucosa is preserved. Once the ethmoid bulla is removed the basal lamella is penetrated just above its horizontal portion ,along its medial aspect .This is done to prevent destabilization of the middle turbinate, to avoid the branches of the sphenopalatine artery and to enter the posterior ethmoid air cells safely below the skull base.

Once the basal lamella is penetrated the diseased cells in the posterior ethmoids are then removed, meanwhile identifying the posterior ethmoid artery and posterior skull base. Anterior skull base boundaries are also elucidated, with the lamina laterally and superior turbinate medially.

The sphenoid sinus ostium which is located medial to the superior turbinate can be approached via two routes trans-ethmoidal or trans-nasal. In the trans-ethmoidal route after dissection of posterior ethmoids, a parallelogram (also known as Bolger's Box) is imagined - the boundaries being lamina papyracea, the skull base, basal lamella of the middle turbinate, and the superior turbinate(22). The sphenoid sinus is entered in the infero medial part of this triangle as it is considered safe and helps in avoiding the carotid artery and the optic nerve (23,24) .Through the ostia is approached via the 60 trans-nasal route , it is typically located 7 cm from the limen nasi and at an angle of

approximately 30° from the nasal floor or 2 cm superior to the choanae(22). The size of sphenoidotomy depends on the extent of the disease in the sphenoid sinus. Approach to the frontal sinus disease remains most challenging among the sinus surgeries because of increased risk of postoperative scarring and stenosis. The basic frontal sinus sinusotomy known as the Draf I procedure removes the anterior ethmoid cells and uncinate process without addressing the superior aspects of the frontal sinus.

When the agger nasi is removed, the anterior boundary of the frontal recess is removed. In addition to this, the superior attachment of the ethmoid bulla is removed to facilitate the posterior drainage. In Draf IIa procedure agger nasi is removed, along with anterior boundary of frontal recess and superior attachment of ethmoid bulla taking care to avoid traumatizing the middle turbinate as it may lead to iatrogenic frontal sinusitis (23). In Draf IIb procedure, in addition to the above steps the floor of frontal sinus along with anterior attachment of middle turbinate is removed. The Draf III procedure or endoscopic modified Lothrop procedure the superior nasal septum, frontal sinus floor, and intersinus septum are removed(21,23).It is not commonly performed as primary sinus procedure unless there is mucocele or significant disease involving frontal sinus. It carries significant higher risks of post operative complications as compared to simple frontal sinusotomy.

**Fungal sinusitis**

Fungus is present in all our surroundings and in the air we inhale but most healthy people do not react to the presence of fungus due to a functioning immune system. But, in rare instances, fungus may cause inflammation in the nose and the sinuses. Fungal sinusitis can occur in several forms, differing in pathology, symptoms, course, severity and the treatment required. It is broadly classified into invasive and non- invasive types. Classification of fungal rhino sinusitis into invasive / non-invasive is important for the accurate predication of prognosis and direct therapy [28]

Classification of fungal sinusitis :

A. **Non-invasive fungal sinusitis**

i. Fungus ball

ii. Allergic fungal Rhino-sinusitis

iii. Non-allergic fungal sinusitis

B. **Invasive fungal sinusitis**

i. Acute invasive fungal sinusitis

ii. Chronic invasive fungal sinusitis

iii. Granulomatous invasive fungal sinusitis

## NON-INVASIVE FUNGAL SINUSITIS

**Fungus Ball:** This is a non-invasive form of fungal sinusitis. Basically there is an overgrowth of fungal elements in the sinuses. Most commonly molds such as Aspergillus are responsible. The sinuses which are most commonly affected are the maxillary and the sphenoid sinuses, because the fungus finds favorable conditions such as warmth and humidity for their growth. Sometimes super-added infection in the sinus can be caused by bacteria affected by the fungus ball. Only a single sinus is involved in most of the cases, and the disease has a typical appearance on CT or MRI scans. Removal of the fungus ball through endoscopic sinus surgery is the treatment of choice

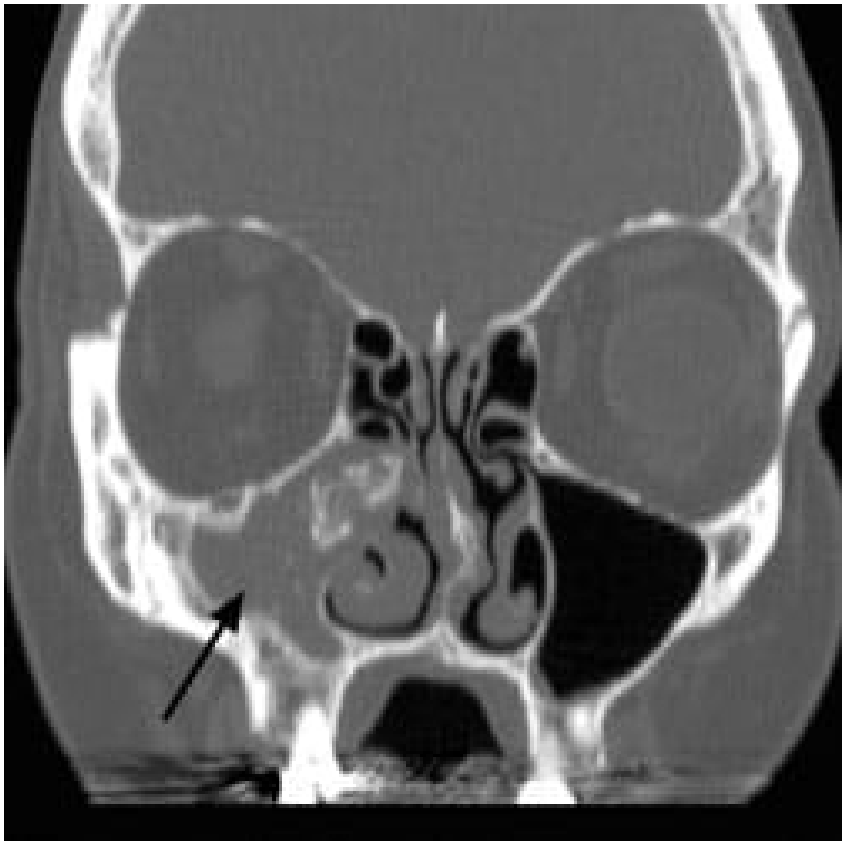

**Fig 13:CT showing Right Fungal Ball**

***Allergic Fungal Sinusitis (AFS):***

Patients develop allergic fungal sinusitis when they are allergic to certain fungi. In AFS, the common fungi belonging to the Dematiaceous family are usually involved. Most common are Alternaria, Bipolaris and Curvularia species. The fungus induces an allergic reaction in the sinuses, resulting in production of allergic mucin and nasal polyps. Mostly, the disease affects more than one sinus on one side.

However, in severe casesall sinuses on both sides may be involved. The most common presenting symptoms of patients with AIFS were facial swelling (64.5%), Fever (62-92%) and Nasal congestion (52.2%) **(31)**

Patients have a typical characteristic appearance on diagnostic nasal endoscopy i.e the presence of allergic mucin and polyps. Allergy testing to fungi is invariably positive. Sinus CT scans also show a typical appearance. Tissue examination under the microscope shows allergic mucin containing fungal elements without tissue invasion(34). The main aim of the endoscopic sinus surgical treatment is to clear all the polyps and allergic mucin present in all affected sinuses, and to restore the ventilation and drainage of sinuses. Endoscopic sinus surgery is now combined with aggressive medical therapy with nasal or systemic corticosteroids . Immunotherapy (allergy shots or drops) and antihistamines are also used in some patients and showed good results. Anti-fungal therapy is usually not required, as it is the reaction to the fungus

that needs to be modulated.

However the use of anti-fungal drugs in severe recurrent disease still remains a controversy .[32,33]

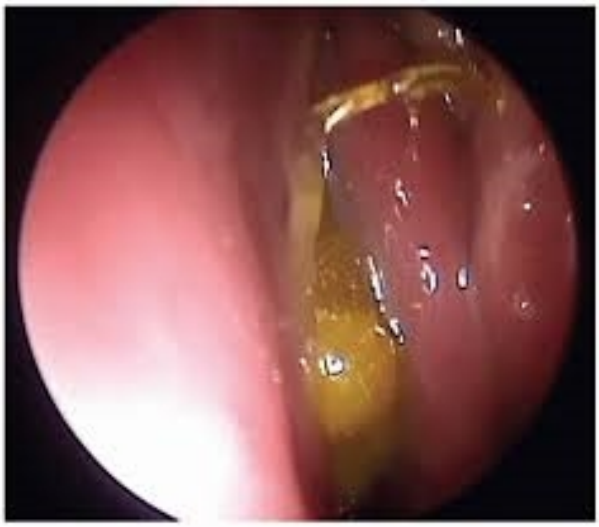

**Fig 14:Endoscopic picture of nasal mucosa with allergic mucin**

**Non-allergic fungal sinusitis:** In rare instances, mucin and fungus may be identified in patients with sinusitis who are non- allergic to fungus. Patients those have already had previous surgery in their sinuses might also house certain fungi [35]. Whether these fungi are responsible for sinus disease still remains unclear

***INVASIVE FUNGAL SINUSITIS***

Acute Invasive Fungal Sinusitis is the most life-threatening and dangerous form of fungal sinusitis. It is very rare, and mostly affects severely immunocompromised patients like patients with leukemia, aplastic anemia,

uncontrolled diabetes mellitus, and hemochromatosis. Patients undergoing anti-cancer chemotherapy or organ/ bone-marrow transplantation are also susceptible. Aspergillus or members of the class Zygomycetes (Mucor, Rhizopus) are the most common causative organisms. The disease often has an aggressive course, with fungus rapidly growing invading through sinus tissue and bone to extend into the surrounding areas of the brain and orbit. The orbit and anterior cranial fossa were the most common adjacent anatomic sites to exhibit disease extension. [30]

Areas of dead tissue and eschar are visualized in nasal endoscopy. Microscopically invasion of blood vessels by the fungus can be visualised causing tissue to die. Acute fulminant invasive fungal sinusitis in an immunocompromised host and Bacterial rhino sinusitis with intracranial or orbit extension are challenging to manage and constitute true emergences [29]

A combination of aggressive surgical and medical therapy is the treatment of choice.

Sometimes repeated and revision surgeries are needed to remove all dead tissue. As this is a deadly disease , medications such as anti- fungal drugs and those that help restore the immune status of the patient are given to improve survival rate,

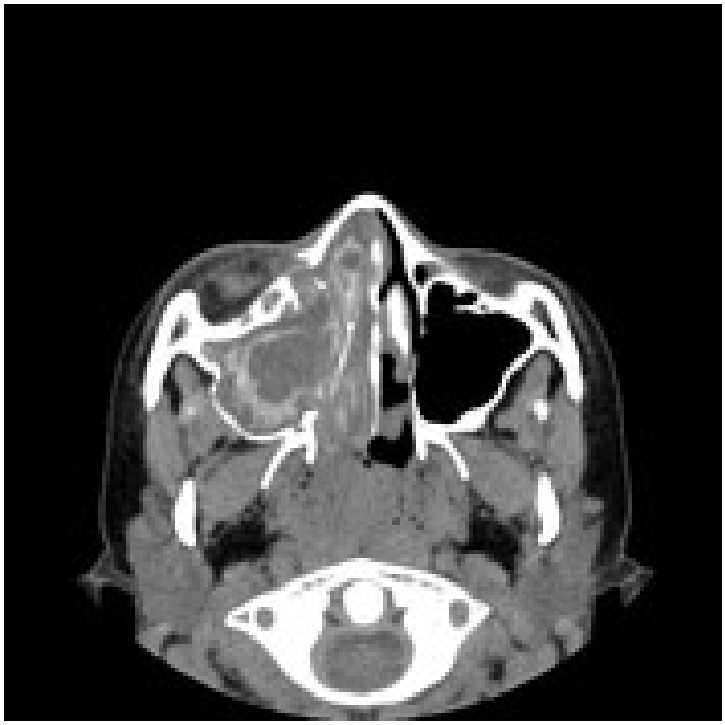

**Fig 15:CT (axial) image showing right invasive fungal sinusitis**

**Chronic invasive fungal sinus**: Chronic invasive fungal sinusitis, unlike acute invasive fungal sinusitis follows a slower and a less destructive course[36]**.** The disease rarely causes vascular invasion, minimal inflammatory reaction and restricted involvement of surrounding structures. It is predominant in patients with diabetes mellitus ,AIDS and patients on corticosteroid therapy for a long period . The disease involves the ethmoid and sphenoid sinuses, but sometimes any sinus may be involved. The duration of the disease is more than 3 months. Aspergillus fumigatus is the most commonly grown fungus in tissue cultures. Treatment involves surgery in combination with medical therapy (anti-fungal drugs and measures to restore the patient's immune system[37]

**Granulomatous invasive fungal sinusitis:** This form of fungal sinusitis is rare in the United States. It is more common in patients from Sudan, India, Pakistan and Saudi Arabia. It usually affects immunocompetent individuals . The disease duration is slow i.e over 3 months, and patients usually present with an enlarging mass in the nose,cheek, orbit and sinuses. Microscopically, it is characterized by presence of granulomas, which helps to differentiate it from chronic invasive fungal sinusitis. Aspergillus flavus is the usual causative organism. Treatment may involve surgery in combination with antifungal agents [(38)].

