HYPOTHESIS

VASOMOTOR RHINITIS – ATROPHIC RHINITIS: TWO ENDS OF AN AUTONOMIC SPECTRUM

R Raman

ABSTRACT

A hypothesis is put forward based on clinical observations and treatment modalities in vasomotor non-allergic rhinitis (V.M.R.) and primary atrophic rhinitis (P.A.R.). The hypothesis postulates that (1) V.M.R. and P.A.R. are two diseases at the ends of an autonomic spectrum (2) The anterior nasal aperture, its dimensions and sensory receptors play a vital role in the etiopathogenesis of both the conditions through reflex autonomic action.

Key words: Vasomotor rhinitis, atrophic rhinitis, anterior nasal aperture.

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INTRODUCTION

Vasomotor non-allergic Rhinitis (V.M.R.)

Vasomotor response as evidenced by swollen turbinates, watery rhinorrhaea and sneezing, induced by causative agents such as allergens, viruses, bacteria etc., are well known. The occurrence of a similar response, where no cause can be elucidated is termed vasomotor rhinitis. The etiology is attributed to autonomic dysfunction (Hilger 1951) or to be more specific, localised para sympathetic overactivity (Golding-Wood 1962). Similar response occurs in the nose on sympathetic interruption (Fowler 1943, Millionig et al 1950) as clinically evidenced after cervical sympathectomy (Golding-Wood 1962).

Histopathological changes observed are thickening of the mucous membrane, increase in the number of serous and mucous glands and vasodilation. The treatment modalities have been aimed at the affected end organ level (nasal mucosa) both medically or surgically in terms of increasing the nasal space by local decongestants, or shrinking and removal of easily accessible swollen tissues such as the inferior turbinate, the rationale being relief of symptoms. The treatment has also been directed at the autonomic level medically or surgically such as sympathomimetics and cholinoceptor antagonists (Dolovitch et al 1985). Or petrosal and vidian neurectomy (Golding-Wood 1979) rationale being interrupting the parasympathetic nerve supply to the nasal mucosa.

Surgery at the nasal level has evolved over the years from inferior turbinate injections, infracturing of inferior turbinates, surface and submucosal diathermy, cryosurgery to total inferior turbinectomy. These methods cause shrinkage or removal of the obstruction component of the disease. Submucous resection of the turbinate (Tremble 1966) removes the non responsive inferior turbinate bone, preserving the mucous membrane and is probably more physiological. Inferior turbinoplasty (Marby 1982) and more recently anterior turbinectomy (Fanous 1986), (removal of the inferior turbinate that projects into the anterior nasal aperture with preservation of the rest), is aimed at the anterior nasal aperture level and has shown better results. Since the anterior tip of the inferior turbinate extends into the nasal aperture, surgery of that structure should have a profound and salutary affect on the nasal airway (Fairbanks 1986). Fanous (1986) believes that the anterior tip of the inferior turbinate provides the key to the treatment of hypertrophic nasal mucosa.

Department of Otorhinolaryngology
Speech & Hearing
Christian Medical College & Hospital
Vellore 632004
S India

R Raman, MS, DLO, Professor

PRIMARY ATROPHIC RHINITIS

Conversely, atrophic rhinitis is a condition characterised by progressive atrophy of the nasal mucosa and underlying turbinates, squamous metaplasia, decrease in the number of submucous glands presenting clinically as abnormally patent nasal passages with secondary crusting and foul odour due to infection by saprophytic organisms. A similar histopathology is seen in terms of drying, shrunken mucosa and reduction of glands and secretions on para-sympathetic interruption (Millionig 1950).

Among the various other etiologies, a neural etiology is postulated namely sympathetic over-activity (Woloschin 1936, Sharma and Sardana 1966), sympathetic disturbance (Riegele 1936, Utrata 1950) and reflex sympathetic dystrophy (Ghosh 1987).

The management has been on similar lines directed at the end organ level to reduce nasal space, increasing secretions and inducing vasodilatation by medical or surgical means. The treatment has been directed at the autonomic level too similarly, medically such as parasympathetic stimulants (Henner & Busby 1943) and surgically such as stellate ganglion block (Woloschin 1936, Sharma and Sardana 1966), cervical sympathectomy and sphenopalatine ganglion blockade or extirpation (Shehata and Dogheim 1986). The evolution of surgery at the nasal level for primary atrophic rhinitis has progressed from various operations to decrease the nasal space with implants (Shehata and Dogheim 1986), through complete closure of nostrils (Young 1967) to partial closure of nostrils (Sinha et al 1977) and more recently vestibuloplasty (Ghosh 1987) with equally good results, similarly ending at the nasal aperture level.

