REVIEW
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A century is passing by since FRANKAEL in 1876 described this chronic distressing condition, incurable yet not fatal. Inspite of tireless efforts by many, the code of its aetiology still remains undeciphered. Many theories and hypothesis are put forth to explain this condition but have failed to catch general acceptance.

Anatomical Considerations

The area under consideration “Nasal Cavity and Paranasal Sinuses” has a complex configuration. Anatomy of paranasal sinuses has been known for several centuries.

Rigmure (1651) defined the maxillary and frontal sinuses. Gross anatomical relation’s of the paranasal sinuses are shown in figs. A, B, C and D (Friedman and seborn, 1982).

Normal Histology

The anterior part of vestibule is lined by Keratinizing squamous epithelium which is a continuation of the skin covering the external nose. The lining on being traced backward becomes pseudostratified, ciliated columnar type which is characteristic of respiratory epithelium. This lining is called Schneiderian membrane after the name of the histologist who was the first described it in 1660. Interspersed amongst the ciliated cells are varying number of goblet cells which are identified as unstained areas under the light microscope.
Numerous mucosal gland of mixed nature are found deep in the lamina propria. The ductular system is not well developed and some times may be difficult to identify on the periphery of these glands are located the myoepithelial cells. The importance of these cells is in the pathogenesis of certain mucosal gland tumours.

Paranasal mucosa is similar to that of the nasal fossa, though there are slight differences, the columnar cells tend to be shorter resulting in thinner epithelium. These cells bear cilia and there are large number of microvilli. The lamina propria also tends to be thinner and usually bears fewer mucosal glands (friedmann and Seborn, 1976).

**NASAL ERECTILE TISSUE**

An interesting feature of the lamina propria of the nasal and occasionally paranasal sinuses mucosa is the presence of complex blood vessels which are designated as “erectile tissue”. The vessels contain substantial amount of smooth muscle fibres which are arranged in spiral fashion, thus giving rise to irregular arrangement an cross section. These muscle fibres are under the influence of the autonomic nervous system and also react to chemical reagents, including some hormones. Another interesting aspect of the nasal cavity is the existance of melanocyte’s which have been demonstrated in the skin of the vestibule (Szabo, 1959) and the epithelial lining of the nasal cavity (AZK and Lawson, 1974).
OLFACTORY MUCOSA

The human olfactory area extends from the roof of the nasal cavity about one centimeter downwards on either side of the nasal septum and on the medial surface of superior turbinate. This area is covered by pseudo stratified epithelium. The olfactory cells are essential bipolar receptor neurones and are provided with distal and proximal processes. The distal process pass through supporting cells towards the surface and are called dendrite. The proximal process is essentially an axon. They form synapse connections with neurone of second order in the olfactory bulb (Lenz, 1977).

INCIDENCE

Atrophic Rhinitis is included as one of the disappearing diseases in the Western countries. Western authors attribute this change to their improvement in living standards. In our country, the disease is still prevalent and Jain (1966) reported a frequency of one case of atrophic rhinitis for an average of 1700 new out patients in Kakinada. He is of the view that the climate, poor hygienic condition and persistance of infections diseases like syphilis and leprosy are all major contributing factors in keeping the disease alive in this country. The disease is more prevalent in yellow races, occurs in Negroes of South, Central and North America, but is rare in Negroes of Africa Oceania and West Indies (St. Clair Thompson).
In 1917, Roy (quoted by Shapiro 1967) published the results of his observation on the incidence of atrophic rhinitis among the various races. He examined the nasal cavities of 5000 negroes in Africa and did not find a single case of atrophic rhinitis. On the other hand Negroes and other people of mixed blood in Brazil as well as in other countries of South and North America Eskimoes, Malayes, Filliphines and North American Indians showed a considerable incidence of this condition.

Girgis (1966) recorded an incidence of 117 cases of atrophic rhinitis in 11365 out patients in United Arab Republic. He observed that the disease was more common in poor citizens.

**AETIOLOGY**

Atrophic Rhinitis is regarded as a disease of the young subjects, peak of incidence between the ages of 10-12 years. The disease shows a prediliction to the females than males. Females are affected 5 times more frequently than males (James, 1963).

Atrophic Rhinitis has been divided into two groups on the basis of the causative factors (James – 1965)

(I) Primary atrophic rhinitis

(II) Secondary atrophic rhinitis
Primary atrophic rhinitis includes those cases, where the exact cause is unknown. It is quite possible to consider multiple factors to produce this condition.