Fungal diseases of nasal cavity create major challenges for the physician, clinical microbiologist and basic scientists[(34,35)]. The incidence of mycotic infections and diversity of pathogenic fungi have been tremendously increased in recent years. Both Immunocompetent and immunocompromised individuals are at high risk of developing the disease. The disease manifests depending on the host's immune response, which can be defined as invasive (Acute granulomatous, chronic), non-invasive(Fungal ball, saprophytic) and allergic. Prognosis and therapy depend on the particular fungal manifestations and the host's immune response. Species of Aspergillus, Dematiaceous fungi, Zygomycetes are the most cases of Fungal rhino-sinusitis.

Inorder to predict the prognosis and to find out the effective therapy ,classification of fungal rhinosinusitis is very important and they are classified as follows

Table of classification of fungal rhino sinusitis

| Host | Immunocompromised | Immunocompetent | Atopic |
|---|---|---|---|
| Fungal Form | INVASIVE | 2)Granulomatous<br>3)Saprophytic<br>4)Fungal Balls | 5)AFRS |

Knowledge of fungal organisms is very important for appropriate anti-fungal therapy.

**Fungi/fungal infections**

Fungi are eukaryotic organisms that appeared before plants & animals. The total estimated number of fungal species exceeds more than 50000. They reside in nature and secrete enzymes that define and recycle organic matter.

The mechanism by which fungi cause disease is by releasing toxins or by inducing allergic response in the host [39]

Each and every fungal cell possesses at least one nucleus with be nuclear membrane, endoplasmic reticulum, mitochondria and secretory apparatus.

**Biology of fungi**

Fungi grow as yeast and moulds. Moulds are characterized by production of multicellular filamentous colonies consisting of branching cylindrical tubules that vary in diameter of 2-10 microns termed hyphae.

Mycelium is a mass of intertwined hyphae that accumulate during active growth in vitro. Hyphae penetrate the supporting medium and absorb nutrients are become vegetative/substrate hyphae [40]. In contrast, aerial hyphae project above the surface of mycelium and usually has the reproductive structures of the mould.

Yeasts are unicellular. They are usually spherical to ellipsoidal in space with a diameter of microns. Reproduction of yeasts is by budding. Pathogenic yeast produces a chain of elongated yeast cells called pseudohyphae. Some species of fungi are dimorphic and are capable of growth either as yeast or mould depending on the environmental condition.

**Concept of Fungal disease**

Initiation of an infection by fungi most commonly involves portal of entry, attachment to host cell, capacity to grow within the host, capability to replicate at 37^C, obtain sufficient nutrients and bypassing the host immune defense mechanisms.

Fungi which colonize on the surface of epithelial tissues without penetrating or invading the below surface are called non-invasive. These fungi may induce a profound inflammatory and immune response. Less potent irritants and enzymes, from the fungi, attack the host cell and lead to physiologic or inflammatory damage in rhino- sinusitis. Another mechanism of disease production in fungal rhino-sinusitis is induced by fungal cell wall

antigens that stimulate allergic response in the host[41,42,43]

Inhalation of fungal spores into the nasal passages. The outcome of inhaling fungal spore depends upon

- No of spores inhaled
- Size of fugal particles (influences the depth of penetration)
- Integrity of host immune response
- Pathologic virulence of fungus

People with impaired cell mediated immunity are most susceptible to mycotic infections[45] . AIDS, hematogenous dyscrasias, transplantation, long term cytotoxic drug and immunosuppressive drug users are at major risk to develop invasive/noninvasive forms of fungal rhino-sinusitis.

**Diagnosis of mycotic infections**

In the diagnosis of fungal infections, the fowling laboratory strategies may be employed.

1. Microscopic examination of specimen
2. Culture & staining
3. Serology & skin testing
4. X-ray(CT PNS)
5. Polymerase Chain Reaction (that detect specific fungal DNA)

The most common and definite methods are Direct microscopic method and culture.

***Specimens***

Specimens such as blood, spinal fluid, synovial, pleural, peritoneal, bone marrow, and biopsies of internal organs and non-sterile specimens such as skin, nail, hair, sputum, urine and paranasal sinuses.

***Microscopic Examination***

Specimens are examined directly by light microscopy. The ideal stain used is a mixture of KOH and calcofluor white. Staining with calcofluor white vastly increases the sensitivity. Exudates can be stained with hematoxylin and eosin, Gomori methenamine silver(GMS) or periodic acids/ Schiff(PAS). The Fontana-Masson melanin stain maybe useful to differentiate Dematiaceous fungi from Aspergillus. Size, morphology, quantity of fungal cell, melanin pigments are also noted during the microscopic examination[46,47].

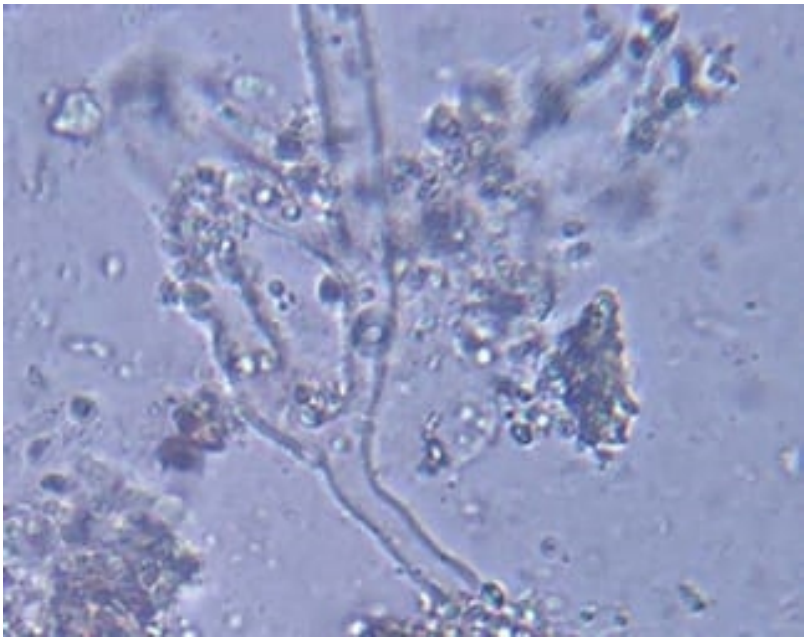

**Fig 16:KOH mount showing Mucor**

***Culture identification***

Most fungi occur in nature and grow readily on simple sources of nitrogen and carbohydrates. In the laboratory, the traditional medium used is Sabouraud's agar which consists of glucose and beef extracts( pH 5.0)[49]. The ideal temperature for culture is 30^C. Most agents causing fungal rhino-sinusitis are moulds.

***Serology***

Precipitins and specific IgE and IgG antibodies are detected in serum by radioimmunoassay and enzyme immunoassay. The antibody class and skin test results can help to differentiate among invasive, noninvasive and allergic fungal rhinosinusitis[52].

# AIMS AND OBJECTIVES

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## AIM

To study the prevalence of Fungal Diseases in all cases of Sinonasal polyposis and chronic rhino-sinusitis admitted for Functional Endoscopic Sinus Surgery in our Hospital.

## OBJECTIVES

1. To find out the prevalence of Fungal Sinusitis in cases of sinonasal polyposis/Chronic rhino-sinusitis
2. To determine the presence of fungal organisms in Chronic rhinosinusitis associated with nasal polyposis by both KOH mount and fungal culture.
3. To identify the type of fungal sinusitis in sinonasal polyposis/Chronic rhino-sinusitis.
4. To identify the fungal isolates most common in chronic sinusitis and sinonasal polyposis
5. To study the clinical and pathological manifestations of fungal infections of the nose and paranasal sinuses.
6. To find the association of Fungal sinusitis with Systemic diseases.

## MATERIALS AND METHODS

**Specimen –** sinus secretions and Polyps

**Sample Size –** 156

Patients of all age groups and of either sex who presented with radiologically proven sinusitis with symptoms > 12 weeks duration and undergoing functional endoscopic sinus surgery was included in the study. Patients were interviewed by structured questionnaire after obtaining informed consent. All the patients were also clinically assessed

***InclusionCriteria***

- All cases of CRS who underwent functional endoscopic sinus surgery in the department of ENT , TVMCH.
- All age groups
- Both male & female

***ExclusionCriteria***

- Patients who were on topical or systemic steroid for the past 1 month before the study period
- All cases with characteristic appearance of fungi in DNE and during surgery
- Cases with Clinically appearing Malignant Nasal mass and

Rhinosporidiosis

**Period of study –** 2years

**Duration :**November 2017 to June 2019.

***Ethical Committee Approval:***

The study was carried out after approval by the Institutional Scientific and Ethics Committee.

**METHODS OF COLLECTION OF DATA.**

- Detailed History taking and clinical examination.
- Relevant radiological investigations (CTscan).
- Obtaining a definitive diagnosis with the help of KOH mount and Fungal Culture reports

***Sample collection and processing***

During my study period , samples of nasal sinus tissue, sinus secretions and allergic mucin from patients undergoing FESS were subjected to mycological culture.

The specimen was collected in sterile saline per operatively and taken to the microbiology lab as early as possible and was processed on the same day.

The sample was be subjected to direct microscopy with 10% potassium hydroxide and culture.

***KOH mount***

The material was teased and placed on a clean glass slide and a drop of 10% KOH added.

A cover slip was placed and the preparation left at room temperature for tissue digestion and then examined by microscopy.

The presence of fungal hyphal elements was be noticed.

**Fungal culture**

The specimen was inoculated in duplicate on Sabouraud's dextrose agar with Gentamicin and Chloramphenicol

The inoculated media was incubated both at 25˚C and 37˚C.It was observed daily for one week and then twice weekly for three more weeks.

Once fungal growth occurred it was identified by observing its macroscopic and microscopic morphology.

The microscopic morphology was studied by Lactophenol Cotton Blue Mount.

Slide culture was done when the morphology is unclear and species identification was not possible in LPCB mount.

## OBSERVATION & RESULTS

The present study of fungal sinusitis was carried out in the Department of ENT , Tirunelveli Medical College, Tirunelveli, over a Period of two years from November 2017 to July 2019. All clinically diagnosed cases of chronic rhinosinusitis in all age groups and of both sexes, attending Otorhinolaryngology OPD & undergoing Functional Endoscopic Sinus Surgery were taken for this study Written informed consent was taken from all the patients prior to surgery.

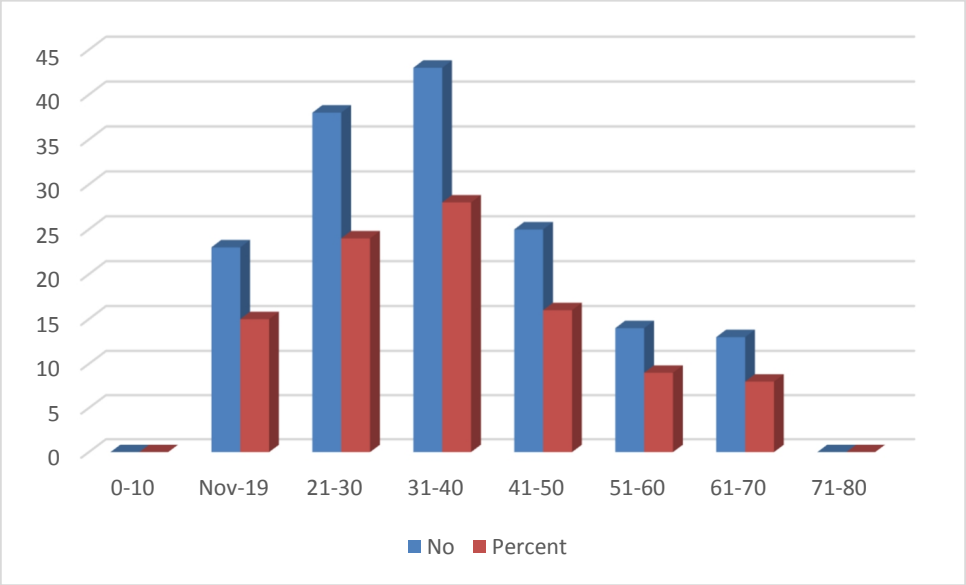
Data was included in a predesigned proforma. It included patient's identification number, name, age, sex, patient's history, clinical presentation, clinical assessment and microbiological diagnosis.

