DISSERTATION ON

THE IMPACT OF SINUSITIS MANAGEMENT IN ASTHMATIC PATIENTS

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CERTIFICATE

This is to certify that dissertation entitled **THE IMPACT OF SINUSITIS MANAGEMENT IN ASTHMATIC PATIENTS** is the bonafide record of work done by **DR.C.BALAJI**, for Degree of Master of Surgery (Otorhinolaryngology) to Tamilnadu Dr. M.G.R Medical University,Chennai is the result of original research work undertaken by her in the department of **ENT And Head and Neck surgery** Thanjavur Medical College, Thanjavur during his Post Graduate Course from 2015 – 2017 . This is submitted as partial fulfillment for the requirement of M.S. Degree Examinations – Branch IV (Otorhinolaryngology) to be held in April 2017.

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DECLARATION

I , **DR.C.BALAJI**,, solemnly declare that dissertation titled **THE IMPACT OF SINUSITIS MANAGEMENT IN ASTHMATIC PATIENTS** a clinical study submitted by me is a result of original work carried out by myself under the guidance of Prof. G.Gandhi, M.S.,D.L.O., Head of the Department, Otorhinolaryngology, Head and Neck surgery Thanjavur Medical College, Thanjavur

I further declare that the result of research has not been submitted previously by myself or other persons in any conferences or journals.

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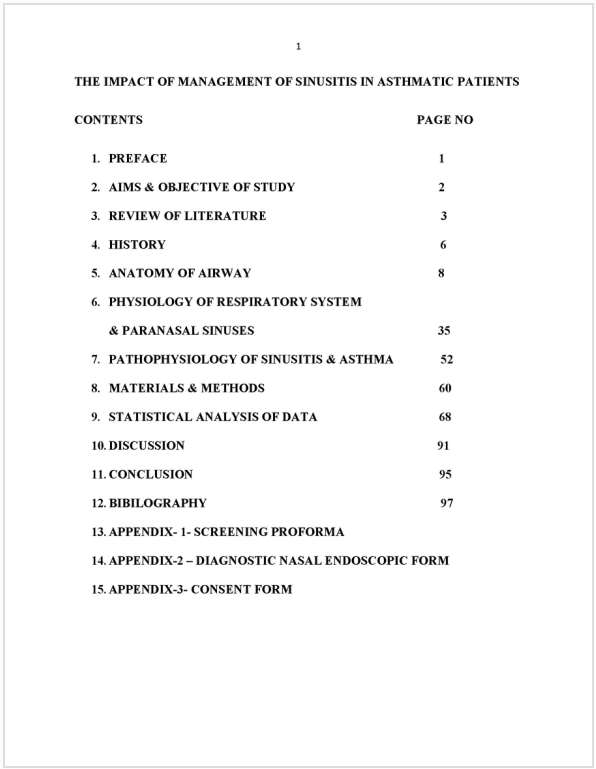

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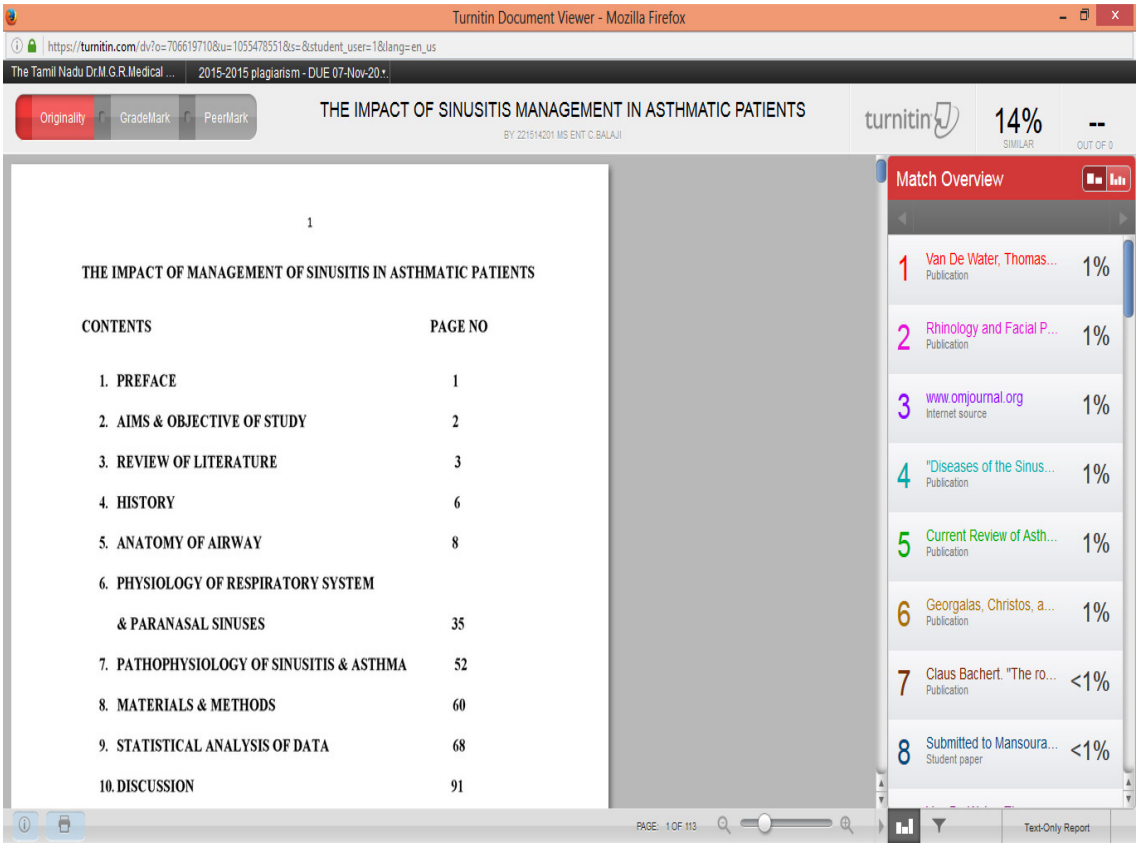
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CONTENTS

INTRODUCTION

Rhinitis,sinusitis and asthma co-exist often. The role played by sinus disease in an asthmatic patient is partially understood always because of the existing deficits in classification of the disease and etiopathogenesis of airway disease. By the upcoming clinical studies in sinus inflammation and airway remodeling a link between the upper airway and lower airway has been established as "united airway disease"[1]. This explains the relationship between sinus disease and asthma(allergic and non-allergic).

This relationship can be demonstrated in terms of epidemiological basis and improvement in signs and symptoms of an asthmatic patient by treating sinusitis either medically or by surgical methods.

This study focuses on the impact occurring in an asthmatic patient in mucociliary clearance, pathogenesis and chronicity of the disease by treating rhinosinusitis.

AIMS AND OBJECTIVE

1. To study the relationship between sinusitis and asthma.
2. To study about benefits of medical and surgical treatment of sinusitis in asthma patients.
3. To study the improvement in lung function test by treating sinusitis in asthma patients.

REVIEW OF LITERATURE

The concept of "united airways disease" putforths the relationship between upper and lower respiratory disease. The link between the two has been reviewed and are cited below.

In 1992, Dr. Raymond G.Salvin[115] and his team studied the mechansims relating the upper and lower airways as eosinophil acting as effector cell, inflammatory mediators and vagal reflex. He suggested that prospective studies on effect of medical and surgical therapy need to be done .

In a rabbit model sterile maxillary sinusitis was induced by chemotactic complement fragment C_{5a} produced a significant airway responsiveness to histamine in contrast to rabbit injected with a saline diluent which had nil inflammation. The mechanism hence putforth was postnasal dripping of cells or cell products in lower airway. This was studied by Dr.Susan.M.Brugman in the year 1993[122] .

Department of Head and neck surgery of University of Missouri-Columbia[116] in 1994 conducted a study on twenty asthmatic patients who underwent functional endoscopic sinus surgery . A concomitant reduction in usage of inhaler and steroids was noticed in patients who underwent FESS with improvement in peak expiratory flow rate too.

Senior and Kennedy in the year 1996 , of University of Pennsylvania [117] described the pathophysiologic links between sinusitis and asthma and outcome in managing the asthmatic patient with sinusitis.

In 1999,I.Annesi –Maseno of France[118], published an article on epidemiological evidence of the occurrence of rhinitis and sinusitis in asthmatics. It provided the information on the prevalence of association and factors which impact on this relationship. Though no genetic coherence between the two was found, environmental factors association in pathogenesis of both the disease was correlated by them.

University of Virginia[119] health system in the year 2002 studied the relationship between sinusitis and asthma.

The histopathological characteristics of chronic sinusitis with bronchial asthma showed marked chronic inflammatory reaction , and that eosinophil infiltration plays as a significant role in inflammatory reaction in both sinusitis and asthma as studied by Hun-Jong Dhong in 2004.

The impact of sinusitis on lower airway disease was studied by Dr.Annie.E.Dixon and Dr.David in year 2006[120] and found out that exacerbation of the lower airway disease was more in patients with sinusitis.

In 2009[121] , Vermont Lung Centre published that sinusitis is the commonest comorbidity associated with asthma. The sequence of disease and inflammatory pathways suggest that there is a common disease process for both sinusitis and asthma.

The Global Allergy and Asthma network of Excellence conducted a survey in representative sample of adults living in Europe to assess the presence of asthma and sinusits in the year 2011[123].

The united allergic airway that connects chronic rhinitis and asthma which seems to be a separate disease are actually a common atopic entity as proposed by Feng and Charles in the year 2012[124].

HISTORY

In ancient Greece,Galenus [2] was the first physician to recognize the link between the nose and lung.

There is evidence that ancient greeks studied about “asthma” under the guidance of Hippocrates in the 4th century B.C..He also believed that sinuses drained mucus from brain to nasal cavity [3] . The word asthma itself is greek in origin mentioned in the ancient text book of Iliad.

In 8th century B.C. Homer was the first to describe the term “thorax”[4].

Leonardo da Vinci in 15th century was the first artist to draw the anatomical appearance of maxillary and frontal sinuses[5].

During 16th century a Belgian physician by name Jean Baptiste Van Helmont wrote that asthma originated in the pipes of lungs. Andreas Vesalius[6] Belgian anatomist described maxillary and frontal sinuses as aerated cavities.

In 17th century Bernado Ramazzini an Italian physician found out the relation between asthma and organic dust.

In 1873, Charles Blackley, “Father of allergology” described nasal and bronchial symptoms after exposure to pollen grains.

In the year of 1919, Sluder proposed the sinopulmonary reflex.

In 1928, Kratchmer French physiologist related the link between nasal stimulation and bronchial hyper responsivness.