Secondary atrophic rhinitis consists of the condition resulting from syphilis, Tuberculosis, lupus vulgaris, leprosy and scleroma or operative destruction of the nasal mucous membrane.

The various views about the probable causes of Primary atrophic rhinitis may be discussed on the following headings: (Girgis 1966, Shapiro 1967)

(i) *Sinus Infections*:
Grunwald, Lautenschlager, Harry L. Pollack were of the opinion that Atrophic rhinitis is always secondary to sinus infection. In chronic sinus infection, the secretions act as an irritant to the connective tissue which proliferates, later resulting in cicatrization, fibrosis and atrophy (Girgis 1967, Ballenger 1969).

James (1965) attributed the cause to a proceedings severe nasal inflammation with associated vitamin nutritional and endocrinal deficiency. Nasal Diptheria and suppurative rhinitis and sinusits of Measles and other exanthemata are considered to be the common anticidents. Those severe infections have been shown to cause necrosis of the glandular and ciliated epithelium and subsequent repair by fibrosis and metaplasia.
(ii) **Hypertrophic Rhinitis**

Bosworth regarded Ozaena as secondary to hypertrophic rhinitis, Lautenschlager, Eggston and wolff considered that all cases of Atrophic rhinitis were due to chronic infection, which induced periarteritis and endarteritis of the nasal mucosa producing atrophic changes.

(iii) **Bacterial Infection**

This remains probably the oldest theory in literature. Many organisms have been isolated and accused of responsible for this condition. Pseudo-Diphtheria bacillus (Belforiti and Dellavedova), coccobacillus foetidis Ozaena (Perez), Bacillus mucosus (Abel) Bacillus foetidis Ozaena (Hazek) and other organisms. The infectivity and pathogenecity of these organism could not be proved. They are possibly secondary invaders and may be responsible for the foetor.

(iv) **Developmental Theory**

Excessive patency of the nasal cavities in relationship to the skull has been put forward by Hopmann, Siebenmann, Garbar and J. Wright. Peste (1949) demonstrated that the large nasal fossa in atrophic rhinitis are at the expense of small under developed antra. The author concluded that the poor pneumatisation of antra is probably the decisive factor in the pathogenesis of the disease.
Stanur showed that the length of nose measured between the anterior and posterior nasal spine is shorter in people suffering from ozaena than in normal persons. He attributes it to the stand still of the development of facial skeleton.

Wachsberger (1934) stressed the importance of abnormal width of the nasal cavities, which increased the evaporation of the already viscous secretions of the ozaena resulting in crust formation.

Shapiro (1967) believes that the abnormal width of the nasal fossa is more likely to be in the nature of an effect than the cause. Supporting this is the fact that the condition is very rare in African Negroes, who have wide short nostrils and much common in Asiatic who have relatively narrow nostrils.

(v) **Hereditary Theory**

Fleischmann believed that it is due to hereditary inhibition of the nasal mucosa, which can run according to Mendelian law. Atrophic rhinitis among the same member’s of the family is reported in the literature.

(vi) **Endocrine Theory**

The occurrence of the disease mainly during puberty, is predilection to the female sex and its worsening during menstruation and pregnancy suggest its relationship to endocrine factors (Lautenschlager 1924, Pratt 1931) Mortimer and his associates by experimental work
postulated a decreased secretion of gonadotrophic hormones at puberty and also a lack of response of mucous membrane to normal oestrogen circulating in the blood. Many recent authors like James (1965) and Ballenger (1969) do not consider this factor in the aetiology of atrophic rhinitis. The alleged role of endocrine dysfunction has fallen into disrepute from lack of evidence (Bernet, 1965).

(vii) **Deficiency Theory**

The disease is more prevalent among the poor class of people and refugee camps. The deficiency of mainly attributed to iron and fat soluble vitamins especially vitamin A (Girgis 1966).

Bernat 91968) observed that iron therapy was successful in atrophic rhinitis, if the nasal mucosa is not irreparably damaged. He concludes that iron deficiency has a definite role to play and improving the diet can reduce the incidence. Gadre et al (1971) observed mild anaemia in their cases. However, Barkve (1968) was unable to find any incidence of iron deficiency in his studies.

Zinc deficiency may manifest as reduced smell and taste sensations (Condas et al, 1977, Boyette, 1982). It is indispensable to cellular function and division (Boyette, 1982) and is essential for the activity of serum alkaline phosphates. Because of this, low levels of alkaline phosphatase can be expected in association with hypozincimia
(Prasad et al, 1978 & 1979, Roth & Kirschgnesner, 1974) and therefore alkaline phosphatase activity may be used an index of clinical zinc deficiency and a monitor of therapy (Kasarkis & Schuna, 1980).