Aspergillus flavus was the most common isolate, while Aspergillus fumigatus, *& Mucor* were the other isolates.

## Age distribution of patients with CRS

## (Table 1 & Graph 1)

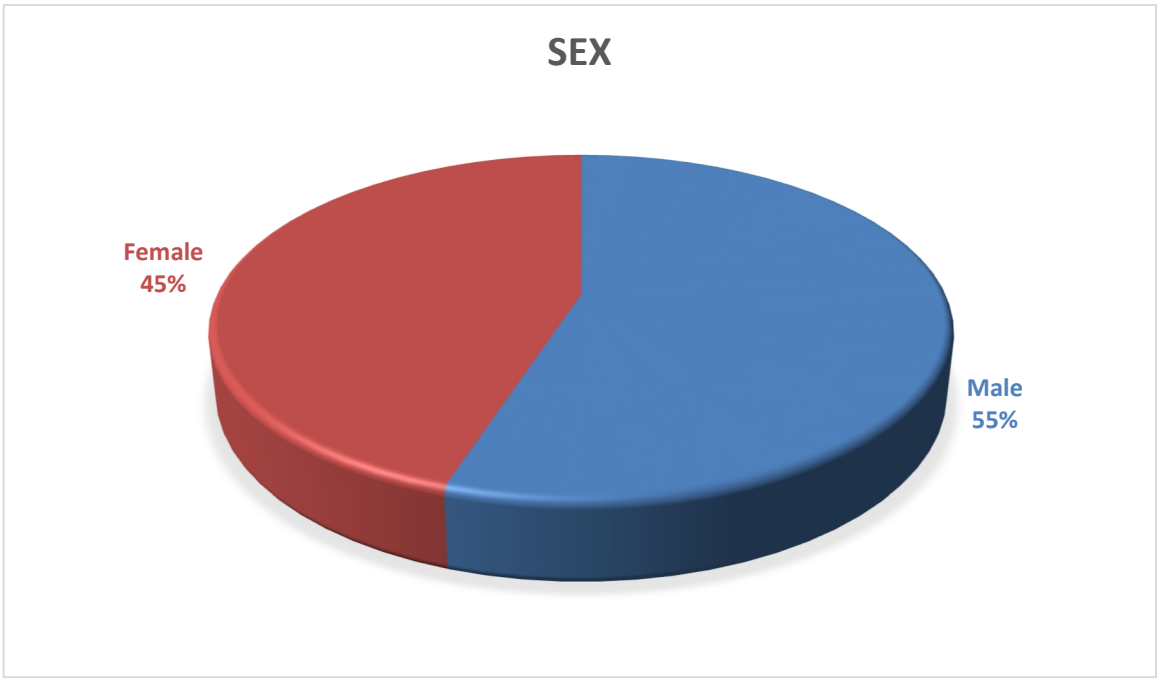
| Range | No | Percent |
|---|---|---|
| **0-10** | 0 | 0 |
| **11-20** | 23 | 15 |
| **21-30** | 38 | 24 |
| **31-40** | 43 | 28 |
| **41-50** | 25 | 16 |
| **51-60** | 14 | 9 |
| **61-70** | 13 | 8 |
| **71-80** | 0 | 0 |



**Sex Distribution of patients with CRS**

**(Table 2 and Graph 2)**

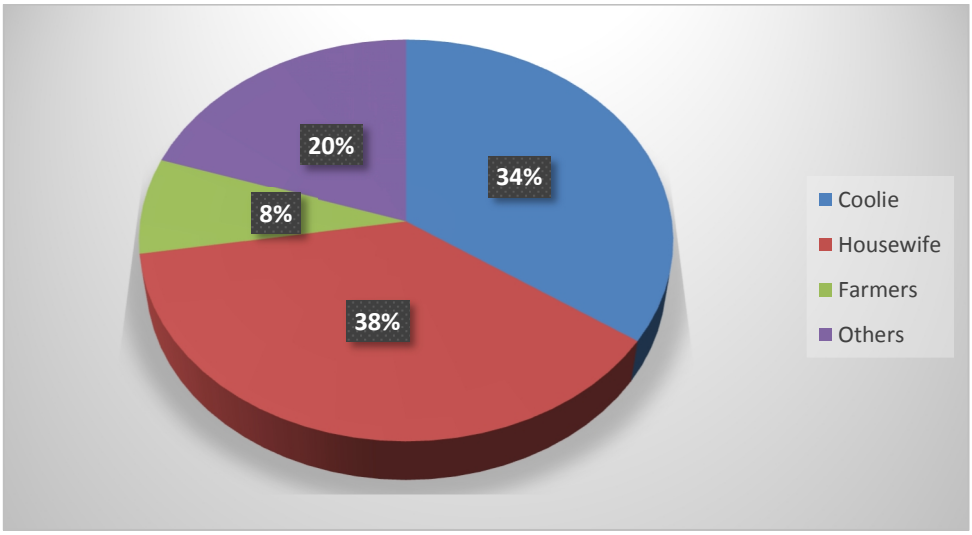
| Sex | No. | % |
|---|---|---|
| **Male** | 86 | 55 |
| **Female** | 70 | 45 |



## Occupation based distribution of patients with CRS

*(Table 3& Graph 3)*

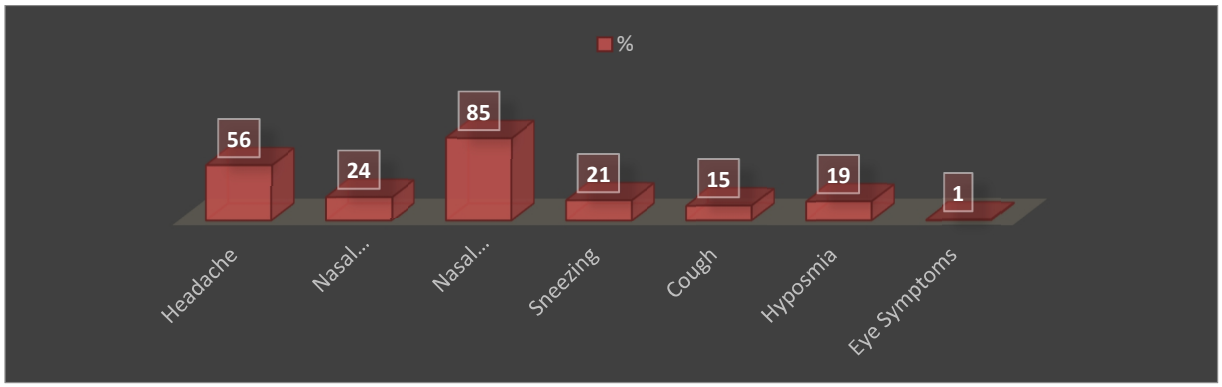
| Occupation | No. | % |
|---|---|---|
| Coolie | 59 | 38 |
| Housewife | 54 | 34 |
| Farmers | 12 | 8 |
| Others | 31 | 20 |



**Presentation of symptoms in patients withCRS**

***(Table 4 & Graph 4)***

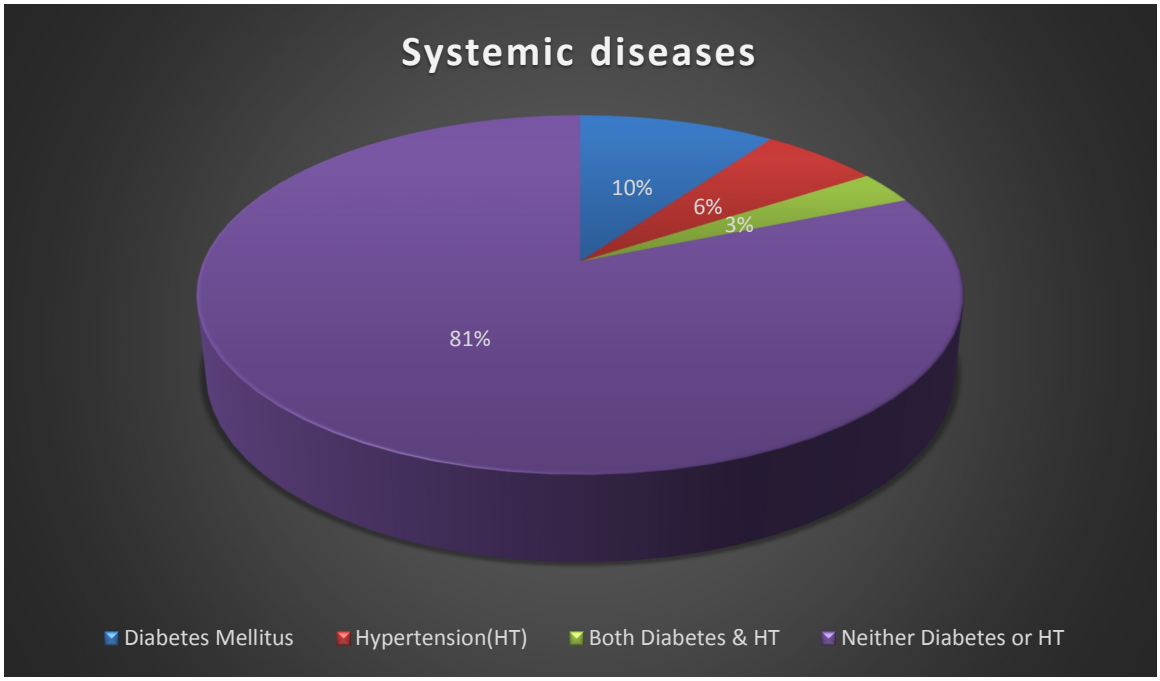
| Symptoms | Yes | % |
|---|---|---|
| **Headache** | **88** | 56 |
| **Nasal Discharge** | **37** | 24 |
| **Nasal Obstruction** | **132** | 85 |
| **Sneezing** | **32** | 21 |
| **Cough** | **23** | 15 |
| **Hyposmia** | **30** | 19 |
| **Eye Symptoms** | **1** | 1 |



**Systemic Diseases in patients with CRS**

***(Table 5 & Graph 5)***

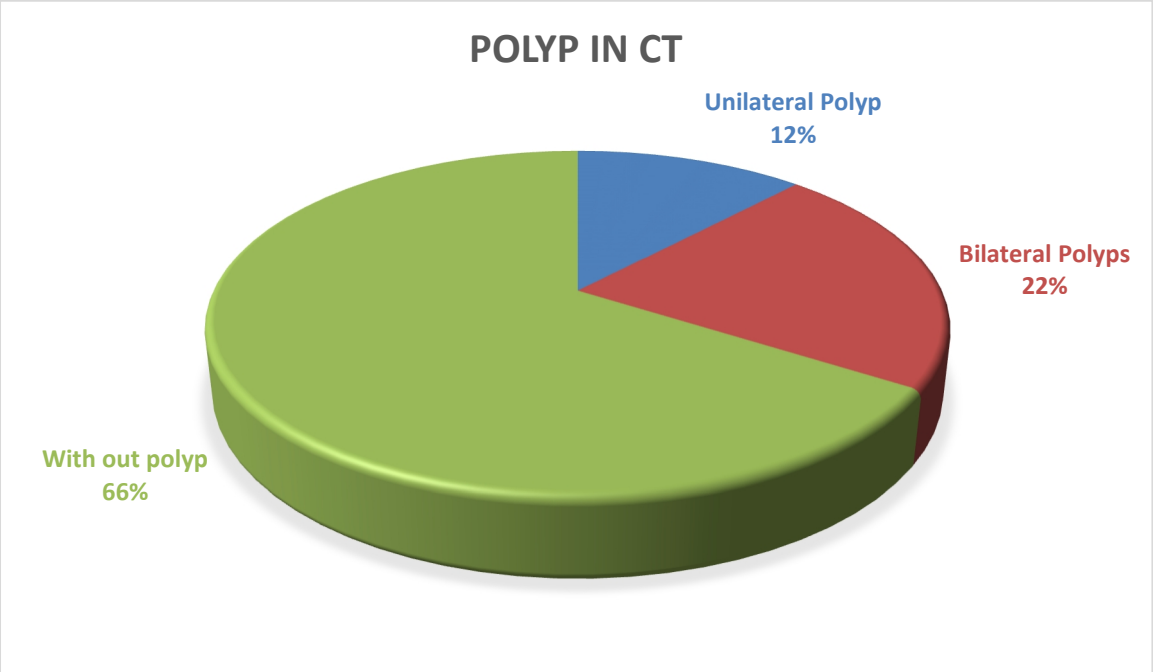
| Systemic diseases | Number | % |
|---|---|---|
| **Diabetes Mellitus** | 15 | 10 |
| **Hypertension(HT)** | 9 | 6 |
| **Both Diabetes & HT** | 4 | 3 |
| **Neither Diabetes or HT** | 128 | 82 |