ANATOMY OF THE AIRWAY

EMBRYOLOGY OF NOSE AND PARANASAL SINUSES

FOURTH WEEK OF INTRAUTERINE LIFE

The face develops from facial prominences[7] around the primitive stomodeum. These facial prominences are neural crest derived mesenchyme of the first pharyngeal arch.Lateral to the stomodeum appears the maxillary prominences[8] and caudal to it the mandibular prominences[9]. The upper border of stomodeum is formed by the frontonasal prominence[10]. On either side of the frontonasal prominence surface ectoderm thicken to form the nasal placodes[11].

FIFTH WEEK OF INTRAUTERINE LIFE

The nasal placodes invaginate to form nasal pits. The nasal pits are surrounded by sheet of tissue known as nasal prominences. The outer end of the nasal prominence is lateral nasal prominence[12] whereas the inner end is known as medial nasal prominence[13].

SIXTH WEEK OF INTRAUTERINE LIFE

The maxillary prominences enlarge in size as a result of which medial nasal prominences compress in the midline.The medial nasal process fuse in midline to form intermaxillary process[18]. The intermaxillary process forms the bridge of nose

First ethmoturbinal ridge regress,ascending portion forms agger nasi[22], descending portion forms uncinate process [23]. The ascending fold is known as ramus ascendens and the descending fold ramus descendens by Stammberger[46].

Second ethmoturbinal ridge gives rises to middle turbinate[24].

The superior turbinate [25] originates from the third ethmoturbinal ridge.

The fouth and fifth ridge fuse to form supreme turbinate[26].

While all the above mentioned ridges are ethmoidal in origin, the maxilloturbinal inferior to the ethmoturbinals gives rise to inferior turbinate[27]. The nasal meati arise from the furrows between the ethmoturbinal ridges. The first furrow or primary furrow's ascending superior part forms frontal recess[28] and its descending anterior part forms the ethmoidal infundibulum,hiatus semilunaris and middle meatus[29]. The second furrow forms superior meatus[30] while the supreme meatus arises from third furrow[31]. The ethmoturbinals cross the ethmoid complex to attach to lamina papraceyae and skull base.

DEVELOPMENT OF PARANASAL SINUSES

Maxillary sinus develops as a bud from the inferior aspect of ethmoid infundibulum[32].

Frontal sinus[33] development is varied. It may arise from (1) Frontal recess,

(2) anterior ethmoid cell (3) anterosupero aspect of ethmoid infundibulum.

Sphenoid sinus develops around third or fourth month of fetal life from the posterior portion of cartilaginous nasal capsule invagination known as cartilaginous cupolar recess[35]. In later months the cartilage ossify and referred as ossiculum of Bertini[36].

The ossiculum of Bertini gets resorbed and attaches to sphenoid bone in second or third year of life.

The secondary lateral nasal wall evagination gives rise to ethmoid bulla, supra bullar and retrobullar recess[34] above and behind to the bulla to constitue the ethmoid sinus or ethmoid complex.

Paranasal sinus	Embryological appearance	Postnatal	Growth spurt
Ethmoid	Begins at third month	Present at birth	First spurt between 1 to 4 yrs and second between 4 to 8 yrs
Maxillary	65th day of gesta-tion	Present at birth	First between birth and 3yrs second between 7 and 12 yrs
Frontal	Begins at fourth month	Detected at 7 to 12 yrs	Adult size by 20yrs
Sphenoid	Begins at third month	Detected at 3 to 4yrs	At 7 yrs starts extending posteriorly towards sella tursica

ANATOMY OF LATERAL NASAL WALL

The lateral nasal wall is characterized by ridges and mounds to simplify under-standing they are better referred to as lamella based on embryological precusors[37].

The first lamella is uncinate process. The second lamella is ethmoid bulla and the third one is basal lamella. The fourth one is superior turbinate. The basal lamella or middle turbinate is significant in pattern of drainage of sinuses dividing them into anterior ethmoid and posterior ethmoid. The sphenoethmoidal recess is located just above, posterior and medial to superior turbinate[38].

The paranasal sinuses can be divided into anterior group of sinuses and posterior group[39]. The frontal sinus, maxillary sinus and anterior ethmoid form anterior sinus group while posterior ethmoids and sphenoid sinus constitute the posterior group.

The anterior group opens into middle meatus while the posterior ethmoid drain into superior meatus and sphenoid into spheno ethmoid recess[40].

The ethmoid air cells are divided into intramural and extramural cells [48] . The intramural cells are further divided into small numerous anterior ethmoid cells and large few posterior ethmoid cells[49]. Based on location of ostia of the cells various classification been proposed as given below and the most accepted nomenclature is Ritter's classification[50].

RITTER'S CLASSIFICATION

ANTERIOR ETHMOID CELLS	**POSTERIOR ETHMOID CELL**
Frontal recess Infundibular Bullar Conchal Extramural cell – agger nasi	Intramural Extramural

As mentioned above dividing the sinuses into anterior group and posterior group according to drainage pattern the following discussion is done.

ANTERIOR GROUP OF SINUSES

The anterior group of sinuses drain into the osteomeatal complex located in the middle meatus.

THE OSTEOMEATAL UNIT OF NAUMANN

The osteomeatal unit refers to various structure draining into middle meatus[41]. The components of osteomeatal unit are

Air cells of anterior ethmoid and their ostia

Frontal sinus ostia

Maxillary sinus ostia

Uncinate process

Ethmoidal infundibulum

The advent of endoscope had made the viewing of osteomeatal complex easier. To start with osteomeatal complex view we have to surpass the agger nasi cell which is the first anterior ethmoidal cell .

AGGER NASI

It has its origin from superior aspect of infundibulum or frontal recess[42]. It is

Anteriorly bounded by frontal process of maxilla

Superiorly by frontal recess

Anterolaterally by nasal bones

Inferomedially by uncinate process

Inferolaterally by lacrimal bone[43]

Agger nasi plays a significant role in frontal sinusitis pathophysiology as it forms the floor of the frontal sinus.

CONSTIUENTS OF OSTEOMEATAL UNIT

UNCINATE PROCESS

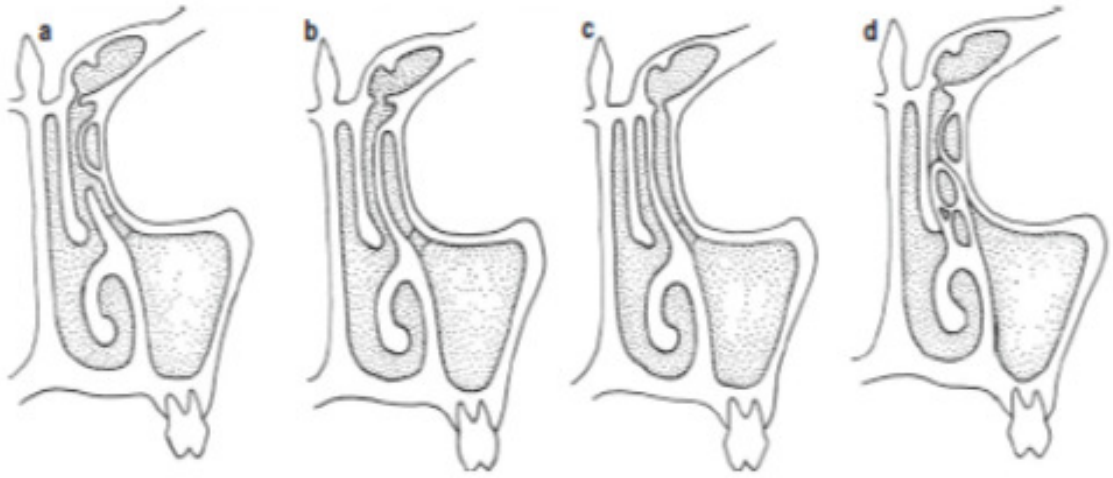
The uncinate is hook shaped and sagitally oriented. Its attachments[44] are

Anterosuperiorly to ethmoidal crest of maxilla

Posteroinferiorly to ethmoidal process of inferior turbinate

Posterosuperiorly to lamina perpendicularis of palatine bone

Superiorly to lamina paprayacea,middle turbinate or cribriform plate. The uncinate may attach to any of the above stuctures superiorly as observed by Stammberger[45]. Depending on its attachment frontal recess drainage differs as suggested by Van Alyea [61].When it attaches to lamina papracea, ethmoidal infundibulum ends blindly in terminal recess[46] and frontal recess opens directly into middle meatus. If it attaches to skull base or middle turbinate the frontal recess opens into ethmoidal infundibulum. Extensive pneumatisation of the uncinate can result in obstruction to the drainage of infundibulum[47]. The posterior free rim forms the anteromedial boundary of ethmoidal infundibulum.



ETHMOID BULLA

It was named by Zuckerkandl[53]it is attached laterally to the lamina papyrcea.

Relationships of bulla[54]

Anteroinferiorly it is separated from the infundibulum by uncinate process. Superiorly from the fovea ethmoidalis by supra bullar recess and posteriorly separating it from the basal lamella by retrobullar recess. Medially it is separated from the middle turbinate by middle meatus and the lumen is known as conchal sinus. The hiatus semilunaris inferior lies between bulla and free edge of uncinate to lead into infundibulum.

ETHMOID INFUNDIBULUM

The term infundibulum was first coined by Baron Alexis de Boyer[51]. It is a three dimensional funnel shaped passage[52] in which the secretions of anterior ethmoids, maxillary sinus and sometimes frontal sinus to drain into middle meatus occur.

Its boundaries are

Medially- uncinate process

Laterally- lamina papyracea

Anterosuperiorly-frontal process of maxilla,frontal recess and frontal sinus ostia

Superolaterally- lacrimal bone

Posteriorly-anterior border of bulla

Posteroinferiorly- maxillary sinus ostia

HIATUS SEMILUNARIS SUPERIOR OF GRUNWALD

It is a two dimensional passage between posterior wall of bulla and basal lamella of middle turbinate to establish a communication between the middle meatus and lateral sinus.

LATREAL SINUS OR SINUS LATERALIS

It constitutes the supra bullar recess and retro bullar recess. The suprabullar recess lies between ethmoid bulla inferiorly and ethmoid fovea superiorly[55]. The retrobullar recess is located between the ethmoid bulla anteriorly and basal lamella posteriorly[56].

MAXILLARY SINUS

It is largest of all the sinus and pyramid in shape. Its roof is formed by orbital floor, floor by the alveolar process of maxilla and hard palate. Its bound medially by uncinate, fontanelles and inferior turbinate, laterally by zygomatic process. Posteriorly it is separated from the infratemporal fossa and pterygopalatine fossa by a thin plate of bone and anteriorly by facial surface of maxilla[57].

