INTRODUCTION

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Atrophic rhinitis is a chronic nasal disease characterised by progressive atrophy of the mucosa and underlying bone of the turbinates and the presence of a viscid secretion which rapidly dries and forms thick greenish yellow crusts which emit a characteristic foul odour sometimes called ozaena (a stench) (Henriksen and Gundersen, 1956 and Chatterji, 1980). There is an abnormal patency of the nasal passages.

It was first described by Frankel in 1876 (El-Shennawy, 1969) as a triad of foetor, nasal atrophy, and crust formation. The disease is described under several terms namely ozaena, atrophic catarrh, coryza foetide, sclerotic rhinitis and stink nose (El-Shennawy, 1969).

It is a widespread and intractable disease, constituting 0.3-7.8% of otonasolaryngopharyngeol affections in China (Chen Han Sen, 1982).

Ruskin (1932) tried to differentiate atrophic rhinitis into two types primary atrophic rhinitis (ozaena), and secondary atrophic rhinitis. He considered the former

to be a primary disease of the nasal mucosa of unknown aetiology, whereas the later is a disease secondary to chronic nasal or sinus infection.

Atrophic rhinitis has been stated to be a disease prevalent among the poor or malnourished individuals. Hence, it is found less and less frequently in more developed countries while it is common in developing ones. It is not uncommon in Egypt (Girgis, 1966).

The exact aetiology of the disease is not yet well established, some attributed it to hereditary (Girgis, 1966 and Batton and Sibert, 1980), poor nutrition (Bernat, 1965), chronic infection in maxillary sinuses in childhood (Bosworth, 1889; Grunwald, 1902 and Adam, 1934), endocrinal factors (Harrison,1957) or developmental factors (Wachsberger, 1934 and Pesti, 1949).

Many organisms have been isolated from nasal cavity in cases of primary atrophic rhinitis, namely klebsiella ozaenae, corynebacterium diphtheriae, staphylococci, pneumococci and bacillus proteus (Rosen et al., 1953 and Barkve and Djupesland, 1968).

Histological examination of the nasal mucosa shows metaplasia of the pseudo-stratified columnar ciliated epithelium to squmous epithelium, atrophy of the entire mucosa, and degeneration of the goblet cells and the glands, and that there are dilated capillaries.

The condition of the maxillary sinus lining mucosa in cases of primary atrophic rhinitis is not well clear. Bosworth (1889) (Quoted from El-Barbary, 1970) and Grunwald (1902) (Quoted from Adam, 1934) reported that the maxillary sinus infection is an associated condition with primary atrophic rhinitis.

Ssali (1973) showed that the walls of the maxillary sinus were thick and made antral washout difficult in cases of primary atrophic rhinitis.

Girgis (1966) reported that the mucous membrane lining the maxillary sinus shares in the atrophic changes and loses its cilia, mucous cells but there are no scales accumulated on its surface.

Many therapeutic measures have been tried for atrophic rhinitis, but none of them has proved to be wholly

successful. As the exact aetiology is not yet clear, the proper line of treatment is not certain. Owing to the failure of medical treatment to accomplish a gratifying result, many surgeons have thought of different procedures to overcome this disease.

Several different techniques have been devised, ranging from total occlusion of the anterior nares, to more conservative implantation procedures using foreign (Silastic, teflon, silicone) or autogenous (dermofat, bone, or cartilage) substances (Shehata and Dogheim, 1986).