**Polyp based on CT findings among CRS patients**

***(Table 6.1 &Graphs 6a )***

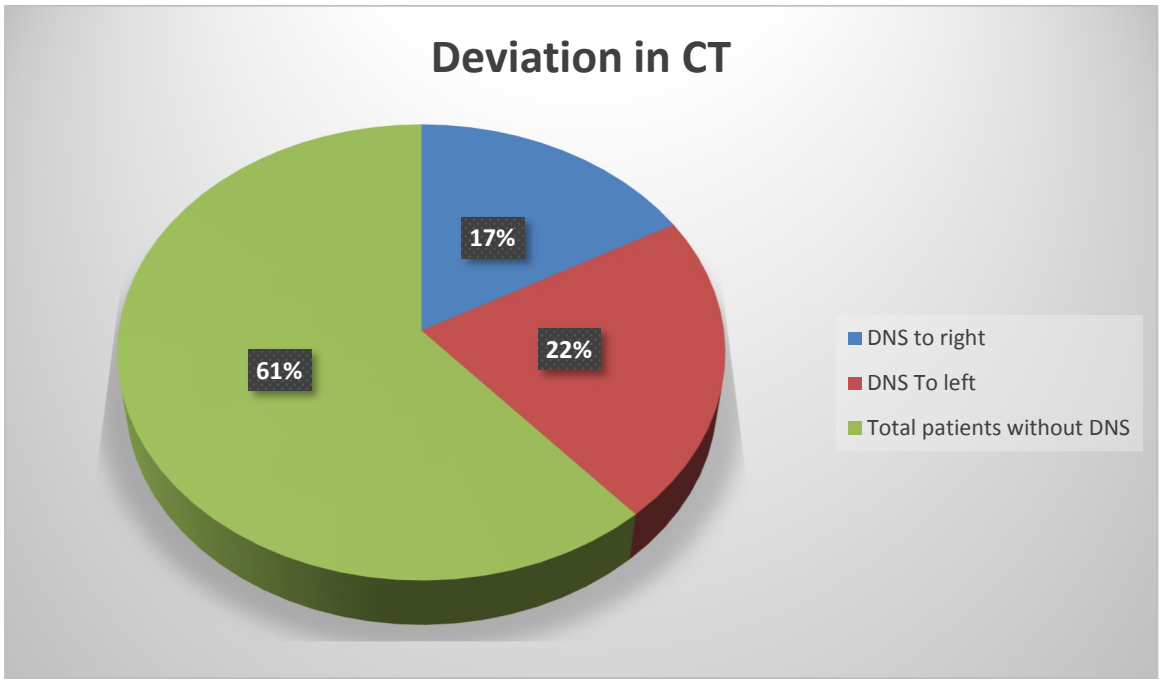
| **Polyp in CT** | **No.** | % |
|---|---|---|
| **Unilateral Polyp** | 19 | 12 |
| **Bilateral Polyps** | 34 | 22 |
| **With out polyp** | 103 | 66 |



**Deviated nasal septum among CRS patients in CT**

***(Table 6.2 &Graphs 6b)***

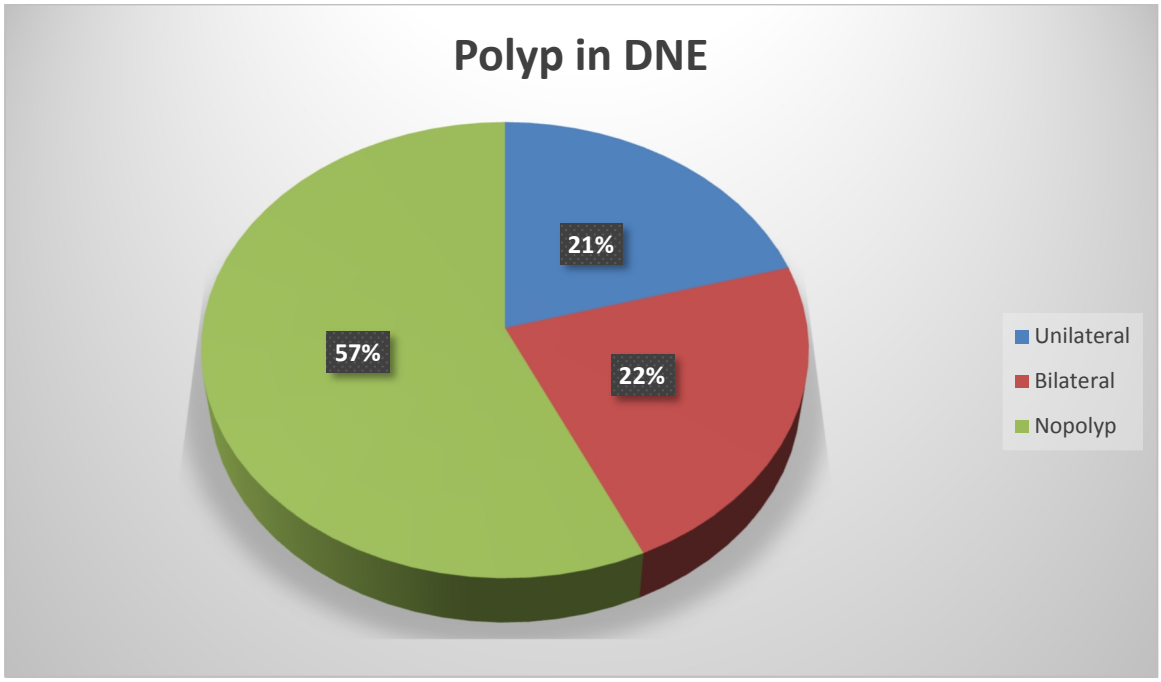
| **Deviation in CT** | **No.** | % |
|---|---|---|
| **DNS to right** | 26 | 17 |
| **DNS To left** | 34 | 22 |
| **Total patients without DNS** | 96 | 62 |



**Polyps in Diagnostic nasal endoscopy**

**(Table 7 and Graph 7)**

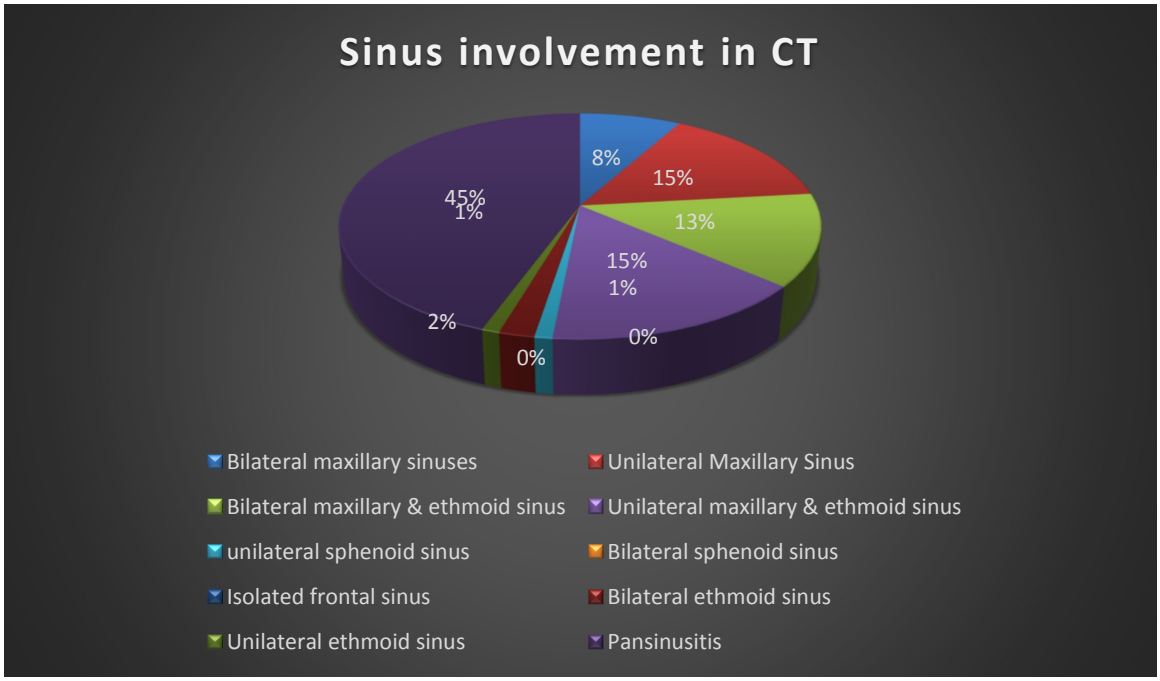
| **Polyp in DNE** | **No.** | % |
|---|---|---|
| **Unilateral** | 33 | 21 |
| **Bilateral** | 36 | 23 |
| **Nopolyp** | 87 | 58 |



**Involvement of sinuses among CRS patients based on CTfindings**

***(Table 8 & Graph 8)***

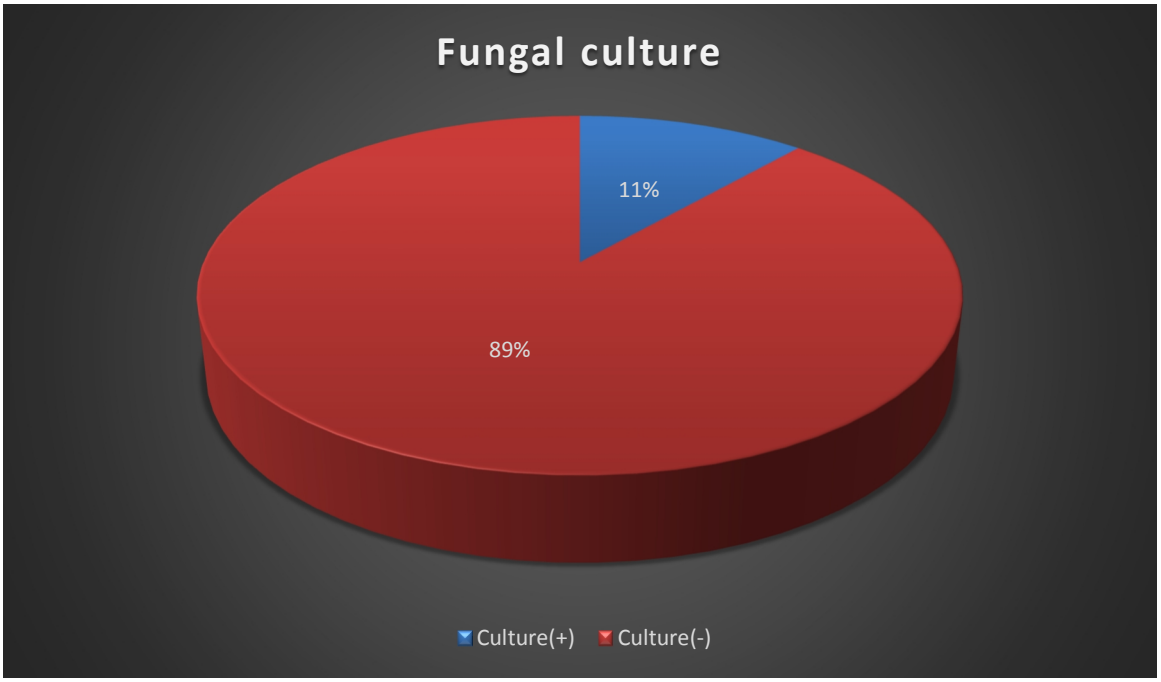
| **Sinus involvement in CT** | **No.** | % |
|---|---|---|
| **Bilateral maxillary sinuses** | 13 | 8 |
| **Unilateral Maxillary Sinus** | 24 | 15 |
| **Bilateral maxillary & ethmoid sinus** | 20 | 13 |
| **Unilateral maxillary & ethmoid sinus** | 24 | 15 |
| **unilateral sphenoid sinus** | 1 | 1 |
| **Bilateral sphenoid sinus** | 0 | 0 |
| **Isolated frontal sinus** | 0 | 0 |
| **Bilateral ethmoid sinus** | 3 | 2 |
| **Unilateral ethmoid sinus** | 2 | 1 |
| **Pansinusitis** | 69 | 44 |