To summarise, it can be seen that these two diseases, vasomotor non allergic rhinitis and primary atrophic rhinitis, are diametrically opposite of each other clinically, histopathologically and even in the approach to treatment medically or surgically. Surgically the treatment directed at the autonomic level has been parasympathetic blockage in vasomotor rhinitis, versus sympathetic blockade for primary atrophic rhinitis, whereas, surgery directed at the end organ level has been increasing the nasal patency by removal of tissues in V.M.R. versus decreasing the nasal patency by implantation in the submucosal level in P.A.H.

Coincidently surgery has evolved to direct at the nasal aperature level, increasing the nasal aperture (Famous 1986) in V.M.R. versus, decreasing the nasal aperture vestibuloplasty (Ghosh 1987) in P.A.R. giving the required result at the nasal passage. Both the diseases if left untreated progress to irreversible changes, hypertrophic in V.M.R. versus atrophic in P.A.R.

DISCUSSION

Autonomic Inervation of the Nasal Cavity

Autonomic inervation of arterioles and venous system has been described (Ritter 1970). Parasympathetic stimulation results in vasodilatation, with an increase in congestion and mucus production. Sympathetic stimulation results in vaso-constriction with increase in nasal patency and decrease in mucus secretion. Stimulation of the parasympathetic produces a marked watery secretion (Golding-Wood 1979). Recent evidence suggests that the nasal glands are inervated by cholinergic endings and are almost devoid of adrenergic fibres (Golding-Wood 1979). The vasculature and secretion from the nasal mucosal glands, which are of prime importance in the constant maintenance of temperature and humidity of the air entering the lower respiratory tract is under reflex control. For this system to function effectively, there must be an area of receptors, which senses the air, sends the messages through afferents to the autonomic centre, which in turn causes the required changes in the vasculature and mucosal secretions through its parasympathetic and sympathetic afferents. The general factors known to affect the autonomic vasomotor mechanisms are emotional, physical, inhalants and ingestants (Hilger & Hilger 1987). It is logically difficult to conceive of a localised autonomic (parasympathetic and/or sympathetic) activity in the hypotholamus or its connections affecting only the nose. A general autonomic response affecting the body in toto to a stimulus at the hypothalamic or cortical level is acceptable. With regards to the nose, the autonomic afferents have been demonstrated earlier (Chorobski and Penfield 1932) and by recent animal experiments on the Vidian nerve (Golding Wood 1979). Study on the local sensory receptors of the nose have been sparse. Cauna et al (1969) have described plexiform nerve endings, terminal arborisation and non myelinated nerves in the lower margins of inferior, and middle conchae and the corresponding areas in the septum. It is only logical to assume that these receptor mechanisms must exist and if they do, they must be at the entrance of the nose i.e. anterior nasal aperture. Recently sensory receptors at the nasal vestibule have been described (Gal'an Corte's et al 1986). It has been demonstrated that the squamous metaplasia occurring at the nasal aperture as a result of exposure to dry inspiratory air, reverts back to normal after the diversion of air stream following tracheostomy or laryngectomy (Ritter 1987).

The anterior nasal aperture constitutes the smallest cross sectional diameter of the entire airway where the respiratory air stream converges and takes a bend to flow into the nose (Proctor 1977). This has been confirmed more recently, that the greater portion of the nasal resistance is situated at the level of the anterior tip of the inferior turbinates (Haight and Cole 1983). Coincidently the evolution of the surgical treatment in vasomotor non allergic rhinitis as well as primary atrophic rhinitis have also reached up to the anterior nasal aperture level in terms of anterior inferior turbinectomy for V.M.R. and partial closure for P.A.R. respectively giving the required good results at the affected end organ level.

The anterior nasal aperture apparently seems to be the site which sensors the air entering the nose, sends the messages via the afferents to the hypothalamic autonomic centre which in turn reflexly causes the required changes in the nasal mucosa via the afferents to modify the air stream. In conclusion it is postulated that VMR and PAR may be the two ends of an autonomopathy the etiological factor being the anterior nasal aperture, in terms of its dimensions and sensory receptors mechanisms. Narrowing the anterior nasal aperture as done in primary atrophic rhinitis may expose less of the receptors and conversely enlarging the anterior nasal aperture as done in VMR possibly exposes more of the receptors. It may be argued that the anterior nasal aperture gets narrowed (VMR) or enlarged (PAR) as an effect and not the cause. It is possible not probable as the long term results of the surgical procedures show.

The postulate that VMR and PAR are opposite ends of the autonomic spectrum is based on clinical histopathological and surgical evidence. The postulate that anterior nasal aperture is the etiological site is based on the surgical evidence only. The second postulate may also provide a clue to the susceptibility of Arabs and Indians to VMR in Britain (Golding Wood 1979) and atrophic rhinitis being more common in yellow races, Latin races and American Negroes (Weir 1979). Further studies of the dimensions of the normal anterior nasal aperture as compared to the dimensions of the same in patients with VMR and PAR, racial differences in dimensions of the anterior nasal aperture, the type and distribution of the sensory receptors in this area are warranted.

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