The ostia is located in the posteroinferior end of infundibulum. The fontanelles are devoid of bone and named as anterior and posterior in reference to its position with uncinate. The fontanelles are usually made up of nasal mucosa and maxillary sinus mucosa[58]. The natural ostia is present in the posterior fontanelle usually.

FRONTAL SINUS AND RECESS

The frontal sinus drains into middle meatus via frontal recess. The frontal recess is an hour glass shaped lumen[59]. The frontal recess[60] is bounded

Laterally- lamina papyracea

Medially- middle turbinate

Anteriorly-posterosuperior wall of agger nasi

Posteriorly- anterior wall of bulla

Depending on the attachment of uncinate the drainage of sinus differs as mentioned earlier .

Van Alyea [62] classified accessory cells in frontal region as

1.Frontal cells- those cells which occupy frontal recess area

2.Invading cells-Supraorbital cells and intersinus septal cells which manage to invade the frontal sinus.

Bent's [63] classification of accessory frontal cells based on location

1.Type I- single frontal cell above agger nasi cell

2. Type II- tier of two or more cells above agger nasi

3. Type III – a single frontal cell which is massive and pneumatize superiorly into frontal sinus

4.TypeIV- the cell is entirely within frontal sinus

The anatomy of middle and inferior turbinate has to be discussed here as their position and attachments influence the drainage of anterior group of sinus.

MIDDLE TURBINATE

The middle turbinate is boomerang shaped charecterised by conchal head, conchal neck and conchal sinus[64] . Attachments of the middle turbinate are

Anteriorly- crista ethmoidalis of maxillary bone and agger nasi[65]

Posteriorly- ethmoidal crest of perpendicular plate of palatine bone.

The middle turbinate can be divided into three parts

PART	**LOCATION**	**PLANE**	**ATTACHMENT**
First	Anterior	Sagittal	Skull base
Second	Middle or basal	Frontal	Lamina papyracea
Third	Posterior	Horizontal	Perpendicular plate of palatine bone

The basal lamella is of utmost importance as it divides the sinus into anterior ethmoid and posterior ethmoid. The boundaries[66] are

Superiorly- fovea ethmoidalis

Laterally –lamina papyracea

Inferiorly – the attachment of third part of middle turbinate to basal lamella

Medially- – the attachment of first part of middle turbinate to basal lamella.

Medially- superior meatus and turbinate

Superiorly – fovea ethmoidalis separating it from anterior cranial fossa

Inferiorly – posterior part of middle turbinate

The posterior ethmoids [70] are important because of their close relationship with orbit and sphenoid sinus. The medial rectus is near to posterior ethmoids than its anterior counterpart. The posterior most ethmoid cell in relation with optic nerve and internal carotid artery is known as "Onodi cell" or sphenoethmoid cell . the posterior ethmoidal artery runs in the roof of sinus and more prone for injury.

SPHENOID SINUS

The sphenoid sinus is located in the centre of the skull. Its relationships[71] are as follows

Superiorly- anterior cranial fossa

Inferiorly – nasopharynx

Anterior – sphenoethmoid recess, superior turbinate and posterior ethmoids

Posterior- basi sphenoid, sella and posterior cranial fossa

Medially – sphenoid septum

Laterally – middle cranial fossa and cavernous sinus

Based on pneumatisation sphenoid sinus classified into conchal, presellar and sellar[72]. The sphenoid sinus ostium located in inferomedial edge of superior turbinate[73] .

Hypoplastic / aplastic maxillary sinus

Aerated crista galli[91]

VASCULATURE OF NOSE

ARTERIAL SUPPLY[76]

ARTERY	BRANCH OF	AREA SUPPLIED
Anterior and posterior ethmoid artery	Ophthalmic artery	Roof of nose, upper lateral wall of septum, ethmoid and frontal sinuses
Sphenopalatine	Maxillary artery	Superior and middle meatus and septum
Greater palatine	Maxillary artery	Posterior part of lateral nasal wall and anteroinferior part of septum
Superior labial	Facial artery	Vestibule of nose
Infra orbital, posterosuperior alveolar and anterosuperior alveolar	Maxillary artery	Mucous membrane of maxillary sinus
Pharyngeal branch of maxillary artery	Maxillary artery	Sphenoid sinus
Branches from internal carotid artery		Sphenoid sinus

TYPE OF CELL	FUNCTION
Goblet cell	Secrete mucous blanket which protects nose
Columnar cell	Promote exchange across the epithelium
Basal cell	Provide adhesion between cells

The basement membrane separated from epithelium by lamina propria. The submucosa is invaded by the nerves , blood vessels and glands. There are three submucus glands as follows anterior serous glands and seromucinous glands responsible for nasal mucosa moisturization, while bowman's glands helps in olfaction[79] .

DEVELOPMENT OF LUNGS

In a four week old embryo lung bud appears as outpouching from the ventral wall of foregut[80] . As it expands the tracheoesophageal ridges appear dividing the foregut into dorsal portion esophagus and ventral trachea and lung buds.

At fifth week , lung buds forms trachea and two lateral outpouchings called bronchial buds[81] enlarging to form right and left bronchi. The right bronchi forms three secondary bronchi and the left forms two secondary bronchi. As a result three lobes in right lung and two lobes on the left side occur.

RIGHT LUNG	LEFT LUNG
Superior lobe- anterior, posterior,apical	Superior lobe- apical, posterior, anterior, superior lingular, inferior lingular
Middle lobe – lateral, medial	Inferior lobe- apical, medial basal,lateral basal, anterior basal,posterior basal
Inferior lobe- superior,medial basal,lateral basal,anterior basal and posterior basal	

BLOOD SUPPLY OF LUNGS

The bronchi, connective tissue and visceral pleura supplied by bronchial arteries branch of descending aorta. The bronchial veins drain into azygos and hemiazygos vein[87] .

NERVE SUPPLY OF LUNG

The root of lung has pulmonary plexus which is composed of afferent and efferent autonomic nerves[88] .

HISTOLOGY OF LUNGS[89]

The epithelia of trachea and principal bronchi is pseudostratified ciliated columnar epithelium. It consists of goblet cells and basal cells. The subepithelial connective tissue has elastic fibres, serous glands to keep epithelium moist and mucous glands.

The bronchi as it becomes smaller is lined by simple ciliated columnar, then non-ciliated columnar and finally simple cuboidal. Six type of epithelial cells has been demonstrated in conducting airways such as ciliated columnar, goblet,clara , basal, neuroendocrine and brush cells. In respiratory airways type I and type II pneumocytes citied[90] .

PHYSIOLOGY OF AIRWAYS

The nose and sinuses is considered as one organ though phylogenitcally nose is organ of olfaction and sinuses help in growth and structure of facial bones.

The functions of nose and sinuses are as follows[102]

1. Respiration
2. Humidification
3. Filtration
4. Nasal resistance
5. Heat exchange
6. Mucociliary clearance
7. Nasal reflexes
8. Voice modification
9. Protection of lower airway
10. Olfaction

Mucociliary transport or clearance is of utmost importance because it is responsible for drainage ,ventilation , humidity,filtration and olfaction purpose of sinuses and nose. The mucus in nose is secreted by the goblet cells and seromucinous glands. About 600 to 700ml of mucus is produced everyday. The two sinuses where goblet cells are large in number are ethmoid and maxillary sinuses. The mucus layer changes every 10 minutes in these sinuses. The genes coding mucin proteins are MUC5AC, MUC5B ,MUC2 and sixteen other genomes has been coded[95] .

COMPOSITION OF MUCUS

95%water

3% mucin

2% other particles

By electrophoresis proteins in mucus found out to be immunogloublins, glycoproteins (sialomucins, fucomucins, sulphomucins) help to trap foreign particles, albumin, prealbumin and lysozyme,lactoferrin, complement,macroglobulin, C-reactive protein and ions[92] . The nasal cilia is of size 7 micrometer long and 0.3 micrometer thick. Each nasal cell may have 200 to 300 cilia with 150 microvilli. Cilia is made up of nine pairs of microtubules around a central pair. There is three types of bond[93] in it

1.Nexin which are elastic doublets and act as small bridges

2. Dynein arms which coordinate the doublets to slide in synchyronisation

3. Spokes which keep the cilia from breaking apart

Understanding the structure of cilia is important to know the phases of mucociliary transport namely “sol” and “gel”. In sol phase or inner serous layer of mucus film when the cilia beat and transport the viscous outer layer or gel phase over it. The cilia beat 10 to 15 times per second. The cilia move individually in synchronized and metachronized pattern[94] . The biphasic motion of cilia has an effective phase and slow recovery phase.

FACTORS AFFECTING CILIARY MOTILITY[96]

Temperature below 32 c and more than 40 c

Bronchitis and asthma where mucus becomes viscous, mucus becomes alkaline unlike in infections where it becomes acidic.

Dehydration , nasal decongestants and diuretics stops cilia.

Deviated nasal septum causes desiccation of cilia.

Viral infections cause clumping of cilia.

Mycoplasma infection cause ciliary disorganization and results in ineffective motility.

Pseudomonas growth causes detachment of ciliated cells.

Allergens cause disruption of basal bodies.

Smoking results in reduction of ciliary beat frequency.

VitaminA overdosage cause squamous metaplasia.

Congenital conditions affecting ciliary motility are Kartagener's syndrome and immotile cilia syndrome.

TRANSPORTATION PATTERN IN SINUSES

The transport within sinuses are genetically programmed in a pattern that directs the flow of mucus in a predetermined manner from the interior of sinus to natural ostia of sinus and from there to nose. Any disruption in this pattern hinders the ventilation and drainage of sinuses resulting in rhinosinusitis.

MAXILLARY SINUS TRANSPORT[97]

The secretions are transported from the floor of the sinus in **stellate pattern.** It is transported along the anterior,medial,posterior, lateral wall and roof of the sinus to converge along the natural ostia.

FRONTAL SINUS TRANSPORT[98]

It is the only sinus where an active inwardly directed transportation of mucus. Mucus is transported along the interfrontal septum, laterally along roof and medially through the floor , inferior portions of the posterior and anterior wall of sinus from there the secretion exits via lateral aspect of ostia. Mucus is not clear in one round it accumulates in a **whorl like pattern**. Secretions from frontal and maxillary sinus merge and drain finally into nasopharynx.

ETHMOID SINUS[99]

Depending on the location of ostia mucus transportation in ethmoid sinus varies,

If in floor of ethmoid cell ostia is present – secretion is towards the ostia.

If ostia is located higher up as in bulla ethmoidalis- spiral pattern seen.

Presence of ground lamella divide the secretion transport between anterior ethmoids and posterior ethmoids . Anterior ethmoids drain anteroinferior to ground lamella whereas the posterior ethmoids drain posterosuperiorly.

SPHENOID SINUS[100]

There is a **spiral pattern** towards the ostia.