Sinus involvement in CT
8%
15%
13%
15%
1%
0%
0%
2%
45%
1%
Bilateral maxillary sinuses
Unilateral Maxillary Sinus
Bilateral maxillary & ethmoid sinus
Unilateral maxillary & ethmoid sinus
unilateral sphenoid sinus
Bilateral sphenoid sinus
Isolated frontal sinus
Bilateral ethmoid sinus
Unilateral ethmoid sinus
Pansinusitis

**Culture result of clinical samples**

***(Table 9 & Graph 9)***

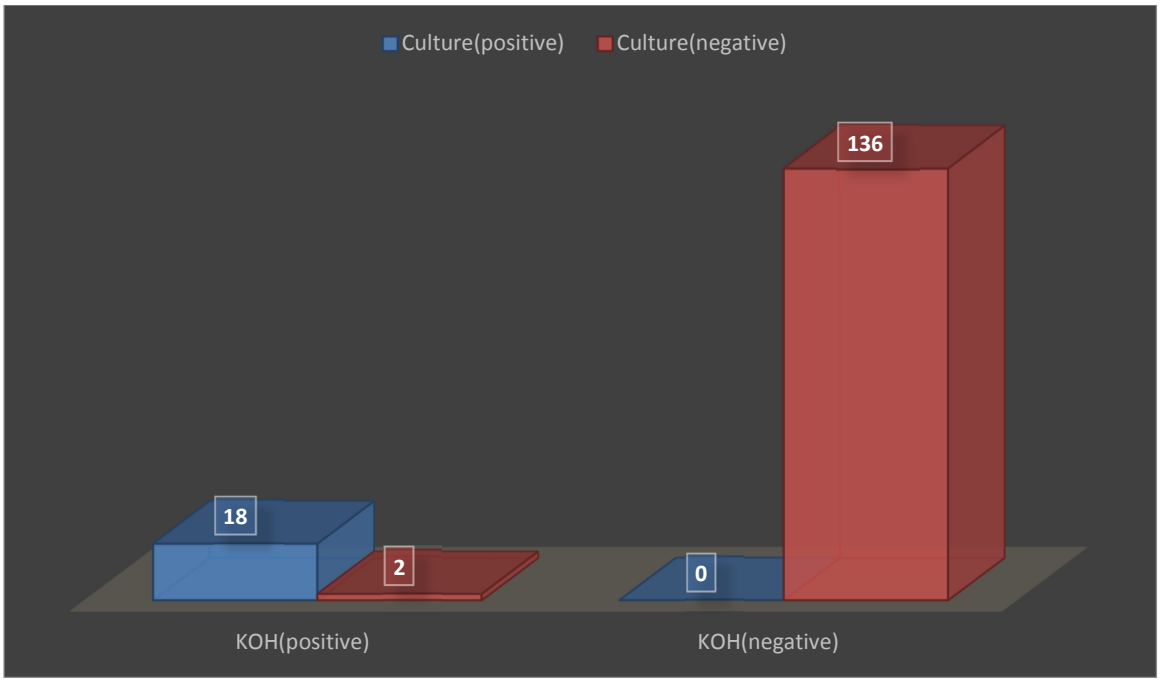
| Fungal culture | No. | % |
|---|---|---|
| **Culture(+)** | 18 | 11.5 |
| **Culture(-)** | 138 | 88.5 |



## Comparison of KOH Mount &Culture

*(Table 10 & Graph 10)*

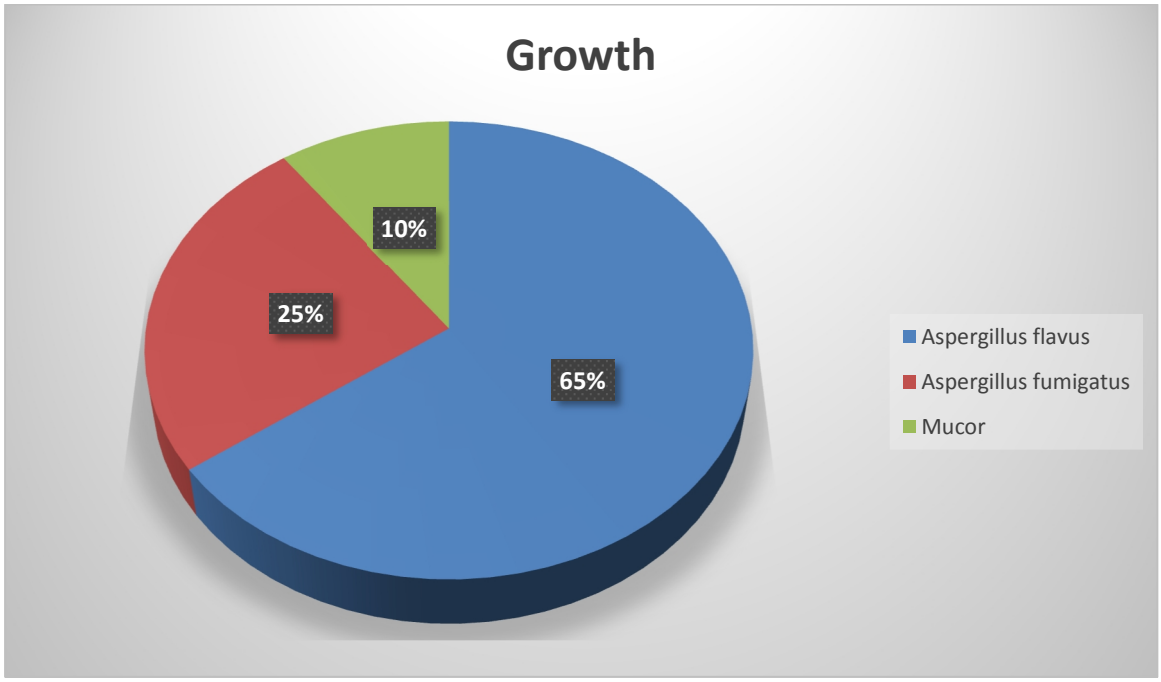
| | Culture(positive) | Culture(negative) | Total |
|---|---|---|---|
| **KOH(positive)** | 18 | 2 | 20 |
| **KOH(negative)** | 0 | 136 | 136 |
| **Total** | 18 | 138 | 156 |



**Distribution of fungal isolates**

***(Table 11 & Graph 11)***

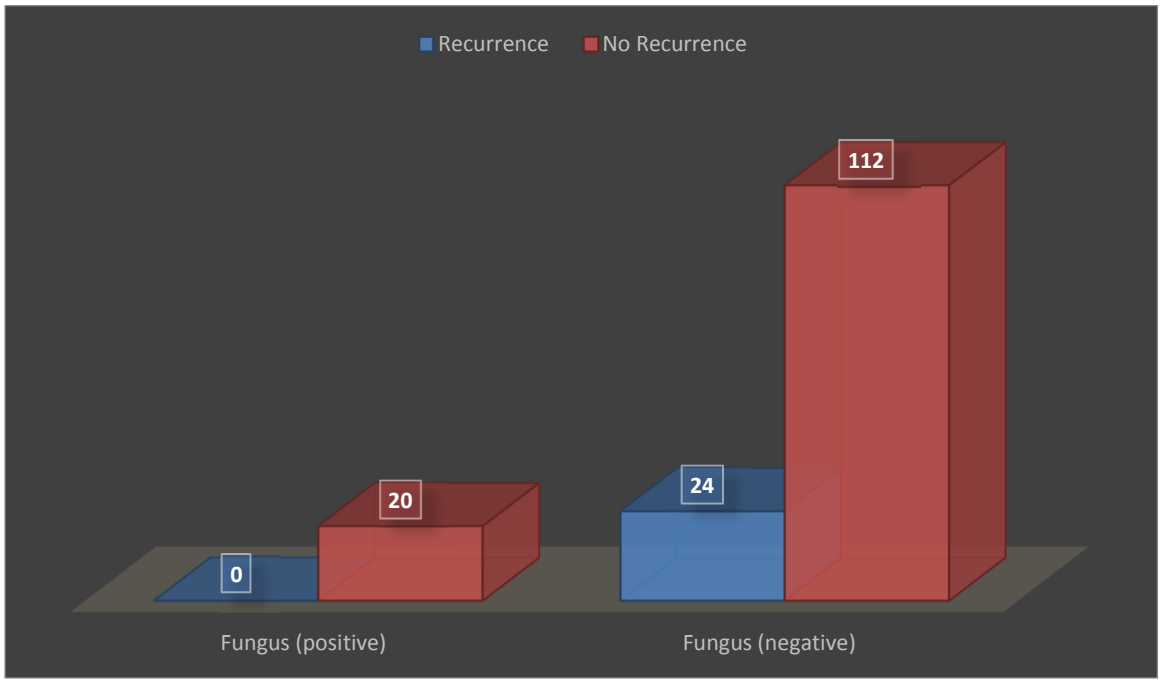
| Growth | No. | % |
|---|---|---|
| **Aspergillus flavus** | 13 | 65 |
| **Aspergillus fumigatus** | 5 | 25 |
| **Mucor** | 2 | 10 |



## Estimation of recurrence

*(Table 12& Graph 12)*

| | Recurrence | No Recurrence | Total |
|---|---|---|---|
| **Fungus (positive)** | 0 | 20 | 20 |
| **Fungus (negative)** | 24 | 112 | 136 |
| **Total** | 24 | 132 | 156 |



## DISCUSSION

Fungal rhinosinusitis, is now being reported with increasing frequency worldwide. In India, fungal diseases of the paranasal sinuses is not only prevalent in southern regions, but also reported from other parts of the country[26,53]. Their importance is increasing due to the morbidity and mortality caused by FRS

In the present study 156 patients of chronic rhinosinusitis were included. Age of the patients varied from 1 to 80 years . Majority of the patients were in the age group of 31 – 40 years. followed by 21-30 years. The mean age of the patients affected with fungal rhinosinusitis was (34.5 yrs). Males were predominant (55%) compared to females ( 45% ). Male to female ratio was (1.3:1). Montone et al. in USA found that the mean age of FRS patients was on the higher side of 45 years with a range of 18–88 years, and male to female ratio was 1.2 : 1,which is similar to this study[55]

In this study most of the patients were from rural background as it is a tertiary care hospital attending the patients from the surrounding villages .Among them 38% were coolie workers ,8% were farmers and 34 %were housewives. The reason could be that the population in rural areas are more exposed to spores, poor hygienic conditions and the hot humid climate .

As per study done by Klossek *et al*, most patients were between 30-59 year old[54]. Female predominance was seen. Majority of patients were from rural

area.

The results of this study is similar to the study done by Das et al in Chandigarh which reported ages of patients with fungal rhinosinusitis from 2 to 81 years.[58] Male preponderance in this study is concordant with the study done by Prateek et al[56](1.33:1) and Shone GR (1.8:1)[57].However study that was done by Micheal et al[59]and Dufour et al[60]showed female predominance. The results which is obtained from this study can be attributed to the fact that males are more commonly exposed to pollutants in traffic, dust and factories.

In the present study the most common symptoms were nasal obstruction 85**%**, headache 56% , nasal discharge 24% , and sneezing 21%**.** In a study done by Shivani et al 216cases, the most common presenting complaints were nasal obstruction 90.74% followed by posterior nasal discharge 74.07%, anterior nasal discharge 58.33%, headache 45.37% and aural symptoms 17.59%.[61]

In this study , 53 patients had nasal polyp 60 had deviated nasal septum , 15 patients had diabetes , 9 had hypertension and 4 had both. Shivani et al found that the most common risk factor was nasal allergy 42(19.44%) followed by deviated nasal septum 24(11.11%) and nasal polyp 24(11.11%)[62,63]. Other risk factors were hypertension 12(5.55%), bronchial asthma 10(4.62%) and Diabetes 9(4.16%) In India bronchial asthma and diabetes mellitus are extremely common and in some patients these conditions remains undiagnosed which predispose them to chronic sinusitis[64].

