MALIGNANT EXTERNAL OTITIS

ESSAY

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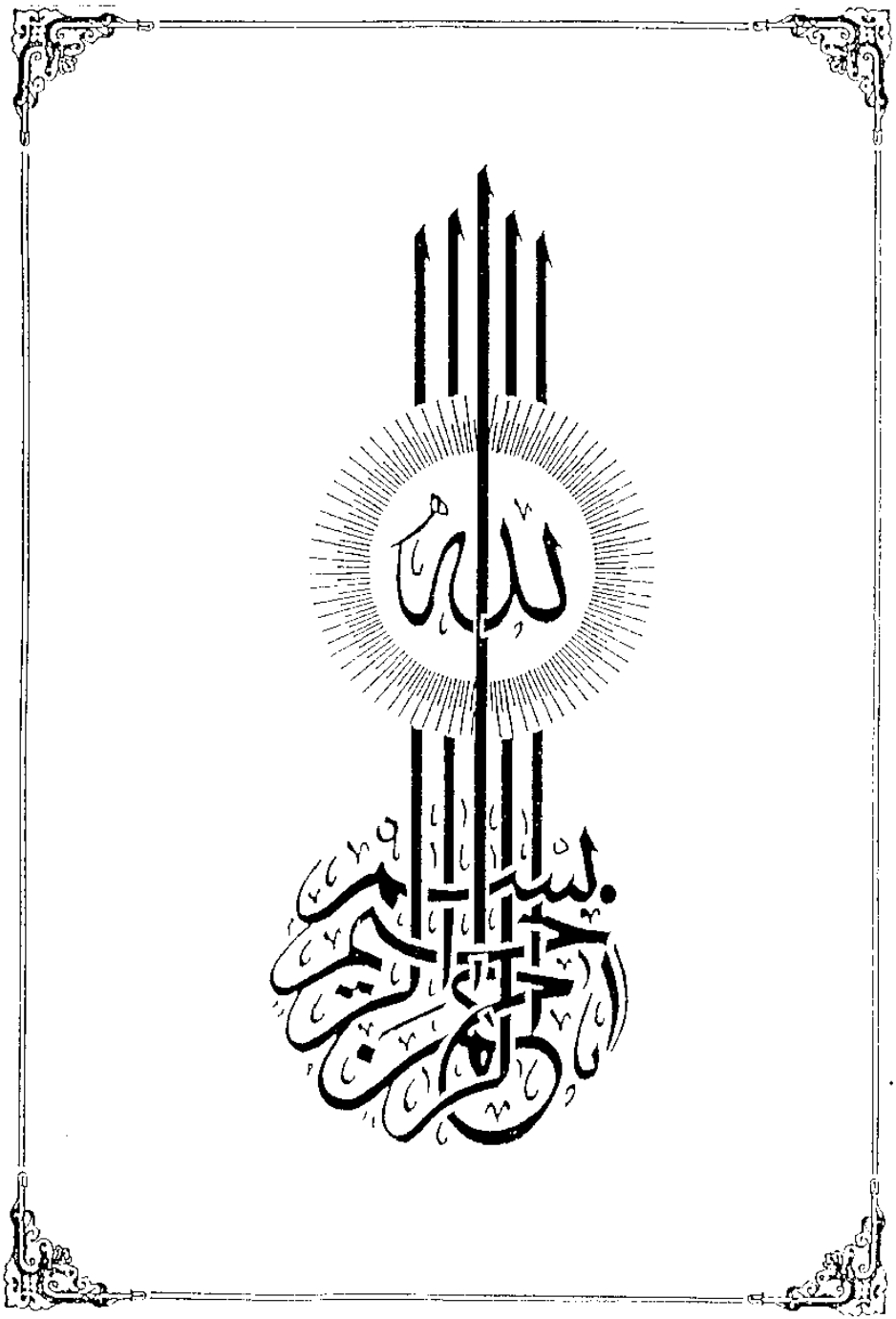

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C O N T E N T S

INTRODUCTION

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Mlignant external otitis is a term coined by Chandler (1968) to describe a particularly severe form of external otitis which primarily affects elderly diabetics. The causative organism in the disease is uniformly the bacterium psueodomonas aeruginosa. Chandler described the disease as malignant because of its resistance to treatment, the destruction of the temporal bone and the base of the skull characteristic of the disease, the multiple cranial nerve palsies associated with this destruction, and the overall poor prognosis for individuals with this disease.

Evans and Richards (1973) suggested that the term "Necrotizing external otitis" rather than "Malignant external otitis" would be more appropriate since the word necrotizing describes more precisely the pathology, and patients will not be mislead by the word "malignant" because it is suggestive of a neoplastic aetiology.

Subsquently, many other authors have described their clinical experiences in treating patients with this disease. Inspite of the voluminous literature dealing with this entity, yet the chronological sequence of complications during the course of the disease remains still a challenging problem for the otologist.

AIM OF THE ESSAY

The objective of the present essay was to revise the literature dealing with malignant external otitis with the purpose of better awareness of the nature of the disease, earlier identification of cases and their ideal management.

PATHOGENESIS

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Certain factors are involved in the pathogenesis of the disease, these are:

Diabetes Mellitus:

It seems that diabetes mellitus is an extremely important predisposing factor for the development of malignant external otitis. Its role in the pathogenesis of the disease is most probably due to weakening of the natural defence mechanism and local tissue resistance as well as its vascular effects, chiefly, atherosclerosis and microangiopathy.

Evans and Richards (1973) suggested that the changes in the ears and temporal bones which take place in malignant external otitis constitute the otological equivalent of the diabetic gangarene which most commonly occurs in the lower limbs.

