شبكة المعلومات الجامعية

# بسم الله الرحمن الرحيم


HOSSAM MAGHRABY

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# التوثيق الالكتروني والميكروفيلم

HOSSAM MAGHRABY

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# جامعة عين شمس

## التوثيق الإلكتروني والميكروفيلم

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علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

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# Sinoscopic Guided Correlation Between Microbiology of Chronic Rhinosinusitis and Its Related Chest Diseases in Adults

*Thesis*

Submitted for partial fulfillment of M.D
In Otorhinolaryngology Head and neck surgery

*By*

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(M.B.,B.Ch.- Master degree in ORL)

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**FACULTY OF MEDICINE**
**MENOUFIYA UNIVERSITY**
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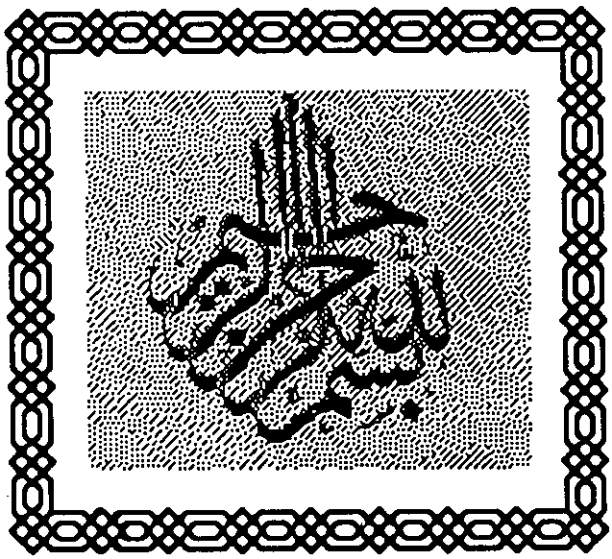
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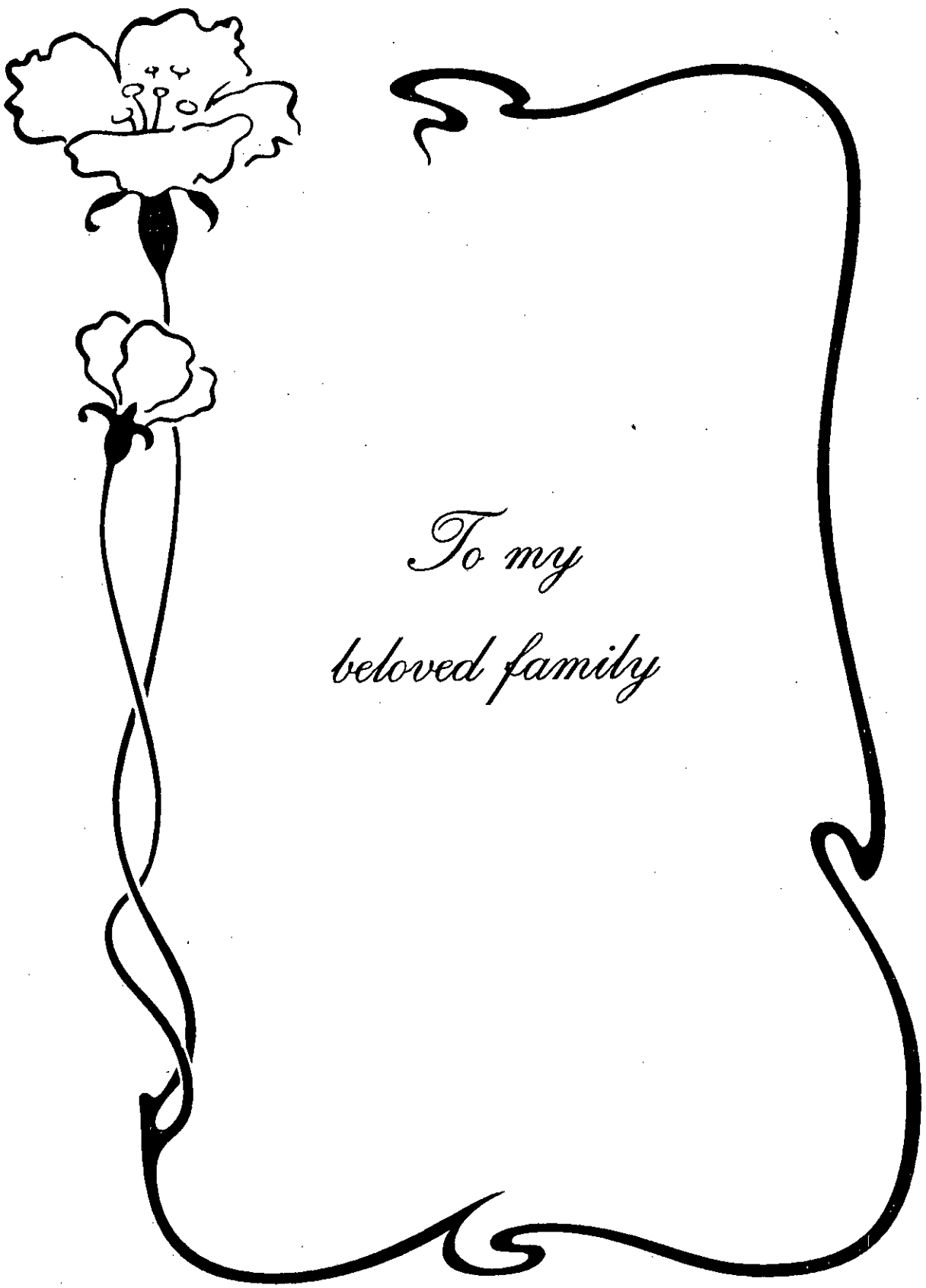