Hollender (1944) believe that trauma, during effect of inspired air and reduction in the blood supply are the principle factors. Turner (1968) also supports the theory that reduction in the blood supply are the principle factors. Turner (1968) supports the theory that reduction in blood supply to the nasal mucosa is the determining factors in atrophic rhinitis.

Jackabfi (1954) considers autonomic dysfunction to be of primary importance for producing atrophic changes, creating suitable conditions for the colonisation of the mucosa by specific capsulated bacteria which than become pathogenic.

All said it seems that the disease is due to more than one factor most probably due to some hereditary or endocrine factors and that ozaena is caused by secondary invasion by saprophytic organism (Girgis 1966).

**PATHOLOGY**

Atrophic conditions seems to be generalised than localised to the nasal mucosa. Pharynx, larynx, Trachea and Bronchi are involved
frequently and atrophic vaginitis is also reported. Girgis (1966) considers atrophic rhinitis related to the syndromes like plummer vinson and scleroderma, fundamentally characterised by dystrophic conditions of mucosal tissue of ectodermal and mesodermal origin.

A good account of the pathology is given by Eggston and Wolf (1947), Taylor and Young (1961).

In the early cases the histopathological picture is that of chronic non specific inflammation. The pathological changes are found in all the elements of the nasal mucosa, namely the epithelium, basement membrane, Tunica propria and even in the underlying bone. The columnar ciliated epithelium undergoes metaplasia to squamous epithelium. All stages can be founds the cells loose their cilia, begin to flatten, goblet cells become fewer and finally a mass of keratin may be seen over the surface.

While Taylor and young (1961) have found the basement membrane when present to be thin, others like Shambaugh (1931) Hollender (1944) have noted thickening of this membrane with more collagen. It is due to an inflammatory process which produces endarteritis and periarteritis of terminal arterioles (Ruskin, 1942, Taylor and Young 1961 and Holopainen, 1967). Many authors have stressed the presence of endarteritis obliterans but German investigators deny this.
Characteristics changes are the dilatation of capillaries. The endothelial cells have more cytoplasm and shows a more positive reaction for alkaline phosphate (Taylor and Young 1961). High concentration of Alkaline phosphatase explains the absorption of bony nasal turbinates.

Sinus show definite diminution of size and this is due to arrest of development as the disease starts in young age. The mucosa of sinus shares in the atrophic changes but no crusting occurs. Bony wall of maxillary antrum shows evidence of sclerosis (Girgis 1966).

**CLINICAL MANIFESTATION**

Clinical features of atrophic rhinitis are typical and are well described by James (1965) Logan Turner (1965), Ballenger (1969).

The most characteristic feature is the cadaveric smell or stench from which the disease derives its name, Ozaena.

In women the foetor becomes more during menstruation and pregnancy. Anosmia of varying degrees are present, while Epistaxis, headache and sense of dryness of mouth are other common complaints. Nasal obstruction is due to accumulation of crusts but even without this the symptom may be complained due to inhibition of sense of passage of air (Girgis 1966).
Externally the nose of person having atrophic rhinitis shows presence of thickened vestibular rim and button like tip (Prond 1947). Depression of nasal bridge due to arrest of development and oblique furrows on either side of nose at the junction of bone and cartilagenous wall due to shrinkage and underdevelopment of the cartilage are usually found (Girgis 1966). Patient may be in drawn and shows evidence of Psychological upset.

Nasal cavities in early stages appear roomy, the mucosa pale and covered by viscid, greenish secretion. Atrophic changes begin’s in the inferior turbinate and middle turbinate. Later the whole lateral wall becomes flattened by atrophy. Nasal cavities get wider and viscid secretion dry up producing greenish or black crust on the mucosa.

Pharynx usually shows atrophic changes and its posterior wall appear dry and glazed. Hoarseness of voice shows laryngeal involvement and indirect laryngoscopy reveals crusting in the inter-arytenoid and subglottic region. In the trachea and bronchi also scales may accumulate and in some cases obstruction persists even after tracheostomy and proves fatal (Girgis 1966).

In atrophic rhinitis the nose is affected Bilaterally but in the presence of gross septal deviation the condition may be absent in the narrow fossa. (Thomas and Negus 1955).