There is an increased tendency of fungal infections to occur in a more invasive form if there is underlying hyperglycemia due to uncontrolled diabetes. Varying data are available regarding the existence of hyperglycemia as a risk factor for development of the disease. Michael *et al.,*(2008) reported uncontrolled diabetes in 38.8% of cases of invasive fungal sinusitis and they have suggested that the study population may have undiagnosed Diabetes mellitus since Diabetes is known to be extremely common in India [61,65].

Mohapatra *et al.,*(2010), has observed that hyperglycemia was noted in 44.8 % of cases. Diabetes mellitus is an important cause of impaired cell mediated immunity which is the primary host defense against fungal infections. Diabetes mellitus patients with uncontrolled blood sugar levels tended to have more aggressive fungal infection and required prolonged hospital stay for control of infection[45,67].Diabetes atlas 2006 by International Diabetes Federation has observed that the current number of people affected by Diabetes is 40.9 Million and is expected to rise to 69.9 Million by 2025 unless preventive measures are taken. So, increased vigilance will be needed to identify fungal rhinosinusitis in the future[68].

In most patients (69) multiple sinuses were involved as revealed by CT scan of the paranasal sinuses. In a study by Alrajhi *et al.,*abnormalities of paranasal sinuses were noted on CT scan in all patients; all sinuses were involved in 61% of patients[69]

In this present study,out of 156 cases of chronic rhinosinusitis, prevalence of fungal rhinosinusitis was found to be 13 %. In this study fungal positivity was found in 20 patients by direct examination (KOH mount) or culture . Out of these 18 were positive by culture and 20 were positive by KOH mounting. 2were KOH positive and culture negative. This could be due to inadequate specimen or contamination of the sample before subjecting it for culture (70).

Ragini *et al*[30,71] proved, 10 out of 47 patients (21.2%) were culture positive and 37 out of 47(78.8%) were culture negative. Klossek *et al* showed ,33 out of 109 patients (30%) were culture positive while 76 out of 109 samples (69.7%) were culture negative. Das*etal*, showed that, 137out of 222 samples(61.7%) were culture positive while 85 out of 222 samples (38.28%) were culture negative. Prateek*etal* proved 21 out of 100 cases (21%) were culture positive while 79 out of 100 cases (79%) were culture negative.

There is emerging evidence that fungi play an important role in exacerbation and perpetuation of mucosal inflammation in CRS, and only in more recent times has the categorization of FRS been more fully defined.

In this study majority of the fungi isolated were *Aspergillus sp*ecies(90%) in particular *A.flavus*. Out of the 18 Aspergillus isolates 13 were Aspergillus flavus and 5 were Aspergillus fumigatus. This is attributed to the ubiquitous nature of their fungal spores in the soil and environment of tropical countries like India. This was parallel to the results got by Shiv sekar chatterjee and Kavitha et al in

their studies[71,72].In a similar study by Saravanan et al., in Chandigarh, among the 32 patients in the AFRS group, the most common culture isolate was *A. flavus* (81.3%), followed by *A.fumigatus* (8.9%), with *Bipolaris* spp. being (6.5%)[73]. In this present study, dematiaceous fungi was not isolated among our isolates. This might be due to different geographical distribution of the fungi in different areas depending on local climate temperature and humidity.

The findings of this study is very similar to the study done by Michael et al in Tamil Nadu where Aspergillus flavus (47.61%) was the most common isolate followed by Aspergillus fumigatus (14.28%)[74].

In North America,dematiaceous fungi, such as Bipolaris spp and Curvularia spp were found to predominate in allergic sinusitis[35,74]. The reasons for this difference are a matter of speculation, but several factors could be involved. In India, a huge proportion of the population live in rural or semi-rural areas and so their exposure to certain fungi will differ from a more urban population in developed countries. Another contributory factor may be due to the type of housing in the two countries. In India, houses are often open to the environment and this may lead to prolonged exposure to fungi that occur in the outside environment. In this study All the cases which are positive for fungus were subjected to medical treatment either with oral itraconazole or Systemic Amphotericin B and they were closely followed up for any chance of recurrence of disease. But the patients did not show any signs of recurrence for the two

year period

Despite the recognition of fugal rhinosinusitis as a serious disease for a long time, our knowledge regarding its epidemiology and medical microbiology is subjected to newer findings and research

# CONCLUSION

CRS is a disease which affects the quality of life significantly. Since the disease has a highly heterogenous nature, sometimes identification of an underlying cause is very difficult.

The sinonasal mycotic infection accounts for 13% of chronic rhinosinusitis. The most commonly affected age group are the third and fourth decade. The sex ratio is more or less equal. Fungal sinusitis should be suspected in those patients with CRS presenting with signs and symptoms such as nasal obstruction, nasal polyps, nasal discharge. With the help of histopathological examination of all sinus specimens, CT scan, Diagnostic Nasal Endoscopy the diagnosis of CRS have become easier nowadays.

Endoscopic sinus surgery followed by antifungal therapy role is the major treatment of fungal sinusitis. Due to the increased incidence in fungal infections of paranasal sinuses, the Otorhinolaryngolgists should keep fungal infections in their mind during their daily practice.

In fungal sinusitis, unilateral involvement of paranasal sinuses is more common. As the ostium of maxillary sinus is placed at a lower level in the middle meatus when compared with the other sinus ostia, it offers an easy access to the microorganisms, Maxillary sinus is the most commonly affected sinus among all the paranasal sinuses in fungal rhinosinusitis .

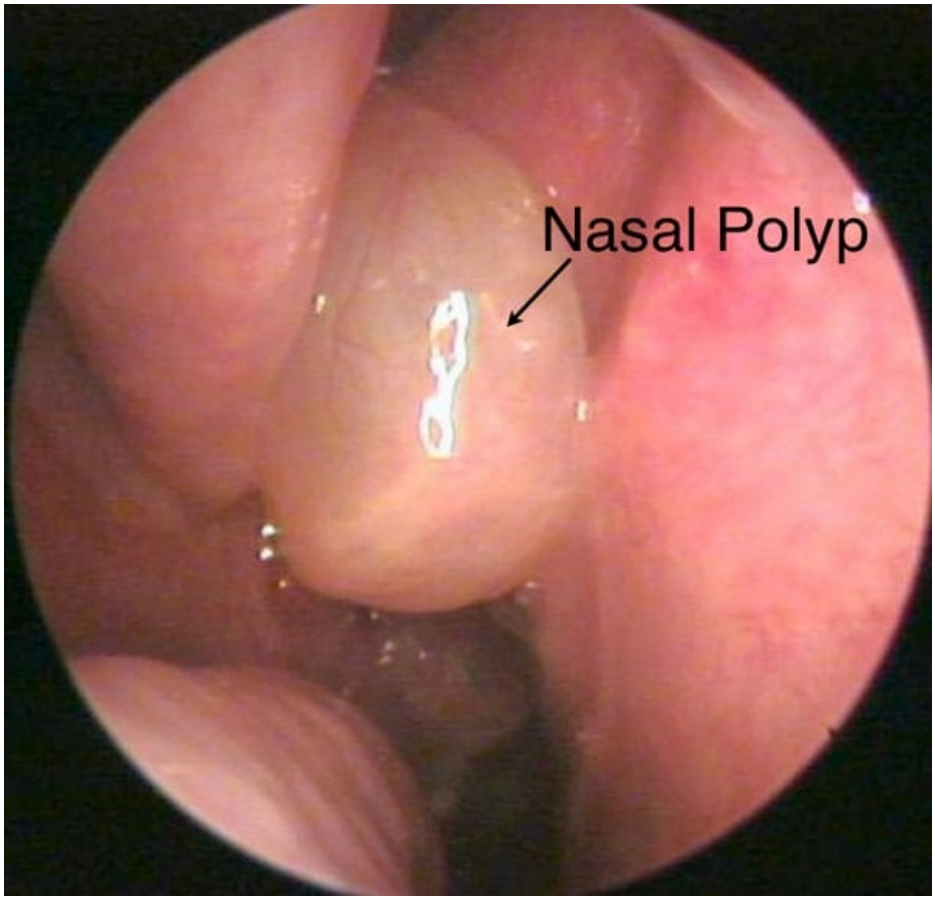



Fig 17:Intra-operative picture of Right Nasal polyp

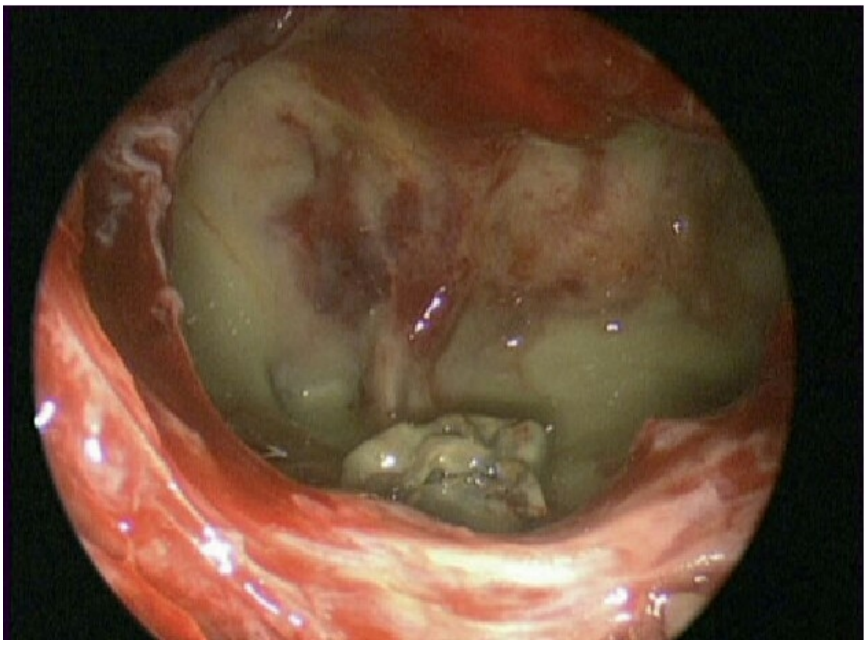


Fig 18:Intra-operative picture of sphenoid sinus showing purulent discharge with cheesy material