*To my*

*beloved family*

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# Abbreviations

AAR : Active anterior rhinomanometry.

ACC : Absolute cell count.

AD : Atopic dermatitis.

AFRS : Allergic fungal rhinosinusitis.

BAL : Bronchoalveolar lavage.

CRS : Chronic rhinosinusitis.

CDC : Cell differential count.

ECP : Eosinophilic cationic protein.

EDN : Eosinophil derived neurotoxin.

EP : Eosinophil peroxidase.

F : Female.

$FEF_{75}$% : Forced 75% expiratory flow.

FESS : Functional endoscopic sinus surgery.

$FEV_1$ : Forced expiratory volume at the 1st second.

FVC : Forced vital capacity.

GM-CSF: Granulocyte macrophage-colony stimulating factor.

GMS : Gomori –methanamine-silver.

HBPT : Histamine bronchial provocation test.

HBHR : Histamine bronchial hyperresponsivness.

H&E : Haematoxlyin and Eosin.

IL-3 : Interleukin 3.

IL-5 : Interleukin-5.

MBP : Major basic protein.

MML : Middle meatal lavage.

n : Number

NCL : Nasal cavity lavage.

NLA : Normal lower airways.

$PD_x$ : Provocative dose of bronchoconstrictor causing FEV1 to fall X%.

SAD : Small airway disease.

Rt : Right.

# *Contents*

# INTRODUCTION AND AIM OF THE WORK

# *Introduction and Aim of the work*

In 1996, the impact of chronic rhinosinusitis alone accounted for 5.8 billion dollars of all medical costs in the United States **(Ray 1999)** .In addition, it had a major impact on the quality of life **(Metson 2000).** When associated with lower airway diseases such as asthma, increased costs were expected, with more restriction of activities due to co-morbidity.