Drainage of sinuses into nasopharynx occur eventually where two routes have been observed.

The frontal sinus, maxillary and anterior ethmoids drain anteroinferior to eustachian tube orifice while posterior ethmoids and sphenoid sinus drain posterosuperior to Eustachian tube orifice[101] .

By way of filtration, humdification and immune propertis of nasal mucus it protects the lower airways.

IMMUNE FUNCTIONS OF NOSE[102]

DEFENSE MECHANSIMS	HUMORAL	CELLULAR
Mechanical	Mucus	Ciliary epithelium
Specific immune response	Complement,lysosome and lactoferrin	Granulocytes, macrophages
Non-specific immune re-sponse	Immunoglobulin	Lymphocytes

INNATE IMMUNITY

The nasal epithelium is a weak barrier against infections unlike mucus which is a protective barrier against invasion of micro organsims and toxic substances. Lysozyme has anti bacterial and bacteriostatic properties against gram positive bacteria, in case of gram negative bacteria it exerts lytic action by antibody activated complement system. Lactoferrin's microbicidal action against organism dependent on iron for metabolism eg: Candida Albicans. Secretory leukoprotease inhibitor, uric acid, peroxidase, amino peptidase,phospholipaseA_2 and defensins present in nasal mucus play major role in innate immune mechanism. Special mention of nitric oxide present in nose and sinuses is a must in defense mechanism of nose .

If the above mechanism fail non specific immune response is established. Release of bioactive and chemotactic factors like interleukins,cytokines and chemokines responsible for inflammation is observed in this type of response.

ADAPTIVE IMMUNITY

Humoral immunity combat infection and I_gE mediated responses by mucosa associated lymphoid tissue(MALT) present in nasopharynx, bronchi and gastro intestinal tract.

NASAL REFLEXES[103]

Nasal reflexes help to understand the central nervous system afferents and efferents mechanisms that cause local mucosal and systemic changes.

FUNCTIONS OF PARANASAL SINUSES[104]

The function of sinuses are as follows

1.Vocal resonance

2. Air conditioning

3.Reduction of skull weight

4.Mechanical rigidity

5.Heat insulation.

PULMONARY PHYSIOLOGY[105]

The lung's primary function is to facilitate exchange of gas , right from inspired gas to removal of carbon dioxide necessary of acid base balance. The inspired gas from upper airway reaches the terminal bronchiole or conducting zones through transition zone and respiratory zone. Thus pulmonary physiology deals with breathing mechanics, ventilation –perfusion interchange and gaseous diffusion.

To understand the above mechanics lung volumes and lung capacities should be discussed. The four standard lung volumes[106] are

LUNG VOLUMES

Tidal volume- volume of gas exchanged by quiet respiration it is 500ml.

Expiratory reserve volume- extra volume of air which is expired forcefully and accounts to 1100ml.

Inspiratory reserve volume- extra volume of air which can be inspired forcefully it is about 3000ml.

Residual volume- amount of air remaining in lungs after forceful expiration is 1200ml.

LUNG CAPACITIES

Functional residual capacity- amount of gas remaining in lungs after normal expiration and is about 2300ml.it is determined by elastic recoil pressure of the lung

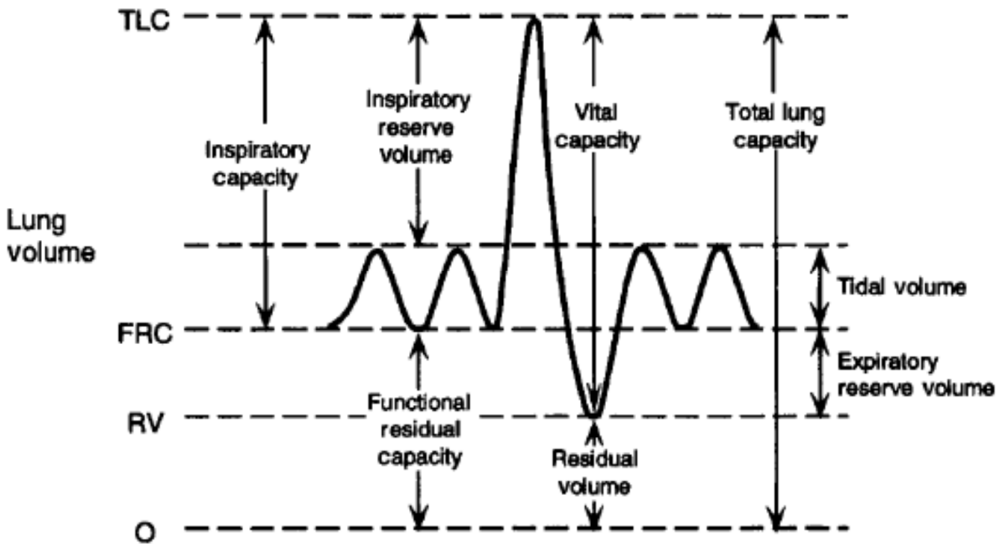
and outwardly directed chest wall recoil pressure(total lung capacity – inspiratory capacity).

Total lung capacity – volume of gas contained in lung after a maximal inspiration (vital capacity + residual volume). It is about 5800ml.

Inspiratory capacity is sum of tidal volume and inspiratory reserve volume. It is about 3500ml.

Vital capacity is sum of inspiratory reserve volume, residual volume and expiratory reserve volume.

All the above mentioned volumes and capacities are measured by **spirometer** as shown below in the diagram



FLOW-VOLUME LOOP [107]

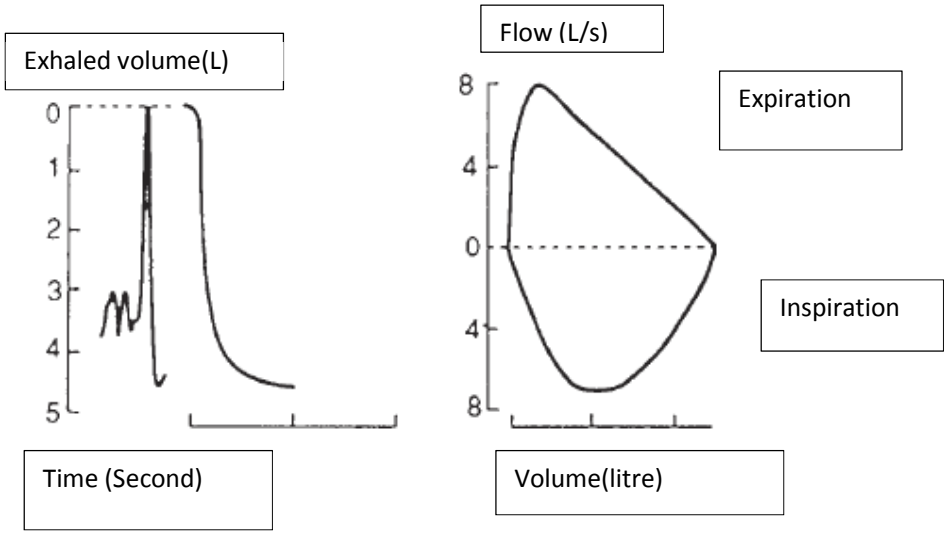
The mechanical properties of lung is measured by flow-volume loops.

The flow rate is plotted against change in lung volume over time.The upper limb of the flow volume loop represent forced expiratory flow rate and the peak of it signifies

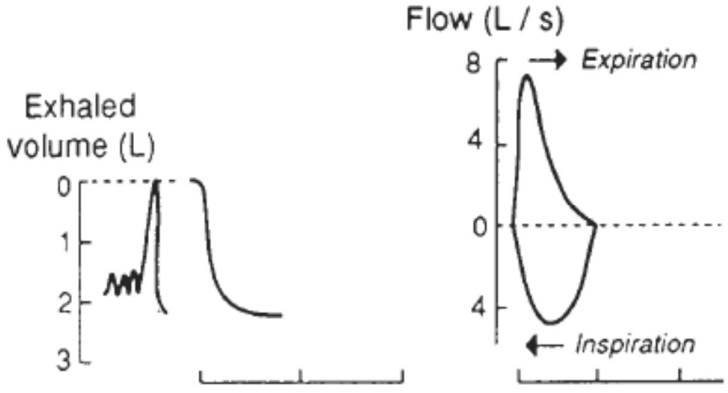
the peak expiratory flow rate(PEFR). PEFR is 8litres per second and occurs at 90% of vital capacity.

In restrictive lung disease there is reduction in PEFR whereas in obstructive lung disease miniature pattern of a normal curve with symmetric reduction in lung volume is noted.

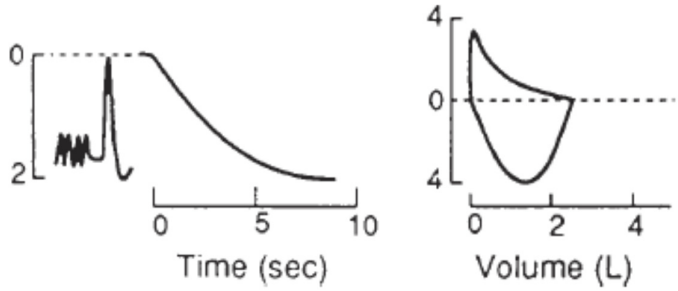
NORMAL FLOW-VOLUME LOOP



RESTRICTIVE LUNG DISEASE



OBSTRUCTIVE LUNG DISEASE



MEASUREMENT OF VENTILATION [108]

RESTING VENTILATION

The resting ventilation is calculated by measuring minute ventilation.

Minute ventilation is the volume of gas exhaled per minute. In turn estimated by multiplying tidal volume and respiratory rate per minute.

Minute ventilation = tidal volume×respiratory rate/minute(500X12= 6litres/min).

DEAD SPACE VENTILATION

Some of the air a person breathes never reaches the gas exchange areas but also fills respiratory passages where gas exchange doesn't occur as in nose,pharynx and trachea. This is called as **anatomical dead space**. When it includes alveolar dead space also it is called as **physiological dead space**.

It accounts to about 150ml and 66ml of it is contributed by extrathoracic pharynx and mouth.

By tracheostomy , anatomical dead space is decreased by 60%.Expired minute ventilation measures the dead space ventilation.