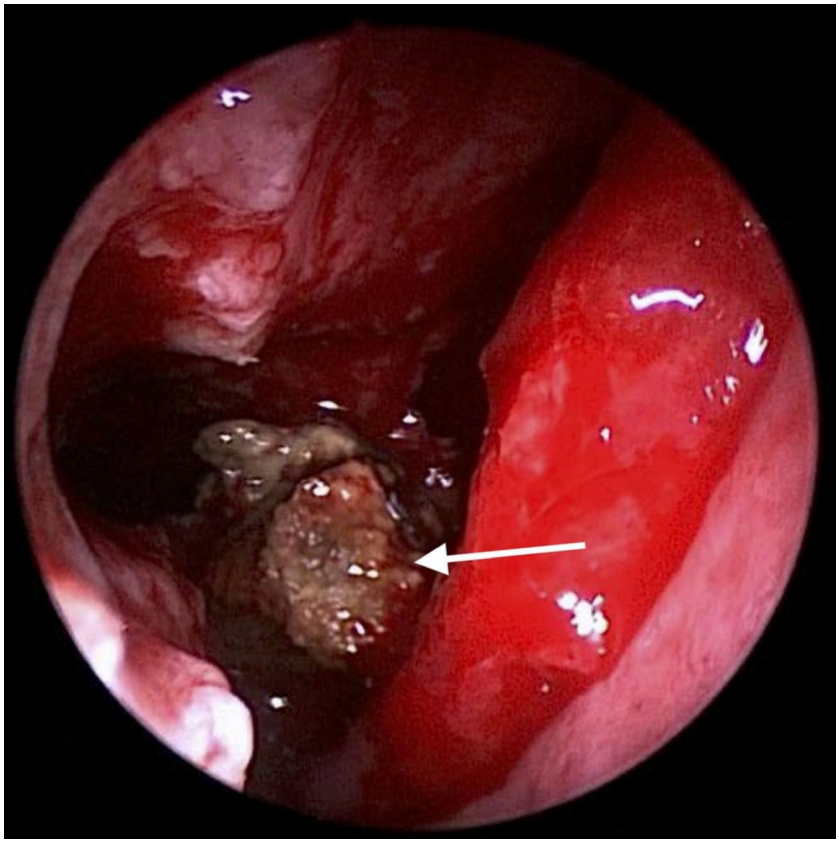


Fig 19:Intra-operative picture of Right maxillary fungal ball

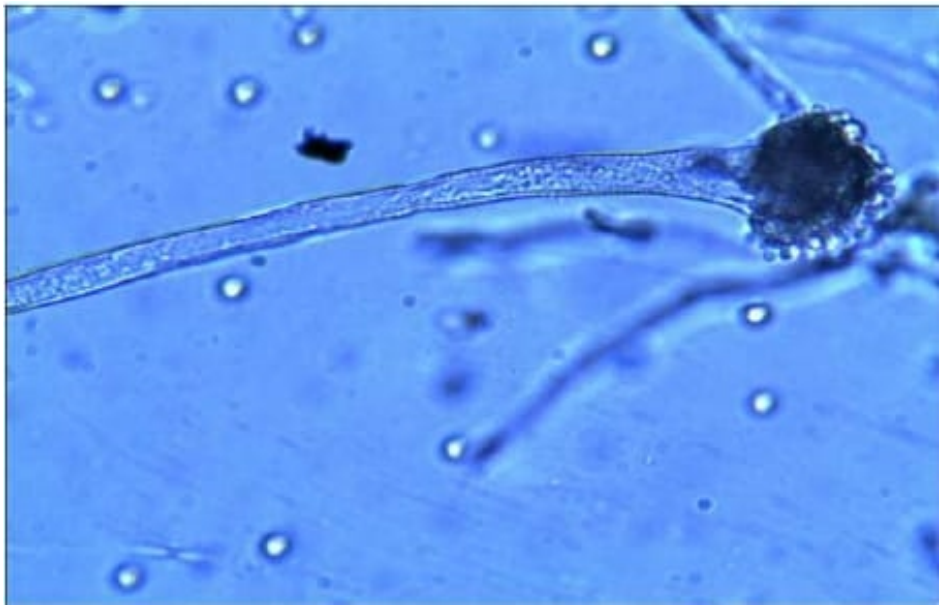


Fig 20: KOH mount of Aspergillus Fumigatus

Fig 21:culture showing Aspergillus fumigatus

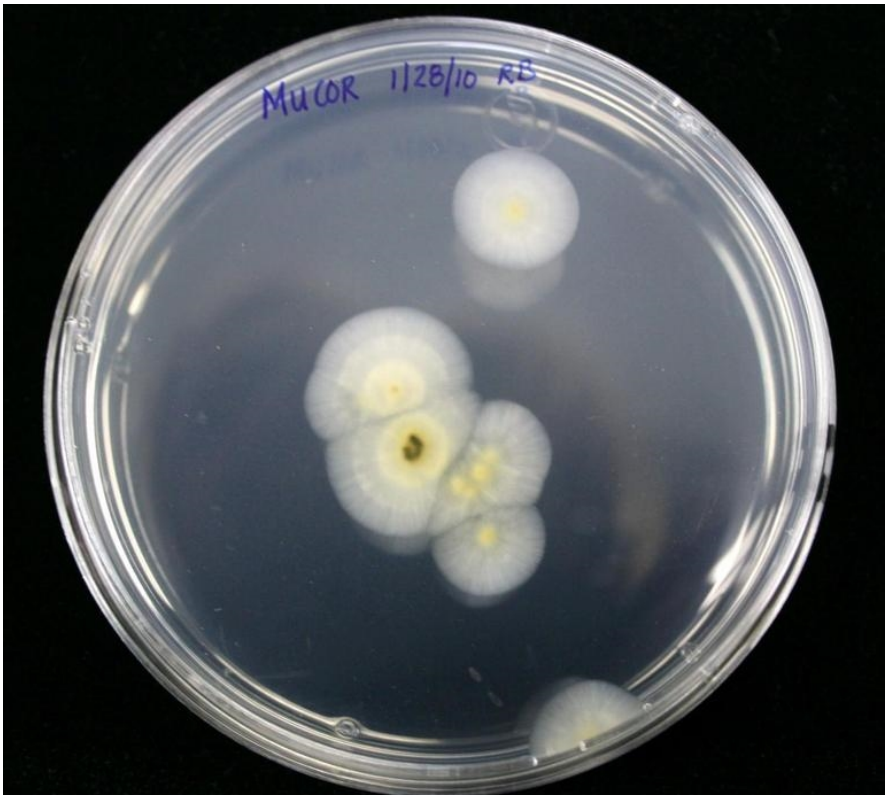



Fig 22:Culture showing Mucor(Rhizopus spp.)

## SUMMARY

- The study was conducted over a period of two months from November 2017 to July 2019.
- Patients who are not clinically diagnosed as chronic rhinosinusitis but undergoing FESS were not included in the study.
- The present study included 156 clinically diagnosed cases of chronic rhinosinusitis undergoing FESS.
- Commonest age group affected were between 31 to 40 years.
- Males were slightly more affected than females.
- Most of the patients were coolie workers, housewives & farmers.
- Nasal obstruction, headache & sneezing were the most common symptoms the patients presented with while the least common symptoms were cough and ophthalmic symptoms.
- About 1/4 of the patients had at least one of the two systemic diseases, Diabetes mellitus & hypertension.
- About 85% of the patients had nasal obstruction & polyp.
- All of the sinuses were involved in 35% of the cases with fungal culture positive.
- *A.flavus* is the commonest isolate followed by *A.fumigates*. & *Mucor*.

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நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

| | | பங்கு பெறுவர் இதனை ✓ குறிக்கவும் |
|---|---|---|
| 1. | நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன். | ☐ |
| 2. | நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். | ☐ |
| 3. | இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். | ☐ |
| 4. | இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன். | ☐ |
| 5. | இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன். | ☐ |

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ..... .....

| S.No | NaMe | Age | Sex | IP No | SyMptoMs | | | | | | | SysteMic Illness | | CT FINDINGS | | | | | | | | | | | | | | Fungal Isolates | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | | | DNS | | SINUS INVOLVEMENT | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | Isolated Maxillary | | Maxillary & Ethmoid | | Ethmoid | | Sphenoid | | Frontal | | All Sinus involvement | | | | |
| | | | | | Nasal Obstruction | Nasal discharge | Head Ache | Sneezing | HyposMia | Cough | Eye Symptoms | DM | HTN | R | L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | KOH Mount | Fungal culture | Fungal Species |
| 1 | PackialakshMi | 47 | F | 93984 | + | - | + | + | - | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 2 | ValarMathi | 26 | F | 94669 | + | - | + | - | - | - | - | | | | + | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 3 | TaMilselvi | 33 | F | 84118 | + | - | + | - | - | - | - | | | | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 4 | Sivaguru | 43 | M | 692 | + | + | + | - | - | + | - | | | | + | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 5 | Mathan | 29 | M | 2109 | + | + | - | + | - | - | - | | | | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 6 | Sruthi | 19 | F | 1391 | - | - | + | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 7 | Vellapandy | 65 | M | 4407 | + | - | + | + | + | - | - | DM | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | + | A.Flavus |
| 8 | Siva Nayagi | 40 | F | 5713 | - | - | + | - | - | - | - | | | | + | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 9 | MariaMMal | 40 | F | 5773 | + | + | - | - | - | - | - | | | + | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 10 | Inbaraj | 32 | M | 6274 | + | - | + | - | - | - | - | DM | | - | + | - | - | - | - | - | - | - | - | - | - | - | - | | | |
| 11 | Shanthi | 28 | F | 9125 | + | + | - | + | - | + | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 12 | MohaMMed Siddiq | 21 | M | 8352 | + | + | + | - | - | + | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 13 | SubraManian | 70 | M | 9824 | + | + | + | - | + | - | - | DM | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 14 | MadasaMy | 31 | M | 13783 | + | - | + | - | - | - | - | | | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 15 | Madakannu | 43 | M | 15476 | + | - | - | - | - | + | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 16 | Rubi | 40 | F | 18575 | + | - | - | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | + | A.Flavus |
| 17 | Mugesh | 13 | M | 21235 | + | + | + | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 18 | SubraManiuM | 27 | M | 21268 | + | + | + | - | - | + | - | DM | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 19 | Abdul Kathar | 31 | M | 23450 | + | - | + | - | - | + | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 20 | SubraManiuM | 66 | M | 23097 | + | - | + | - | - | - | - | | | + | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 21 | Shalini | 17 | F | 26276 | + | + | - | - | - | + | - | | | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 22 | Chandru | 15 | M | 28044 | + | - | + | - | - | - | | | | + | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 23 | SonMuthu | 32 | M | 30082 | + | - | - | - | - | - | | | | + | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 24 | Edison | 30 | M | 38061 | + | + | - | - | - | + | | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | - | A.Flavus |
| 25 | SankaraMMal | 65 | F | 29852 | + | - | + | - | - | - | | | | - | - | + | - | - | - | - | - | - | - | - | - | - | | | | |
| 26 | PonnusaMy | 65 | M | 30792 | + | - | - | + | + | - | - | | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 27 | ParaMeshwari | 28 | F | 31549 | + | - | + | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 28 | Raju Divya | 16 | F | 31570 | + | - | - | - | + | - | - | | | - | + | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 29 | SubbaMMal | 58 | F | 31972 | - | - | + | - | - | - | - | | HTN | - | + | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 30 | Balakandan | 22 | M | 31812 | + | - | - | + | - | + | - | | | - | + | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 31 | Divya | 24 | F | 51523 | + | - | - | - | + | - | - | | | - | + | - | - | - | - | - | - | - | - | - | - | - | - | | | |
| 32 | Ajay | 16 | M | 32446 | + | - | - | + | - | - | - | | | - | - | - | + | - | - | - | - | - | - | - | - | - | + | | | |
| 33 | Mariappan | 55 | M | 30545 | + | + | + | - | - | + | - | DM | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | - | A.Fumigatus |
| 34 | SubbaMMal | 45 | F | 32118 | + | - | - | + | - | + | - | DM | | - | + | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 35 | Annadurai | 35 | M | 33294 | + | - | - | - | + | - | - | | | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 36 | Maharajan | 45 | M | 32912 | + | - | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 37 | ShannMugavel | 49 | M | 34483 | + | - | + | - | - | - | + | DM | | - | - | - | - | + | - | - | - | - | - | - | - | - | - | + | + | Mucor |
| 38 | Syed FathiMa | 64 | F | 32386 | - | - | + | - | - | - | - | DM | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 39 | Lusiya | 18 | F | 35506 | - | - | + | - | - | + | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |

| S.No | NaMe | Age | Sex | IP No | SyMptoMs | | | | | | | SysteMic Illness | | CT FINDINGS | | | | | | | | | | | | | | Fungal Isolates | | |
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| | | | | | Nasal Obstruction | Nasal discharge | Head Ache | Sneezing | HyposMia | Cough | Eye Symptoms | DM | HTN | DNS | | SINUS INVOLVEMENT | | | | | | | | | | | | KOH Mount | Fungal culture | Fungal Species |
| | | | | | | | | | | | | | | R | L | Isolated Maxillary | | Maxillary & Ethmoid | | Ethmoid | | Sphenoid | | Frontal | | All Sinus involvement | | | | |
| | | | | | | | | | | | | | | | | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | | | |
| 40 | RaMalakshMi | 29 | F | 37372 | + | - | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 41 | Gilbert | 33 | M | 37398 | + | + | - | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 42 | Eswaran | 35 | M | 37380 | + | - | - | + | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 43 | Muthuselvi | 34 | F | 37787 | - | + | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 44 | Sundarajan | 18 | M | 38470 | + | - | + | - | - | + | - | | | + | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 45 | MariaMMal | 34 | F | 39315 | - | + | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 46 | IMtyaz | 45 | M | 39487 | + | - | + | - | - | - | - | | | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 47 | PalaniaMMal | 55 | F | 42782 | - | + | + | - | - | - | - | | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 48 | Mahendra SelvaM | 32 | M | 44635 | + | - | - | - | - | - | - | | | + | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 49 | Madan Raj | 14 | M | 45728 | + | - | - | - | - | - | - | | | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 50 | EsakiaMMal | 37 | F | 49712 | + | - | + | - | - | - | - | | | - | + | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 51 | Ayisha Banu | 45 | F | 46452 | + | - | - | - | + | - | - | | HTN | + | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 52 | Jebaraj | 55 | M | 46549 | + | - | - | + | + | - | - | - | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 53 | Ashok KuMar | 22 | M | 46518 | + | - | + | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 54 | MohaMMed | 17 | M | 46490 | + | - | + | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | + | | | |
| 55 | Muppidathy | 48 | M | 48237 | + | - | - | + | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 56 | Nagoorkani | 23 | M | 48684 | + | - | + | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 57 | Soosai RathnaM | 65 | F | 43457 | + | - | + | + | - | - | - | DM | HTN | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 58 | SaMithai | 35 | F | 47089 | + | + | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 59 | Sudalai Mani | 29 | M | 50581 | + | - | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | | | |
| 60 | Alagu Thirunachi | 39 | F | 51783 | - | + | + | - | - | - | - | - | - | - | + | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 61 | Muthu KuMar | 45 | M | 51825 | + | - | - | - | - | - | - | - | - | - | + | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 62 | UMa Maheswari | 11 | F | 52423 | + | - | - | - | - | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 63 | Sankara Subbu | 31 | M | 54109 | + | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | + | - | - | - | - | - | | | |
| 64 | VellaMMal | 65 | F | 52624 | + | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | | | |
| 65 | Mariya Raj | 11 | M | 55781 | + | - | - | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 66 | GoMathi | 58 | F | 58562 | + | - | - | + | - | - | - | DM | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 67 | MohaMMed Yunis | 12 | M | 62177 | + | + | - | - | - | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 68 | VelMail | 26 | M | 62161 | + | - | + | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 69 | Manikandan | 29 | M | 62170 | + | - | + | - | - | + | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 70 | Saravana kuMar | 23 | M | 62761 | + | - | + | - | - | - | - | - | - | - | - | + | + | - | - | - | - | - | - | - | - | - | - | | | |
| 71 | Packia LakshMi | 32 | F | 62731 | + | - | + | - | - | + | - | - | - | - | + | - | - | - | - | - | - | + | - | - | - | - | - | | | |
| 72 | Arul Alex | 20 | M | 65004 | + | - | - | - | - | + | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 73 | ThangaperuMal | 44 | M | 65763 | + | - | - | - | + | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 74 | MuthuMari | 34 | F | 65696 | + | - | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 75 | Paulkani | 36 | F | 66794 | + | + | + | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 76 | SelvakuMar | 26 | M | 67885 | + | + | + | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 77 | Ayyapan | 19 | M | 67537 | + | + | + | - | - | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 78 | Paulkani | 49 | F | 69290 | + | - | - | - | + | - | - | - | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 79 | Kalister | 37 | M | 70317 | + | - | + | + | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |

| S.No | NaMe | Age | Sex | IP No | SyMptoMs | | | | | | | SysteMic Illness | | CT FINDINGS | | | | | | | | | | | | | | Fungal Isolates | | |
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| | | | | | Nasal Obstruction | Nasal discharge | Head Ache | Sneezing | HyposMia | Cough | Eye Symptoms | DM | HTN | DNS | | SINUS INVOLVEMENT | | | | | | | | | | | | KOH Mount | Fungal culture | Fungal Species |
| | | | | | | | | | | | | | | | | Isolated Maxillary | | Maxillary & Ethmoid | | Ethmoid | | Sphenoid | | Frontal | | All Sinus involvement | | | | |
| | | | | | | | | | | | | | | R | L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | | | |
| 80 | MariMuthu | 12 | M | 69879 | + | - | - | + | - | - | - | | | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 81 | SubbulakshMi | 34 | F | 70325 | + | - | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 82 | Mariyaselvi | 32 | F | 71121 | + | + | + | - | - | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 83 | ThangasaMy | 33 | M | 75170 | + | - | - | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 84 | MariaMMal | 49 | F | 75204 | + | - | - | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 85 | BalaMurugan | 38 | M | 79410 | - | - | + | - | - | - | + | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 86 | Chandra | 60 | F | 81649 | + | - | - | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | + | A.Flavus |
| 87 | Aravind | 13 | M | 80915 | + | + | + | - | - | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 88 | Murugan | 22 | M | 83807 | - | - | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 89 | Esaki Pandi | 23 | M | 85946 | + | - | - | - | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 90 | Iruthaya Raj | 55 | M | 89299 | + | - | + | - | - | - | - | | | + | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 91 | MasanaM | 50 | M | 87549 | + | + | + | - | - | - | - | | | + | - | - | + | - | - | - | - | - | - | - | - | - | - | + | + | A.Fumigatus |
| 92 | Suganya | 24 | F | 90501 | + | - | + | - | - | - | - | | | + | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 93 | SivaraMa Krishnan | 23 | M | 1031 | + | + | + | - | - | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 94 | Saravanan | 35 | M | 1083 | + | - | - | + | + | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 95 | Kalaiarasi | 26 | F | 1769 | - | - | + | - | - | + | - | | | - | - | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 96 | Selvi | 50 | F | 5155 | + | - | + | - | - | - | - | | | + | - | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 97 | Alexander | 44 | M | 5092 | + | - | - | + | + | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 98 | Durai Murugan | 28 | M | 5751 | - | + | + | - | - | - | - | | | - | - | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 99 | Sakthi Murugan | 24 | M | 9803 | + | - | + | - | - | + | - | - | - | - | + | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 100 | MohaMed Syed Ali | 35 | M | 11716 | + | - | - | - | + | - | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 101 | Selvi | 45 | F | 12445 | + | - | + | - | - | - | - | DM | - | - | + | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 102 | Sanvasi Rajan | 46 | M | 12443 | + | - | - | + | + | - | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 103 | Subbiah | 62 | M | 12487 | + | - | + | - | - | - | - | DM | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 104 | Nadarajan | 33 | M | 14741 | + | - | - | - | - | - | - | - | - | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 105 | Pravin | 18 | M | 17354 | + | - | - | + | + | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 106 | Malathi | 26 | F | 18291 | + | + | + | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 107 | Vinayaga PeruMal | 39 | M | 19355 | + | - | + | - | - | - | - | | | - | + | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 108 | Nallathai | 35 | F | 30120 | + | - | - | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 109 | HaMitha Banu | 43 | F | 21877 | + | - | - | + | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 110 | Jeyalalitha | 45 | F | 21802 | + | - | + | - | - | - | - | | | - | + | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 111 | JayalakshMi | 36 | F | 54482 | + | - | + | - | - | - | - | | | - | - | - | - | - | - | - | + | - | - | - | - | - | - | | | |
| 112 | InnasiaMMal | 57 | F | 53866 | + | - | - | - | - | - | + | DM | | - | - | - | - | - | - | + | - | - | - | - | - | - | - | + | + | Mucor |
| 113 | Chelladurai | 27 | M | 9238 | + | - | + | - | - | - | - | | | + | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 114 | Manikandan | 28 | M | 8991 | + | - | - | - | + | - | - | | | + | - | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 115 | EsakiMani | 35 | M | 9828 | + | - | + | - | - | + | - | | | - | + | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 116 | Murugan | 30 | M | 9546 | + | - | - | - | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 117 | Sudalai Vadivu | 60 | F | 11468 | + | + | - | - | - | - | - | DM | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 118 | ShanMuga sundari | 37 | F | 12158 | + | + | + | - | - | - | - | | | + | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 119 | Ganesan | 51 | M | 13730 | + | - | - | + | + | - | - | DM | | - | + | - | - | - | - | - | + | - | - | - | - | - | - | + | + | A.Flavus |

| S.No | NaMe | Age | Sex | IP No | SyMptoMs | | | | | | | SysteMic Illness | | CT FINDINGS | | | | | | | | | | | | | | Fungal Isolates | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | | | DNS | | SINUS INVOLVEMENT | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | Isolated Maxillary | | Maxillary & Ethmoid | | Ethmoid | | Sphenoid | | Frontal | | All Sinus involvement | | | | |
| | | | | | Nasal Obstruction | Nasal discharge | Head Ache | Sneezing | HyposMia | Cough | Eye Symptoms | DM | HTN | R | L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | U/L | B/L | KOH Mount | Fungal culture | Fungal Species |
| 120 | Rohini | 33 | F | 15467 | + | + | + | - | - | - | - | | | - | + | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 121 | PetchiaMMal | 20 | F | 16158 | + | - | - | - | - | - | - | | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 122 | GoMathy | 36 | F | 16220 | - | - | + | - | - | - | - | - | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 123 | MurugaMMal | 30 | F | 17352 | - | - | + | - | - | + | - | | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 124 | Jothi Gowri | 22 | F | 18025 | + | - | - | - | + | - | - | | | + | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 125 | SubraManian | 52 | M | 19102 | + | - | - | + | + | - | - | | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 126 | Radhika | 27 | F | 19112 | + | - | - | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | + | A.Flavus |
| 127 | Vasuki | 18 | F | 19782 | + | - | - | + | - | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 128 | Devi | 21 | F | 19873 | + | - | + | - | - | - | - | | | - | + | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 129 | LakshMiaMMal | 47 | F | 22682 | + | + | + | - | - | - | - | | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + | + | A.Flavus |
| 130 | Murali | 28 | M | 24245 | + | - | + | - | - | - | - | | | - | + | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 131 | Jeena | 36 | M | 24235 | - | + | + | - | - | + | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 132 | Murugeswari | 36 | F | 24480 | + | - | - | + | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 133 | Murugesan | 45 | M | 25126 | + | - | - | - | - | - | - | | | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 134 | Esaki raj | 16 | M | 26723 | + | - | + | - | - | - | - | | | + | - | - | + | - | - | - | - | - | - | - | - | - | - | | | |
| 135 | Israth Banu | 19 | F | 23346 | + | - | | - | - | - | - | | | + | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 136 | Santhana Mari | 47 | F | 27848 | - | - | | - | - | + | - | | | - | - | - | - | - | - | - | + | - | - | - | - | - | - | | | |
| 137 | Elizabeth Rani | 35 | F | 28548 | + | - | - | + | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 138 | Manikandan | 42 | F | 29401 | + | + | - | - | - | - | - | | | + | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 139 | Mohan | 43 | M | 31848 | + | - | - | - | + | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 140 | kanipriya | 52 | F | 33376 | + | - | - | - | - | - | - | | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 141 | Selvaganapathy | 38 | F | 37756 | + | - | - | + | - | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 142 | SavariMuthu | 36 | M | 39757 | + | - | | - | - | - | - | | | - | + | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 143 | PothigaiMalai | 65 | M | 39719 | - | - | | - | - | - | - | DM | | - | + | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 144 | KuMar | 52 | M | 42321 | - | - | | - | - | - | - | DM | HTN | - | - | - | - | - | + | - | - | - | - | - | - | - | - | + | + | A.Fumigatus |
| 145 | Ganesan | 63 | M | 41988 | - | - | | - | - | - | - | | HTN | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 146 | MuthukuMar | 30 | M | 45533 | - | - | | - | - | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 147 | Papathi | 55 | F | 46198 | - | + | | - | - | - | - | DM | | + | - | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 148 | ThangaMani | 22 | M | 48055 | + | - | | - | - | - | - | | | + | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 149 | LakshMi | 22 | F | 49228 | - | + | | - | - | - | - | | | - | - | - | - | - | + | - | - | - | - | - | - | - | - | | | |
| 150 | Karthik | 25 | M | 44840 | + | + | - | - | - | - | - | | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | + | + | A.Flavus |
| 151 | Thalavaipani | 62 | M | 49897 | - | - | | - | - | - | - | DM | | - | - | + | - | - | - | - | - | - | - | - | - | - | - | | | |
| 152 | Selvarani | 40 | F | 51076 | + | - | | - | - | - | - | | HTN | - | + | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 153 | Mariselvi | 24 | F | 51778 | + | - | - | + | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 154 | Pillayar | 68 | M | 56504 | + | - | - | - | + | - | - | | | - | - | - | - | - | - | - | - | - | - | - | - | - | + | | | |
| 155 | PathMapriya | 13 | F | 57087 | + | - | - | - | - | - | - | | | + | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |
| 156 | Balaguna | 43 | M | 81681 | + | + | | - | - | - | - | | | - | - | - | - | + | - | - | - | - | - | - | - | - | - | | | |