▪ Summary:

Vasomotor rhinitis (VMR) is the most common form of chronic nonallergic rhinitis (NAR). Diagnosed in the absence of infection, allergy, eosinophilia, hormonal changes (such as pregnancy, hypothyroidism), and exposure to drugs (oral contraceptives, estrogens, angiotensin-converting enzyme (ACE) inhibitors, B-blockers, antihypertensives, aspirin, chlorpromazine, nonsteroidal anti-inflammatory drugs, and topical nasal decongestants).

Vasomotor rhinitis characterized by nasal obstruction, rhinorrhea, sneezing and/or nasal itching. VMR has been thought to result from an imbalance in the autonomic input to the nasal mucosa, perhaps hyperactive parasympathetic system and symptoms of VMR may be provoked by changes in temperature or humidity, odors, perfumes, smoke, alcohol, sexual arousal, and emotional factors.

Different pathogenic mechanisms are involved in the pathogenesis of VMR. (1) neurogenic and molecular mechanisms which demonstrate the role of sensory neuropeptides in generation of symptoms and signs of VMR as result of hyper-response of trigeminal nerve endings in nasal mucosa, (2) the role of nitric oxide (NO) which is strong vasodilator and generated through a cAMP-dependent mechanism in the pathogenesis of VMR, (3) the role of peroxynitrate in the pathogenesis of VMR.

Treatment of VMR varies from local or systemic nasal decongestants, intranasal and/or oral antihistamincs, intranasal steroids and intranasal ipratropium bromide to surgical interference through vidian nerve neurectomy, sphenopalatine ganglion block and inferior turbinectomy according to case severity.