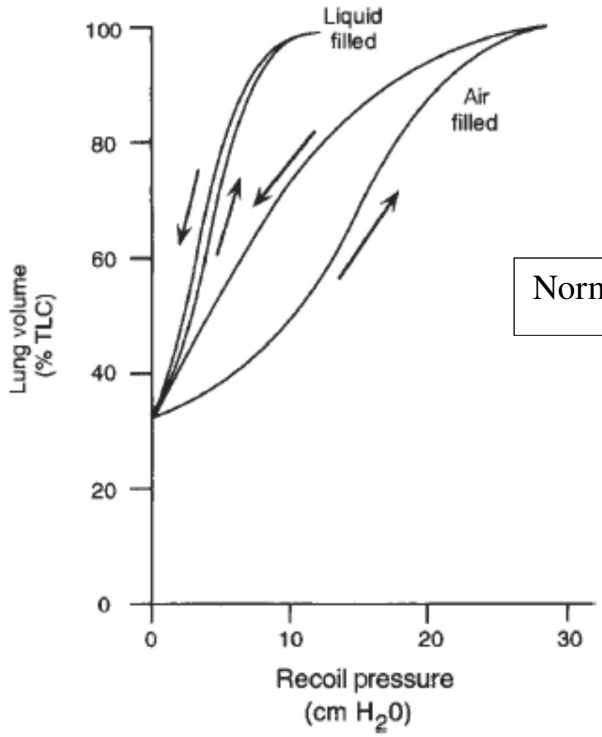
Expired minute ventilation = alveolar ventilation+volume of gas exchanged between blood and alveoli+ volume of gas wasted ventilating dead space.

COMPLIANCE OF LUNG[109]

Lung compliance is the change in lung volume observed per unit change in pressure gradient between the intraplueral space and alveoli. A static pressure- volume curve of the lung is marked by interrupting expiratory or inspiratory maneuver using a esophageal balloon tipped catheter present in mid esophagus. The midcurve slope of pressure-volume curve represents static compliance.

The curve is shifted upward and to left in cases of emphysema and old age where static compliance is increased.

It is shifted downward and to right in cases of interstitial lung disease,pneumonia, adult respiratory distress syndrome and cardiogenic pulmonary edema.



Normal static compliance curve

PATHOPHYSIOLOGY OF SINUSITIS [110]

The effective functioning of paranasal sinuses require

Patent osteomeatal complex

Adequate mucociliary clearance

Normal mucus production and consistency

Anatomic or acquired obstruction of the ostia is primary factor in intiation of sinusitis. Obstruction of ostia could be caused by local or systemic factors. Local factors like septal deviation, polyps, concha bullosa while systemic factors like bacterial or viral infections cause mucosal edema resulting in ostia obstruction.

Mucociliary clearance is interrupted by intrinsic and extrinsic and factors.

Intrinsic factors include ciliary dyskinesia ,alteration in local nitric oxide production and cystic fibrosis which alters the mucus consistency. Extrinsic factors like exposure to tobacco smoke, infection with viral upper respiratory pathogens.

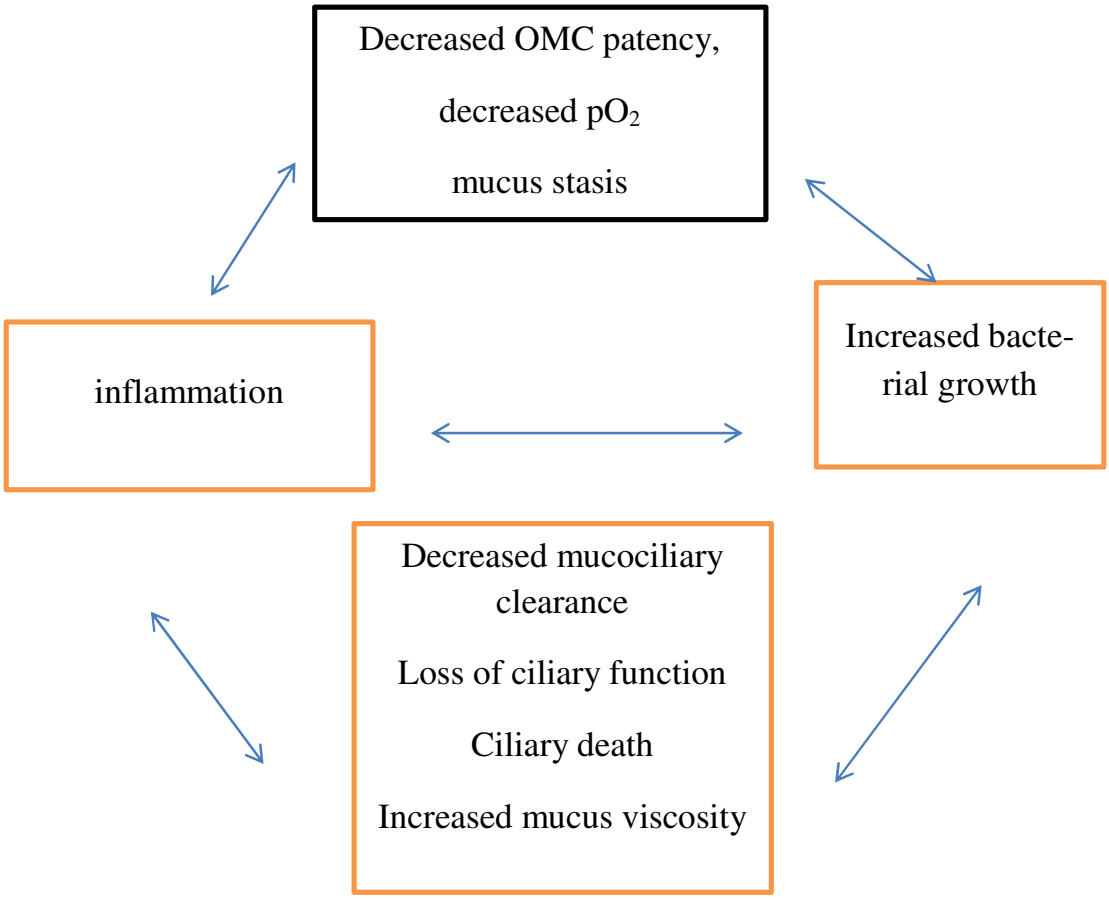
Viral infections induce production of cytokines eliciting cellular inflammation thereby increasing vascular permeability as a result edema and ciliary dysfunction.

In inflammatory conditions nitric oxide production is altered leading to decreased antiviral and bacteriostatic protection of the mucus and favouring bacterial colonization and infection.

DEFENSE MECHANISMS IN SINUSITIS[111]

Humoral immunity carried out by B-lymphocytes mediated by I_gA antibodies in detecting and removal of microbial agent by opsonization.

Cell mediated immunity by T-lymphocytes,macrophages and defensin cytokines. Defensins present in mucus binds to microbial agent causing membranr disruption, internalization of defensins and cell death eventually. Interruption in any of the above factors lead to sinusitis.



The classification of sinusitis is based on duration of symptoms and signs suggested by 1996 AA0-HNS Task Force Criteria is as follows

Major factors	**Minor factors**
Facial pain/pressure Facial congestion/fullness Nasal obstruction/blockage Nasal discharge/purulence/discolored postnasal discharge Hyposmia/anosmia Purulence in nasal cavity on examination Fever	Headache Fatigue Halitosis Dental pain Cough Ear pain/pressure/fullness

Classification	Duration	History
Acute	< 4 weeks	>2 major factors, 1 major factor and 2 minor factors or nasal purelence on examination
Subacute	4-12 weeks	>2 major factors, 1 major factor and 2 minor factors
Recurrent acute	>4 episode per year, with each episode lasting 7 to 10 days with absence of signs and symptoms of chronic sinusitis	>2 major factors, 1 major factor and 2 minor factors
Chronic	>12 weeks	>2 major factors, 1 major factor and 2 minor factors

PATHOPHYSIOLOGY OF ASTHMA[112]

Asthma is characterized by hyperreactive airways, leading to episodic , reversible bronchoconstriction,owing to increased responsiveness of the tracheobronchial tree to various stimuli.

The airflow obstruction of asthma is due to bronchial smooth muscle contraction, mucosal edema and thick mucus secretion.small bronchi and bronchioles are involved with excessive airway denudation due to thinning of epithelium and hyperplasia of goblet cells.

The basement membrane is thickened by collagen deposition, infiltration of CD4+ lymphocytes,mast cells,eosinophils and neutrophils. The submucous muscle is contracted. Submucous glands are hyperplastic secreting mucus. Reduction in small airway diameter increases airway resistance as a result expiratory flow rates and flow volumes are decreased. The lumen of bronchi are filled with mucus,eosinophils, edema fluid, Charcot- leyden crystals, inssipitated mucus plugs and Crushmann spirals.

SIMILARITIES BETWEEN UPPER AND LOWER AIRWAY[113]

Pseudostratified ciliated columnar epithelium

Lamina propria

Triangular basal cells

Goblet cells

MECHANSIMS RELATED TO SINUSITIS AND ASTHMA[114]

The mechansims which link sinusitis and asthma are as follows

EOSINOPHIL

Eosinophil plays a major role in mediating injury to epithelium caused in bronchial asthma and sinusitis. The deposition of eosinophils in sinus mucosa correlates with cytokine production such as interleukin-2(IL-2) and granulocyte macrophage colony stimulating factor (GM-CSF) resulting in extracellular deposition of major basic protein and sinus mucosa damage. The same mechanism has been described in case of bronchial asthma also and demonstrated by histopathological specimens. All these suggest eosinophil acts as an effector cell in chronic inflammatory disease of sinus and lower airway.

INFLAMMATORY MEDIATORS

Sinusitis acts as an aggravator for bronchial asthma by stimulating irritant receptors leading to bronchospasm by the inflammatory mediators. The induction of Th2-type cells by the inhaled antigen or allergen causes release of interleukins IL-4 and IL-5.

Activation of eosinophil and recruitment of mast cells are taken care by these interleukins. This forms the base for I_gE mediated hypersensitivity in acute and late response. The sensitized mast cells cause mediator release and penetration of the antigen to the submucosal mast cells. Bronchoconstriction occurs by central and local reflexes explained below in the neural pathways. This is acute phase reaction. The late phase reaction is triggered by the release of cytokines from mast cells and other inflammatory cells.

The leukotrienes C_4,D_4, and E_4 are potent mediators that cause prolonged constriction of the bronchi , increased vascular permeability and mucus production. The smooth muscles of airway is constricted by acetylcholine released from the interpulmonary motor nerves by stimulation of muscarinic receptors.

Local mediators of inflammation like histamine, prostaglandin D_2 , platelet activating factor may be aspirated to lungs resulting in seeding of lower airways with nasal mucopurulent secretions.

NEURAL PATHWAYS

The neuroanatomic reflex pathway which connects nose and lung is as follows

Receptors in nose , pharynx, paranasal sinuses form afferent fibres of trigeminal nerve which relay in reticular formation in brain stem.

The dorsal stem nuclei of vagus in reticular formation gives rise to parasympathetic vagal efferent cholinergic fibres which reach bronchi smooth muscle.