S.C. Gupta considered that atrophic changes are seen on the concave side and if deformity is gross, obstructive symptoms occurs on the convex
side of the septum. An excessively large air space in the nasal cavity is believed to be are the causative factors in atrophic rhinitis (1985).

**TREATMENT**

The various methods of treatment fall into two groups: Medical and Surgical and it can be confidently said the latter has completely eclipsed the former because of the comparatively lasting and definite result.

Medical treatment presents never ending list and are mainly in the form of proper nasal hygiene, alkaline nasal douches, glucose glycerine paste and oily drops.

Oestrogenic substance was tried based as theory of oestrogen deficiency as a cause of the disease (Blaisdell 1938), Streptomycin was used both systemically and as solution locally by Moselella (1950) with disappearance of crusting and smell. Sen used Nicotinic acid, the peripheral vasodilator with encouraging results with the same idea of nasal vasodilatation. Arnulf did some stellate ganglion block with good but temporary results. This was supported by the work of Sharma and Sardana (1965).

At present the attention is mainly concentrated on surgical treatments and constant efforts are provided to come out with a perfect procedure. The basis of the surgery has sprung from the observation of Sanger and
Sounderman (1894) that most nasal cavities resulted from using meatal obturator for the nostrils.

Most of surgical methods are aimed to reducing the nasal air way. The drying effect of inspired air is a major factor in causation or progression of the disease. Partial obliteration of nasal cavity breaks this viscious circle, set into action by the factors, which cause atrophy of the mucosa and thus widening of the cavity (Girgis 1966).

Review of the methods may be conveniently divided into

(i)  **Traumatic Procedures**

Lautenschlager (1917) was the first to describe the surgical technique to narrow the nasal cavity. By a caldwell Luc’s approach, he reached the nasoantral wall, separated it upto the posterior part by chisel and hammer and pushed the whole segment medially against the septum.

Lautenschlager’s procedure was further modified by Halle (1918) Wittmack (1919), Hensburg (1921) Wachsberger (1934) and Rethi (1948).

While Halle tried a simpler intra nasal approach for the mobilisation of lateral wall of the nose, Hens berg tried to keep the mobilised wall in the midline by suturing it to the septum with magnisium wires. But in most of
these cases the infactured segment tended to spring back to the original position.

Wittmack (1919) followed the lautenschlager technique and as an addition transplanted the stenson’s duct in side the maxillary antrum with the idea of moistening the nasal mucosa by the saliva. Though there was clinical improvement, some patients were left with a distressing salivary fistulae.

(ii) **Submucus Injections**

Beginning of this line of treatment was marked by Gersung (1900) who injected liquid paraffin submucosally into the nose.

Paraffin injection were also done by Banstein but sloughing was a common feature.

Chung et al (1964) introduced medical silastic S-5392 which when catalysed and injected submucosally became solid in a few minutes. The material is clear some what viscid fluid, which when mixed with catalyst became solid of rubbery consistency in several minutes. The time required for this depends on the temperature, humidity and the amount of catalyst used.
(iii)  \textit{Implantation Techniques}

Good range of work has been done on this line using various autogenous, homogenous and heterogenous materials from the beginning of this century. Harry L Pollock (1927) after having had disappointing results with Lautenschlager and Halle operations began displacement of septal flap towards the lateral wall of the nose with implantation of various autogenous material like fascia, lata abdominal fascia, bone and cartilages.

Kelmer (1931) and Rasnetz (1939) have tried Ivory implants with instantaneous relief but extrusion of implants was common.

In Cairo, Iskander H. Girgis (1966) has done laborious work on this problem. He first started using autogenous living tissue flaps as material for implantation. Through a sublabial incision a local periosteo-fascial flap is raised from the cheek with a pedicle kept intact medially beside the 'Pyriform aperture of the nose. This is then tucked under the floor of nose. But he dropped this technique in favour of using Dermofat taken from anterior aspect of thigh, for narrowing the wide nasal fossa.

The latest to the addition of various surgical procedures for treating atrophic rhinitis is complete nostril closure advocated by Austin Young (1967) from Sheffield. The skin of the vestibule is raised as a flap by an incision on the mucocutaneous junction and dissecting it forwards. This is than sutured in mid line. After a period of six months a complete closure on apparently normal mucous membrane is restored in the nasal cavity.
J.F. Neil (1967) followed the Young's operation in one case of unilateral atrophic rhinitis with deviated septum: Results is reported good.

Even after taking with various forms of treatment one really wishes to have a better solution for this chronic distressing disease.