In his report in 1977, Chandler stated that he had been unable to confirm the suggestion that the extent of malignant external otitis was related to the severity of diabetes mellitus.

Few cases, however, were recorded in non-diabetic patients. Chandler in his first report (1968) recorded

a case of a non-diabetic woman who suffered the disease in its full blown picture. Schwarz et al. (1971), presented another case of malignant external otitis in a non-diabetic elderly man. Diabetes mellitus was not found in the case described by shanon, et al., in 1972. John and Hopkin (1978) described a case of a non-diabetic patient who survived bilateral necrotizing otitis externa with multiple cranial nerve palsies. Another case of an elderly non-diabetic patient suffering from malignant otitis externa was reported by soliman (1978). Joachims (1976) and Cóser, et al. (1980) reported the disease in non-diabetic, undernourished children and infants with poor general health.

- Age:

Malignant external otitis is common in old age. Older people with narrowed arteriosclerotic blood vessels and poor tissue nutrition are subject to the same soft tissue infections as those of the diabetics. However, cases of malignant external otitis were reported in infants and young children. Joachims (1976) has described two cases, one of them was a 2-year old child and the other was a 7 month-old infant. Both cases were non diabetic, but in poor general condition. Another infant aged 2 months was briefly reported in Chandler's review in 1977. Cóser et al. (1980) reported also two cases of

the disease in non-diabetic, undernourished infants. One of them was 5-month-old and the other was 6 months of age. Merrit, et al. (1980) encountered a 15-year-old adolescent with diabetes mellitus and malignant external otitis.

- Pseudomonas Aeruginosa :

Pseudomonas aeruginosa is the causative organism in the majority of cases. It is a slender, rod shaped, flagellated, motile, gram-negative bacillus, measuring 1.5 to 3.5 microns in length and 0.8 microns in width. It is an aerobic, non-sporing and non-capsulate organism. It ferments only glucose and produces water soluble green pigments, exotoxins and endotoxins. The optimum temperature for its growth is 37°C. It is ubiquitous organism, having been cultured from water, soil, sewage, humidifiers, faucets, sponges, sinks, anaesthetic equipments, skin and upper respiratory tract. It is also an intestinal commensal in man. The organism is resistant to most known antibacterial drugs. Chandler (1968 and 1977) as well as Shanon, et al. (1972), believe that the organism is not normally found in the external auditory canal and that it is of low pathogenecity. It has been thought to be a troublesome secondary invader of a staphylococcal infection which, under suitable host conditions, may result in an

extremely resistant invasive type of infection. However, Morgenstein and Seung (1971) and Scott-Brown (1979) are of the opinion that Pseudomonas aeruginosa is a normal commensal in the external ear. The former authors, from recent clinical experience, concluded that the organism was capable of great virulence producing fulminating acute process as a primary patogen.

Recently, anaerobic organisms, probably of the bacteroids group, have been incriminated as the primary causative agent (Maran and Stell, 1979; Khout and Lindsay, 1979).

The above mentioned characteristics of Pseudomonas aeruginosa in combination with old age and basic pathological changes of diabetes, may account for the pathogenesis and relentless course of what is usually a benign and self limited disease in other individuals.

The infection starts in the external auditory canal, especially at the floor. Ordinarily it does not gain access to tissues outside the canal, but under certain conditions known for decreased vitality e.g. diabetes mellitus or other debilitating diseases such as granulocytopenia and leukaemia,the infection results in a cellulitis or furuncle. As deeper structures become involved, it has the capacity for "selective

vasculitis" with invasion of arteriolar, capillary and venule walls with or without haemorrhage accompanied by thrombosis with focal coagulation necrosis of surrounding tissues. The necrosis may lack the usual inflammatory cell response. The mechanism of invasion of the ear canal appears to be due to local bone necrosis, as in osteomyelitis of other location. This inturn extends to the submucosal vasculature of the pneumatic spaces establishing one or several sites of bone destruction. The histologic evidence suggests that the routes to the sites of active destruction are mainly determined by the pneumatic pathways of the temporal bone.

The trabecular bone in malignant external otitis is not particularly involved in the osteolytic process. On the other hand, once the nidus of osteolytic activity is established all bone, particularly compact bone is on a broad front destroyed. It seems that the compact bone in malignant external otitis is more susceptible than the bone of the pneumatic spaces.

The recent above mentioned histopathological findings of the temporal bone of a case of malignant external otitis, as described by Khout and Lindsay in 1979, differ from those identified by Meltzer and Keleman in 1959.

In the latter authors' specimen, there was extensive involvement of the diploeic bone, trabecular destruction was prominent and the pneumatic spaces were filled with pus or in other areas obliterated.

Starting in the external auditory canal, as previously mentioned, the process extends inferiorly into the soft tissue at the base of skull at the junction of the cartilaginous and osseous portions of the external auditory canal or through the fissures of Santorini (usually present along the anterior cartilaginous wall of the external auditory meatus). Becoming a soft tissue infection, the disease involves the parotid gland, cartilage, bone, nerves and blood vessels (Fig. 1).

The infection may and frequently does extend into the bony canal wall of the external auditory canal and through the mastoid cortex or its tip. Through this route or through the fallopian canal, secondary mastoiditis develops. In other words, necrosis and sequestration of bone permit the direct extension of the infection into the pneumatized air cells of the mastoid process.

Recently, some interesting findings concerning the site of the lesion were made. Cohn (1974) observed that the pseudomonas infection might take a similar rapidly