All the above said mechanisms putforth the theory of unified airway or single airway linking upper airway and lower airway and relationship between pathogenesis of sinusitis and bronchial asthma.

MATERIALS AND METHODS

INCLUSION CRITERIA :

1. History of bronchial asthma .
2. History of persistence of symptoms of asthma for atleast six weeks inspite of COPD management.
3. 18-55 year age group
4. Asthmatic patients with history suggestive of chronic sinusitis

EXCLUSION CRITERIA :

The patients with

1. H/o chronic smoking
2. Systemic illness
3. Pregnancy
4. Lactating mothers
5. H/o allergy to antibiotics

PLACE OF STUDY : Thanjavur medical college

PERIOD OF STUDY : July 2015 to September 2016

EVALUATION OF PATIENTS

Patients with asthma usually present with symptoms of shortness of breath (dyspnea), wheeze ,cough and chest tightness as suggested by National heart,lung and blood institute guidelines for diagnosis and management of asthma[125] .

The patients were graded according to National Asthma Education and Prevention Programme[126]

DISEASE SEVERITY	SYMPTOMS	NOCTURNAL OCCURRENCE	LUNG FUNCTION
Mild intermittent	Symptoms less than two times a week Asymptomatic and normal PEF between exacebrations	< 2times per month	PEF >80% of predicted PEF variability <20%
Mild persistent	Symptoms >2times a week but less than 1 time a day Exacebrations may affect activity	>2times a month	PEF >80%of predicted PEF variability 20-30%
Moderate persis-tent	Daily symptoms Daily use of short acting β_2 agonist in-haler Exacebrations>2times a week and it affects activity	>1time a week	PEF >60% but <80% predicted PEF variability >30%
Severe persistent	Continual symptoms Limited physical activity Frequent exacebrations	Frequent	PEF <60% predicted

PEAK EXPIRATORY FLOW RATE[127]

Measurement of peak expiratory flow is of value in assessment and follow-up of patients with asthma. Wright's peak flow meter has been used here .It is the maximal flow by expiration in maximal force after a full inspiration. There is a diurnal variation in peak expiratory flow rate , the lowest values occurring in morning and highest during 4 to 6 pm.

Each measurement should be highest value of three consecutive maneuvers before usage of bronchodilators. Peak expiratory flow rate provides objective measurements to follow and monitor therapy.

The values are usually divided into three zones

ZONE	**PEFR VALUE**	**GRADE OF ASTHMA**
GREEN	>80% expected PEFR	Mild
YELLOW	50- 80% expected PEFR	Moderate
RED	< 50% expected PEFR	Severe

FORMULA FOR PEFR CALCULATION[128]

MALES

AGE	7-15Yrs	16-40Yrs	>40yrs
FORMULA	(5×Ht in cms)-420	(2.74×Ht in cms)+53.4	567-(2.24×age)
Normal value	80-450 l/min	400-650 l/min	300-500 l/min

FEMALES

AGE	7-15Yrs	16-40Yrs	>40Yrs
FORMULA	(5×Ht in cms)-430	(4.65×Ht in cms)-360	438-(2.24×Age)
NORMAL VALUE	70-400 l/mins	250-400 l/min	200-400 l/min

Diagnostic criteria of chronic rhinosinusitis

1. Persistence of symptom for 12 weeks or more with findings of sinusitis on examination and or radiological evaluation.
2. One of these signs in association with the symptoms
   a) Discoloured drainage, nasal polyp or polypoidal mucosa on examination with anterior rhinoscopy or endoscopic examination.
   b) Edema or erythema of middle meatus in nasal endoscopic examination.
   c) Generalized edema or erythema, granulation tissue(if middle meatus or ethmoid bulla not involved imaging is required.
   d) CT scan of paranasal sinuses demonstrating mucosal thickening, air- fluid level or bony changes.
   e) Plain X-ray of paranasal sinuses showing mucosal thickening more than 5mm or complete opacity.

For diagnosis presence of two major factor or one major and two minor factors or mere presence middle meatus purulence is enough.

When the above criteria are met, patients are subjected for diagnostic nasal examination using 0º and 30º Hopkins rod endoscope under local anesthesia. The endoscopic findings were graded using Lund Kennedy endoscopic grading system and scored as

Polyp – 0 – absent,1- within the middle meatus, 2- beyond the middle meatus

Discharge – 0- absent,1- thin clear , 2- thick purulent

Edema -0- absent , 1-mild, 2- severe

In post operative endoscopic examination scoring of scarring and crusting was also considered

Scarring -0-absent, 1-mild, 2-severe

Crusting -0-absent, 1-mild, 2- severe

The radiological imaging scoring by Lund MacKay system with each side sinuses and osteomeatal complexes considered separately and scored. The status of maxillary, anterior ethmoids, posterior ethmoids, frontal and sphenoid sinuses as

0- No abnormality

1- Partial opacity

2- Complete opacification

Osteomeatal complex

0- No obstruction

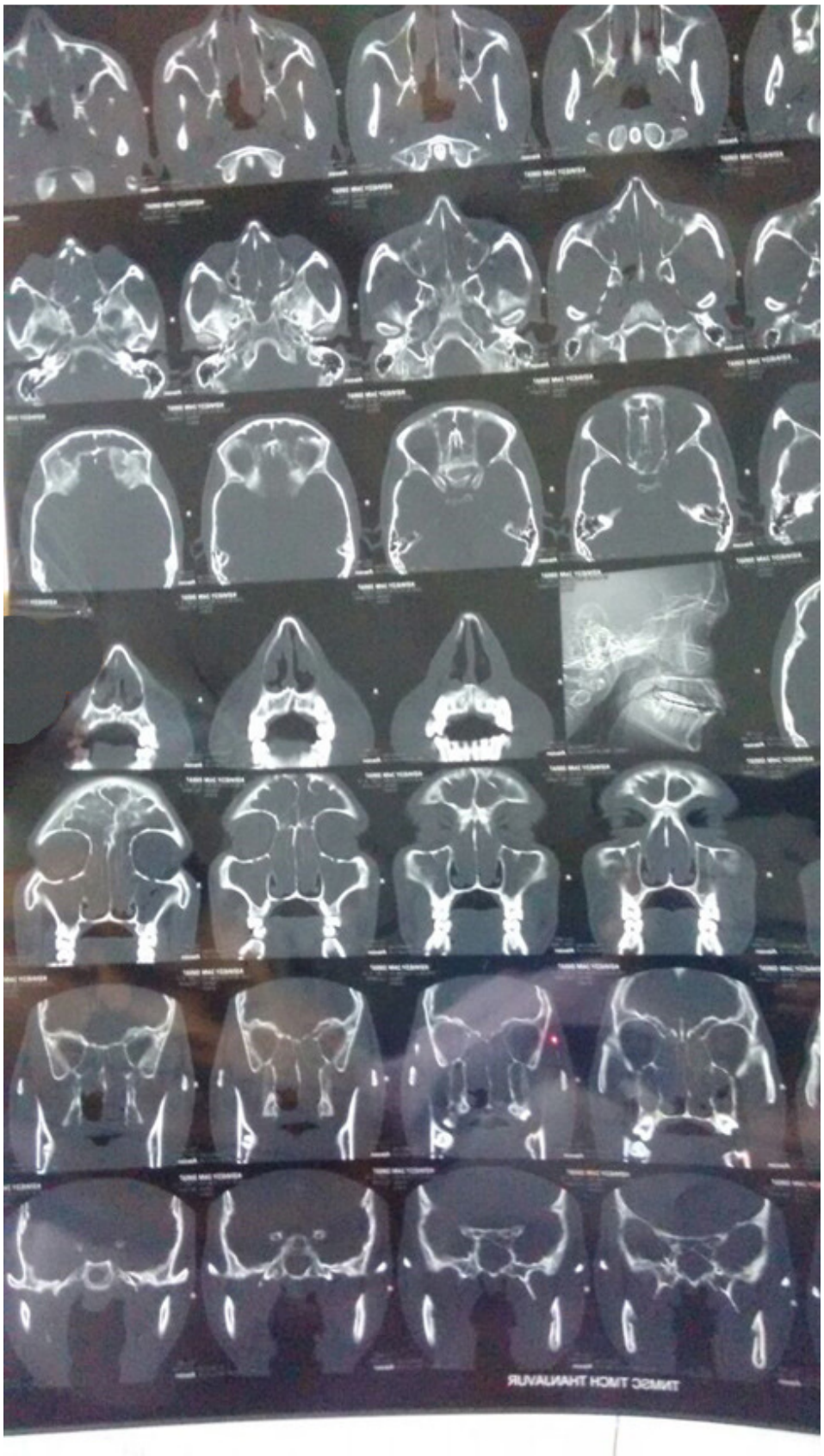
2-obstructed

Drug consumption in treatment of asthma was also considered in the study giving an arbitrary score of one point each for prednisolone tablet, salbutamol tablet, theophylline tablet and inhaler puff usage. Two points for nebulization and injection.

RESULTS AND OBSERVATION

50 asthmatic patients who presented with sinusitis were screened using Task force criteria and subjected to nasal endoscope and plain X-ray of paranasal sinuses(Water's view) and CT scan of paranasal sinuses if necessary. Out of the 50 patients , 8 patients were selected as control group by random selection method. The remaining 42 patients were treated medically with antibiotics, antihistamines and nasal decongestant drops for a period of three weeks.

Before starting medical management peak expiratory flow rate was recorded in all of them. 25 patients responded well with medical treatment and 17 patients were refractory to medical management. In those patients who were treated medically peak expiratory flow rate was recorded six weeks after stopping medical management . The 17 refractory patients to medical management were subjected to functional endoscopic sinus surgery and measurement of peak expiratory flow rate done after six weeks postoperatively .



AGE DISTRIBUTION CHART

Patients were selected between 18 to 55yrs of age and the distribution as follows

AGE	TOTAL	FEMALE	MALE
15-20	3	1	2
21-25	12	3	9
26-30	8	2	6
31-35	11	5	6
36-40	3	3	0
41-45	5	2	3
46-50	2	2	0
51-55	6	6	0

MEDICALLY MANAGED PATIENTS

AGE /SEX	BEFORE MEDICATION (cells/cu.mm)	AFTER MEDICATION (cells/cu.mm)
19/F	783	385
21/F	672	376
24/F	527	330
28/F	439	234
31/F	432	231
31/F	863	308
33/F	544	264
36/F	512	236
37/F	843	323
39/F	636	307
44/F	480	237
48/F	428	236
52/F	819	205
53/F	567	342
55/F	921	325
55/F	537	208
20/M	440	236
21/M	754	247
22/M	611	344
24/M	933	311
25/M	504	219
26/M	657	264
29/M	864	262
29/M	859	307
31/M	536	276

SURGICALLY MANAGED PATIENTS

AGE/SEX	BEFORE SURGERY (cells/cu.mm)	AFTER SURGERY (cells/cu.mm)
18/M	566	321
22/M	819	335
25/M	1023	347
25/M	413	282
28/M	532	302
30/M	510	340
33/M	497	276
35/M	382	180
41/M	372	198
41/M	526	334
24/F	527	330
30/F	908	327
35/F	618	332
42/F	873	310
46/F	456	204
51/F	523	318
53/F	921	325

CONTROL GROUP

AGE/SEX	START OF STUDY (cells/cu.mm)	AFTER SIX WEEKS (cells/cu.mm)
21/M	633	602
25/M	791	785
29/M	1033	1034
33/M	434	420
33/M	590	586
35/M	948	950
44/M	604	604
31/F	473	475

DRUG CONSUMPTION CHART

The changes in consumption of usage of drug was noted before the start of study and end of six weeks after starting treatment.

