PATIENTS' DEMOGRAPHIC DATA IN DIFFERENT TYPES OF FUNGAL SINUSITIS

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Submitted by
Mohammad Salah Mohammad Mahmoud
M.B.B.Ch.
Faculty of medicine, Ain Shams University

Supervised by

Prof. Dr. Badr Eldin Mostafa

Professor of otorhinolaryngology
Faculty of medicine, Ain Shams University

Dr. Mohamed Elsharnouby

Assistant Professor of otorhinolaryngology
Faculty of medicine, Ain Shams University

Dr. Hesham Abdel Aty Abdel kader Elsersy

Lecturer of otorhinolaryngology
Faculty of medicine, Ain Shams University

Faculty of medicine
Ain Shams University
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LIST OF ABBREVIATIONS

Abbreviation	Meaning
AC	Air Conditioner
AFIFS	Acute fulminant invasive fungal sinusitis
AFRS	Allergic fungal rhinosinusitis
AUC	Area under the curve
CI	Confidence interval
CIFS	Chronic invasive fungal rhinosinusitis
CRS	Chronic rhinosinusitis
CT	Computed tomography
FRS	Fungal rhinosinusitis
G-CSF	Granulocyte colony-stimulating factor
GIFS	Granulomatous invasive fungal rhinosinusitis
GMS	Grocott methanamine silver
IFS	Invasive fungal sinusitis
IgE	Immunoglobulin E
IU	International unit
MRI	Magnetic resonance imaging
N	Number
PCR	Polymerase chain reaction
ROC	Receiver-operating characteristic
SE	Standard error
SFB	Sinus fungus ball
US	United States

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Introduction

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Acute or chronic rhinosinusitis is estimated to occur in 20% of the population at some time in their lives (Howarth and Holmberg, 1995). Acute sinusitis is often associated with an upper respiratory tract viral infection or, less frequently, because of bacterial infection. It is usually either self-limited or well-controlled with supportive treatment, and surgery is usually not needed. Chronic sinusitis usually follows a protracted course, which may require surgery to control the disease process and to provide tissue for an accurate diagnosis. Although chronic sinusitis may be caused by many conditions, a major one is fungal infection (Vennewald et al., 1999).

It is important to classify fungal rhinosinusitis to accurately predict prognosis and optimized effective therapy. Fungal disease of the nose and paranasal sinuses can be classified based on the clinical, radiologic, and histologic manifestations of the host-pathogen relationship (Ferguson, 2000).

The most commonly accepted classification system divides fungal rhinosinusitis into invasive and noninvasive diseases based on histopathologic evidence of fungal elements penetrating host tissue. These may be further subdivided into five distinct entities along the immunologic spectrum. Saprophytic fungal infestation (SFI), sinus fungus ball (SFB) and allergic fungal rhinosinusitis (AFRS) are fungal disease manifestations in the absence of fungal invasion of host tissue.

Acute invasive fungal rhinosinusitis which is referred to as acute fulminant invasive fungal sinusitis (AFIFS), chronic invasive fungal rhinosinusitis which is referred to as chronic invasive fungal sinusitis (CIFS) and granulomatous invasive fungal rhinosinusitis which is referred to as granulomatous invasive fungal sinusitis (GIFS) exhibit histopathologic evidence of hyphal forms within sinus mucosa, submucosa, blood vessels or bone but are distinguished by yet other histopathologic features, time course, clinical and host immunocompetence (deShazo et al, 1997).

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This study was conducted to correlate between demographic data of the patients and fungal sinusitis.

Review of literature

Allergic fungal rhinosinusitis

Definition:

Allergic fungal rhinosinusitis (AFRS) is a disease of the sinonasal complex affecting mainly the young, immunocompetent adults who complain of chronic rhinitis and/or recurrent nasal polyposis despite medical and/or surgical treatment (Corradini et al., 2003).

Differences in the criteria used by several researchers to define AFRS explain the wide variation in the reported incidence of allergic fungal disease as well as differences in opinion regarding its treatment. AFRS can be defined as a chronic condition characterized by:

1. Presence of nasal polyps.

2. Hypersensitivity indicated by atopic history, skin test or serological testing.

3. Characteristic CT scan features.

4. Histologic features of allergic mucin.

5. Non invasive fungal hyphae as evidenced by histological examination or culture (Dhiwakar et al., 2003).

Epidemiology:

The incidence of allergic fungal sinusitis in cases of chronic rhinosinusitis treated surgically has been approximately 6 to 7%. The incidence of aspirin sensitivity has been demonstrated to be 27% in patients with allergic fungal sinusitis. Asthma has been associated in 65% of the patients. Incidence of allergic fungal sinusitis is high in temperate regions with relatively high humidity (Cody et al., 1994). AFRS is more widely recognized at present because of changes in fungal taxonomy and improved culture techniques (Novey, 1998).

AFRS is noted more commonly in a younger age group; with a mean age at diagnosis of 21.9 years (Thorp et al., 2012). The disease seems to be more common in warmer, humid climates (Manning and Holman, 1998). Some studies have noted fairly equal sex predilection for AFRS, where as Manning and Holman noted a male predominance of 1.6 males per females, Patients with AFRS are by definition atopic, but they have an otherwise normal immune system (Schubert and Goetz, 1998).

Pathophysiology:

AFRS is initiated when atopic individual is exposed to inhaled fungi. It is estimated that an active man inhales approximately $5.7x10^7$ spores of various species within a 24 hours period (Novey, 1998). The fungi deposit within a sinus cavity and an escalating immunologic reaction, Gell and Coombs type I (and possibly type III), takes place to the non invading organism. Mucosal edema, stasis of secretion and inflammatory exudates all combine to obstruct the sinus ostium. This process then may expand to involve adjacent sinus and

may produce sinus expansion and bony rarefaction. Secondary bacterial infection may occur, simulating an acute exacerbation of underlying chronic sinus disease (Marple and Mabry, 1998).

The Aspergillus organism itself is believed to impair mucosal defenses. Not only are Aspergillus antigens immunogenic, they are also capable of altering host immune response through macrophage and T cell suppression specifically. The alteration of mucosal defenses includes:

1. Reduced ciliary beat frequency.

2. Impaired function of host fungicidal proteins within the mucus blanket.

3. Fungus antigens are able to inactivate the complement system.

4. Aspergillus interferes with phagocytosis and intra cellular killing.

5. Aspergillus release proteolytic enzymes with elastolytic and collagenolytic activity which can disrupt the host basement membrane (Kauffman and Tomee, 1998).

Clinical presentation:

The diagnosis of AFRS should be suspected in any patient with CRS refractory to medical management, especially anyone who has a history of recurrent nasal polyposis and multiple prior surgeries. Up to 75% of those patients have asthma. AFRS itself has spectrum of disease which range from mild allergic symptoms, polyps and scanty allergic mucin with few scattered hyphae to an extreme atopic state