Since the time of Galen (130-200 AD), physicians have been aware of the coexistence of a syndrome including upper airway inflammatory disorders and associated lower airway involvement **(Campanella 2001).** This has been interpreted in two opposing ways: either the upper airways disease and the asthma are different and separate entities, with the former pathology being a potential risk factor for the later, or the upper airways disease and the asthma are an expression of the same disease, involving simultaneously upper and lower airways, probably because of a common aetiology **(Maesono 1999).**

Diagnosis of chronic rhinosinusitis (CRS) is supported by the characteristic symptoms and findings during the physical examination, and confirmed by radiological CT sinus involvement **(Lanza 1997).** Chronic rhinosinusitis patients can have associated lower airways problems. Some are obvious from the clinical picture of the patient and some are hidden in spite of lower airways inflammation **(Maesono 1999).**

Nasal secretions probably represent the first line of defence in which leukocytes play an important role **(Jankowski 1995).** It is also well known that secretions from most of the paranasal sinuses (the frontal, maxillary, and anterior ethmoidal complexes) drain into the middle meatus. Therefore, an inflammation of the sinus mucosa will quantitatively and qualitatively modify the leukocytes ratios in the mucus that drains towards the middle meatus. These inflammatory changes may influence the remaining lower respiratory system and induce related inflammatory changes in the bronchoalveolar lavage (BAL) **(Jankowski 1995).**

The cause of these cellular changes may be related to microbiological relationship. Although the role of viruses and bacteria in the cause of acute infection in sinus diseases are well established the role of bacterial infection in CRS and its related lower airways involvement are less well defined **(Kaliner 1997) .** In CRS while surgical intervention may be needed in a small portion of cases the current mainstay of therapy is medical predominantly involving antibiotics directed towards the causative bacteria. Most cases respond to empirical therapy. However when the medical therapy fails it is important to identify the causative bacterial pathogens **(Brook 2001).**

Fungi can be pathogenic to the sinonasal passages via several different mechanisms and can cause diseases ranging from such benign entities as allergic rhinitis to fungal antigens, aspergillomas, allergic fungal sinusitis, chronic indolent sinusitis and invasive or fulminant

disease **(Corey 1992).** The immune status of the host, the environmental load or mass of the fungi present, and local structural conditions of the sinuses or other tissues causing tissue hypoxia are all factors that predispose toward the development of a particular sinus disease **(Houser 2000).**

The relation between the fungus spores and paranasal sinuses in allergic fungal rhinosinusitis (AFRS) is not clear yet. Type 1 and Type III hypersensitivity are one form of this interaction, which was described as allergic fungal rhinosinusitis **(Manning 1998).** Another dominant role is eosinophylic interaction **Kuhn (2000),** which was described by **Ponikau (1999)** as eosinophyilic fungal rhinosinusitis. Another manner of interaction was shown in experimental studies and also in allergic bronchopulmonary aspergillosis that fungal spores impair mucosal defence not only because immunogenic interaction but because they are capable to alter the host immune response through macrophages and T cell suppression **(Knusten 1998).** One form was determined in experimental study with the interaction of fungal proteases present in fungal extracts with epithelial cells leading to morphologic changes and induction of proinflammatory cytokines **(Kaufman 2000).**

Recently multiple controversies raised concerning the relation of fungi to chronic rhinosinusitis and its related lower airways problems and the possibility of using local antifungal drugs.

In CRS endoscopicaly guided sinonasal culture hold a promise as a viable alternative to maxillary sinus aspiration **(Kaliner 2000).** The bronchoalveolar lavage (BAL) is the window of the lungs. It provides direct and safe sampling that can be compared to endobronchial or transbronchial biopsies **(Reynolds 2000).**

Therefore **the aim** of this prospective study was to identify in CRS the individual bacterial species of the middle meatus and the BAL fluid. Furthermore we correlated the eventual positive middle meatal bacterial cultures and the associated inflammatory changes in different subgroups of lower airway involvement.

To identify in CRS patients the different cultured fungi in various sites of the nasal cavity and lower airways and to correlate these cultivated fungi with the cellular inflammatory changes. Also to study the existence of fungi histopathologically in sinus tissue specimens.

To study the cytology of the middle meatal lavage (MML) in CRS patients and compare it with the potential inflammatory changes found in the BAL of the same patients. Furthermore, these cellular changes were correlated with spirometric lower airways functional changes.

To identify the surgical scores of FESS operation in different lower airway involvement subgroups.