AT START OF STUDY

Drug	Medically managed patients	Surgically managed patients	Control group
Prednisolone(1)	25	17	8
Salbutamol(1)	8	7	2
Theophylline (1)	12	6	5
Inhaler / Rotacap(1)	4	4	1
Nebulization (2)	1	2	1

In the patients who were managed by medications, all 25 of them were on steroids, 8 of them on salbutamol, 12 on theophylline, 4 on usage of inhaler or rotacap and one of them required nebulization along with other drugs usage.

In surgically managed patients all 17 of them were on steroid , 7 on salbutamol, 6 on theophylline, 4 on inhaler and 2 requiring nebulization along with other medications.

In control group the whole sample of 8 require steroids , 2 on salbutamol, 5 on theophylline and one inhaler and nebulization.

IN MEDICALLY MANAGED PATIENT

The following chart shows the changes in PEFR before and after medication

AGE/SEX	HEIGHT IN Cms	EXPECTED VALUE (l/min)	BEFORE MEDICATION (l/min)	AFTER MEDICATION (l/min)
19/F	138	282	186(66%)	225(80%)
21/F	142	300	252(84%)	282(94%)
24/F	135	268	206(77%)	241(90%)
28/F	150	338	250(74%)	314(93%)
31/F	155	360	298(83%)	313(87%)
31/F	160	384	215(56%)	326(85%)
33/F	148	328	255(78%)	295(90%)
36/F	152	347	277(80%)	326(94%)
37/F	154	356	252(71%)	320(90%)
39/F	168	421	286(68%)	362(86%)
44/F	157	340	292(86%)	312(92%)
48/F	153	331	251(76%)	307(93%)
52/F	144	322	280(87%)	302(94%)
53/F	153	320	220(69%)	272(85%)
55/F	162	315	204(65%)	277(88%)
55/F	147	315	264(84%)	283(90%)
20/M	147	457	301(66%)	374(82%)
21/M	158	487	354(73%)	438(90%)
22/M	136	426	332(78%)	391(92%)
24/M	152	470	385(82%)	413(88%)
25/M	160	491	387(79%)	427(87%)
26/M	166	508	320(63%)	447(88%)
29/M	170	519	446(84%)	482(93%)
29/M	153	473	335(71%)	425(90%)
31/M	148	459	312(68%)	399(87%)

PEAK EXPIRATORY FLOW IN SURGICALLY MANAGED PATIENTS

The following chart shows changes in PEFR pre and postoperative

AGE/SEX	HEIGHT IN Cms	EXPECTED VALUE(l/min)	PREOPERATIVE (l/min)	POSTOPERATIVE (l/min)
18/M	152	470	376(80%)	423(90%)
22/M	160	491	392(80%)	451(92%)
25/M	148	478	239(50%)	320(67%)
25/M	155	459	275(60%)	358(78%)
28/M	163	501	350(70%)	420(84%)
30/M	168	514	334(65%)	442(86%)
33/M	174	530	275(52%)	397(75%)
35/M	161	495	331(67%)	396(80%)
41/M	156	475	256(54%)	327(69%)
41/M	164	475	399(84%)	446(94%)
24/F	132	254	152(60%)	190(75%)
30/F	140	291	226(78%)	241(83%)
35/F	150	338	216(64%)	270(80%)
42/F	154	348	295(85%)	323(93%)
46/F	147	335	244(73%)	288(86%)
51/F	156	324	197(61%)	301(93%)
55/F	147	320	197(57%)	284(89%)

PEAK EXPIRATORY FLOW RATE IN CONTROL GROUP

AGE	HEIGHT IN Cms	EXPECTED (l/min)	AT START OF STUDY	AFTER SIX WEEKS
21/M	163	500	330(66%)	330(66%)
25/M	158	486	398(82%)	388(80%)
29/M	154	475	351(74%)	351(74%)
33/M	170	519	290(56%)	290(56%)
33/M	156	481	365(76%)	365(76%)
35/M	148	459	307(67%)	298(65%)
44/M	151	468	397(85%)	374(80%)
31/F	152	346	214(62%)	214(62%)

STASTICAL ANALYSIS OF DATA

MEDICALLY TREATED PATIENTS

The sample is 25. Applying paired sample ‘t' test

	Mean	S.D	Correlation value	Statistical inference	mean	S.D	T	d.f	Statistical inference
Pair1									
Expected value	386.96	81.01	.880	.000<.05 significant	97.0	38.84	12.48	2	.000<.05 significant
Before medication	289.96	66.48							
Pair2									
Expected value	386.96	81.01	.976	.000<.05 significant	44.1	18.61	11.8	24	.000<.05 significant
After medication	342.80	72.847							
Pair3									
Before medication	289.96	66.48	.917	.000<.05 significant	52.84	29.01	9.105	24	.000<.05 significant
After medication	342.80	72.847							

SURGICALLY MANAGED PATIENTS

The sample size is 17 and applying paired sample 't' test

	Mean	S.D	Correla-tion value	Statisti-cal in-ference	Mean	S.D	t	Df	Statisti-cal in-ference
Pair 1									
Expected value	417.53	91.52	.752	.001<.05 significant	138.76	60.67	9.43	16	.000<.05 significant
Pre-operative	278.76	75.01							
Pair2									
Expected value	417.53	91.52	.885	.000<.05 significant	71.82	42.67	6.94	16	.000<.05 significant
Post-operative	345.71	78.59							
Pair3									
Pre-operative	278.76	75.01	.925	.000<.05 significant	66.94	30.009	9.19	16	.000<.05 significant
Post-operative	345.71	78.59							

CONTROL GROUP

The sample size is 8 and applying paired sample 't' test

	Mean	S.D	Correlation value	Statistical value	Mean	S.D	T	df	Statistical inferences
Pair1									
Expected value	466.75	52.262	.631	.093>0.05 Not significant	135.25	49.491	7.73	7	.000<.05 significant
At time of study	331.50	61.349							
Pair2									
Expected value	466.75	52.262	.669	.070>.05 Not significant	140.50	44.779	8.875	7	.000<.05 significant
After 6 weeks	326.25	57.238							
Pair3									
At time of study	331.50	61.349	.992	.000<.05 significant	5.25	8.362	1.776	7	.119>.05 Not significant
After 6 weeks	326.25	57.238							

Applying Chi-square test for

GENDER SAMPLE - is significant

Gender	Medically treated	Surgically treated	Control	Total	Statistical inference
MALE	9(36%)	10(58.8%)	7(87.5%)	26(52%)	X^2=6.920 Df=2 P value .031<.05 significant
FEMALE	16(64%)	7(41.2%)	1(12.5%)	24(48%)	

DISCUSSION

Upper and lower respiratory tracts respond to stimuli in similar manner and provocation of upper airway produce changes in lower airway has been demonstrated. The pulmonary component of asthma when treated alone produces only short term relief and response in patient health condition but, when combined treatment of upper airway also ensures entire disease management.

In this study which comprise of patient from 18 to 55years age group,6% of sample size by 15-20 age group, 24% of them were in 21-25 age group followed by 16% of sample size by 26-30 age group,31-35 age group comprising 22% of the sample size.36- 40 yr age group account for 6% of sample size, 10% by 41-45 age group, 4% by 46-50 yrs sample and 12% by 51-55 yr age group of the sample size.

There was significant influence of allergic component and environmental factors visualized by 52% of study population was male and rest 48% by females , supported by Chi-square test results.

Monitoring of eosinophil count showed good improvement in the counts following the treatment of sinusitis in asthmatic patients. In the medically managed patients the eosinophil mean which was 646 at the start of study improved to 280 after six weeks of treatment .In the surgically treated patients also significant change in mean value prior to surgery was 615 and after surgery was 297. Whereas in the control group there was no change in mean value of eosinophil count. The overall shift of baseline mean value of the eosinophil count from 642 to 350 following treatment of sinusitis was noted. This suggest control of pathogenesis of asthma in

concomitant treatment of sinusitis documented by the decrease in eosinophil count which is the major effector cell in release of cytokines in both acute and late phase reaction of asthma triggering the mast cells and causing bronchoconstriction . The stimulation of airways significantly reduce following treatment of sinusitis.

The drug monitoring in patients showed reduction in usage of steroids, salbutamol, theophylline , inhaler and nebulization. At the start of study 100% of the sample size was on steroid therapy accounting to 50% of the sample in medically managed category, 34% in surgically treated patients and 16% of the sample size in control group.

In the 25 patients medically managed patients, 100% of them needed steroid medication for asthma which reduced to 64% after addressing treatment for sinusitis. 32% of the patients were on salbutamol which fell down to 16% after treatment for sinusitis. 48% of the patients on theophylline reduced to 20% and the usage of inhaler in 16% of patients did not require them after treatment for sinusitis.

In the 17 surgically treated patients, 100% of the patients in this category were on steroid usage reduced to 45% after surgery for sinusitis. In case of salbutamol usage 35% of the patients before surgery trickled down to 20% after surgery. 30% of the patients on theophylline documented a reduction to 10% after surgery. The 20% of the patients requiring inhaler subsided to 10% after surgery.

In the control group there was no change in the requirement of medications neither at the start of study nor at the end of six weeks starting the study.

The overall effect of sinus treatment in asthma showed reduction in usage of steroids in 64% of patient, 57% reduction in β_2 agonists and almost nil usage of nebulization.

PEFR was used as tool in assessing clinical improvement in patient's health and day to day activites, also to measure efficacy of treatment prior to start of study and after start of study in tackling sinusitis in asthmatic patients.

In medically treated patients the PEFR mean baseline value prior to treatment was 289.96 l/min substantial increase was observed at the end of six weeks after starting medications to 342.80 l/min.

In surgically managed patients, the PEFR value at the start of trial was 278.76 l/min grossly increased to 345.71 l/min postoperatively.

Whereas in the control group there was no change in the PEFR value. All these have been statistically analysed and found to have 'p' value which was significant in medically managed and surgically treated group unlike control group where 'p' value was insignificant by paired sample 't' test.

Similar observations has been studied by Slavin[129] in 1982 where he found that corticosteroid dependency reduced following simultaneous treatment of sinusitis in asthmatic patients.

In the study conducted in Government medical college of Aurangabad[130] in year 1986 evaluated the PEFR value changes in asthmatic patients following treatment of sinusitis with antibiotic, antihistamines and nasal decongestants. The baseline PEFR mean value 197.2 l/min increased to 250.4 l/min after sinusitis treatment. There was 60% reduction in asthmatic drug medication following sinusitis treatment.

Royal national throat,nose and ear hospital in London[131] conducted a study in the year 2005 found out improvement in asthma symptoms and reduction usage of asthmatic medications by addressing problem of sinusitis with medications and functional endoscopic sinus surgery.

The effectiveness of medical therapy is due to combined administration of antibiotics, antihistamines and nasal decongestants which control the inflammatory response in upper airway by decreasing the microbiological load, reducing number of leukocytes and downregulating cytokines and inflammatory mediators.

Surgically managed patients also showed improvement in both subjective and objective lower airway parameters.

CONCLUSION

Sinusitis and asthma may co-exist and impact the effect of pathogenesis of the disease on one another.

Identification of asthmatic patients with sinusitis is an important task which may be carried out by proper history, examination , necessary investigation and imaging. CT scan of paranasal sinuses is the key diagnostic tool in hand with endoscopic examination to detect isolated sphenoid and ethmoid sinusitis, anatomical abnormalities, osteomeatal complex obstruction by playing an upper hand in diagnosing sinusitis than conventional X-rays .

The role of sinusitis in pathogenesis of asthma is accomplished by neuro anatomical pathway that connects paranasal sinuses to lungs. Production of inflammatory mediators, eosinophil, mast cells in sinusitis resulting in subsequent recruitment of the same into lungs suggest the common effector pathway in pathogenesis of sinusitis and asthma.

Proper medical and surgical management of sinusitis in asthmatic patients results in improvement in lung function, allergic response, PEFR value increase as been observed in this study. Decreased exacerbation of asthmatic attacks, decreased consumption of drugs, minimal usage of inhaler and emergency hospitalization thus improves the quality of life style in asthmatic patients along with decrease in overall cost of health care. It has also been documented in this study medical therapy is superior to surgical management in course of the study.

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APPENDIX -1

SCREENING PROFORMA

Name : Age: Sex: Occupation:

Chief complaints:

History :

Nasal discharge

Nasal obstruction

Headache

Facial pain

Postnasal discharge

Halitosis

Ear pain/ear fullness

Dental pain

Abnormalities in smell

h/o epistaxis

H/o sneezing

H/o allergy

Fever &cough

Change in voice

H/o breathlessness

H/o asthma

Frequency of asthmatic attacks, diurnal and nocturnal variation

Asthmatic drugs used

Precipitating and relieving factors

Family h/o asthma, COPD, TB, DM,HT

Personal H/o : smoking, alcohol intake, snuff usage,betel nut usage, previous nasal surgeries

GENERAL PHYSICAL EXAMINATION

Built:

Nourishment :

Pallor/Icterus/lymphadenopathy/Clubbing/Cyanosis

Vital signs

Pulse rate: Respiratory rate : Temperature:

Blood pressure:

SYSTEMIC EXAMINATION

Cardiovascular system:

Respiratory system

Central nervous system

Abdomen examination

LOCAL EXAMINATION

NOSE

External appearance

Anterior rhinoscopy :

Vestibule

Columella

Floor of nasal cavity

Septum

Turbinate – inferior and middle

Meatus – inferior and middle

Post nasal examination

Cold spatula test

Paranasal sinus tenderness elicitation- Frontal,Maxillary,Ethmoid sinus

Ear and Throat examination

APPENDIX -2

DIAGNOSTIC NASAL ENDOSCOPIC FORM

	RIGHT	LEFT
FIRST PASS		
SEPTUM		
NASAL MUCOSA		
INFERIOR TURBINATE		
INFERIOR MEATUS		
CHOANA		
EUSTACHIAN TUBE		
FOSSA OF ROSENMUL-LER		
SECOND PASS		
MIDDLE TURBINATE		
MIDDLE MEATUS		
UNCINATE		
BULLA ETHMOIDALIS		
ACCESSORY OSTIUM		
THIRD PASS		
SPHENOETHMOIDAL RECESS		
SUPERIOR TURBINATE		
SUPERIOR MEATUS		

APPENDIX – 3

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by Department of Otorhinolaryngology, Thanjavur medical college hospital and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Place :

Date : Signature of participant

MASTER CHART

MEDICALLY MANAGED PATIENTS

S.NO	NAME AGE/SEX	HEIGHT IN Cms	SINUSITIS EVAL-UATION		ASTHMA EVALUATION BEFORE MEDICATION		AFTER MEDICATION	
			DNE	CT	GRADE	PEFR	GRADE	PEFR
1	Sumathi 19/F	138	M,E	M,E	Mo	186	Mi	225
2	Saranya 21/F	142	E	E	Mi	252	Mi	282
3	Vimala 24/F	135	M,E	M,E	Mo	206	Mi	241
4	Irudaya mary 28/F	150	M,E	M,E,S	Mo	250	Mi	314
5	Sugashini 31/F	155	M,E	M,E	Mi	298	Mi	313
6	Kalpana 31/F	160	M,E	M,E	Se	215	Mi	326
7	Malarvizhi 33/F	148	M	M	Mo	255	Mi	295
8	Jeyakodi 36/F	152	M,E	M,E	Mi	277	Mi	326
9	Kala 37/F	154	M,E	M,E	Mo	252	Mi	320
10	Sasikala banu 39/F	168	M,E,S	M,E,S	Mo	286	Mi	362

S.NO	NAME AGE/SEX	HEIGHT IN Cms	SINUSITIS EVALUATION		ASTHMA-EVALUATION BEFORE MEDICATION		AFTER MEDICATION	
			DNE	CT	GRADE	PEFR l/min	PEFR l/min	GRADE
11	Sulochana 44/F	157	M,E	M,E	Mi	292	312	Mi
12	Banumathi 48/F	153	M,E,S	M,E,S	Mo	251	307	Mi
13	Shanthi 52/F	144	M,E	M,E	Mi	280	302	Mi
14	Lakshmi 53/F	153	M,E	M,E	Mo	220	272	Mi
15	Yogeswari 55/F	162	M,E	M,E	Mo	204	277	Mi
16	Manimegalai 55/F	147	M,E,S	M,E,S	Mi	264	283	Mi
17	20/M Shankar	147	M,E	M,E	Mo	301	374	Mi
18	Senthil kumar 21/M	158	M,E	M,E	Mo	354	438	Mi

S.NO	NAME AGE/SEX	HEIGHT IN Cms	SINUSITIS EVALUATION		ASTHMA EVALUA-TION BEFORE MEDICATION		AFTER MEDICATION	
			DNE	CT	PEFR	GRADE	PEFR	GRADE
19	Suresh 22/M	136	M,E	M,E	332	Mo	391	Mi
20	Murugesan 24/M	152	M,E,S	M,E,S	385	Mi	413	Mi
21	Balakrishnan 25/M	160	M,E	M,E	387	Mo	427	Mi
22	Nihamathulla 26/M	166	M	M	320	Mo	447	Mi
23	Thiruvengadam 29/M	170	M,E	M,E	446	Mi	482	Mi
24	Kannan 29/M	153	M,E	M,E	335	Mo	425	Mi
25	Ramesh 31/M	148	M,E	M,E	312	Mo	399	Mi

SURGICALLY MANAGED PATIENTS

S.NO	NAME AGE/SEX	HEIGHT IN Cms	SINUSITIS EVALUA-TION		ASTHMA EVALUATION PRE- OPERATIVELY		POST OPERA-TIVELY	
			DNE	CT	PEFR	GRADE	PEFR	GRADE
1.	18/M Veeramani	152	M, CB,	M,CB	376	Mi	423	Mi
2	Chandrasekar 22/M	160	B/L SP	B/LSP	392	Mi	451	Mi
3	Raja 25/M	155	M,E,S	M,E,S	239	Se	320	Mo
4	Baskar25/M	148	M,E	M,E	275	Se	358	Mo
5	Mahesan 28/M	148	M,E, CB	M,E,CB	350	Mo	420	Mi
6	Arokya dhass 30/M	163	M,S,E	M,S,E	334	Mo	442	Mi
7	Durai pandiyan 33/M	168	M,E	M,E	275	Se	397	Mo
8	Palaniswamy 35/M	174	M,E,	M,E	331	Mo	396	Mi
9	Sachidanandham 41/M	161	M,E,PM	M,E,PM	256	Se	327	MO
10	Adaikalaraj 41/M	156	M,E	M,E	399	Mi	446	Mi
11	Muthulakshmi24/F	132	M,E	M,E	152	Se	190	Mo
12	Malathi 30/F	140	M,E	M,E	226	Mo	241	Mi
13	Jeyanthi 35/F	150	B/L SP	B/L SP	216	Mo	270	Mi
14	Jothi 42/F	154	M,E	M,E	295	Mi	323	Mi
15	Vetrikodi 46/F	147	M,E,S	M,E,S	244	Mo	288	Mi
16	Yogam 51/F	156	M,E	M,E	197	Mo	301	Mi
17	Pattammal 53/F	147	M,E	M,E	182	Se	284	Mi

CONTROL GROUP

S.NO	NAME AGE/SEX	HEIGHT IN Cms	SINUSITIS EVALUATION		ASTHMA EVALUATION AT START OF STUDY		AFTER SIX WEEKS	
			DNE	CT	PEFR	GRADE	PEFR	GRADE
1	Srikanth 21/M	163	M,E	M,E	330	Mo	330	Mo
2	Tamilarasan 25/M	158	M,E	M,E	398	Mi	388	Mi
3	Singaravelan 29/M	154	M,E	M,E	351	Mo	351	Mo
4	Manikandan 33/M	170	M,E	M,E	290	Se	290	Se
5	Subramani 33/M	156	M	M	365	Mo	365	Mo
6	Eswaran 35/M	148	M,E	M,E	307	Mo	298	Mo
7	Mariappan 44/M	151	M,E	M,E	397	Mi	374	Mi
8	Vetrikodi 31/F	152	M,E	M,E	214	Mo	214	Mo

KEY TO MASTER CHART

M- Maxillary sinusitis

E- Ethmoid sinusitis

S- Sphenoid sinusitis

CB- Concha Bullosa

SP- Sinonasal polyposis

Mi- Mild

Mo- Moderate

Se-severe