A STUDY OF THE CHROMOSOMAL PATTERN AND CELL PROLIFERATION IN PRIMARY ATROPHIC RHINITIS

By
Assem A. Abdel-Azim and *El-Sayed A.F. Allam
Histology and Cytology, and *ENT Departments,
Benha Faculty of Medicine, Zagazig University.

ABSTRACT

Chromosomal pattern and cell proliferation studies were performed on blood samples of 20 male and female control subjects as well as 19 male and female primary atrophic rhinitis (AR) patients. The chromosome anomalies were noticed to be increased significantly in AR patients in whom the chromosomes were affected mainly in those regions containing the genes regulating immunoglobulin synthesis. Accordingly, a relative immunodeficiency was proposed; an opinion which was supported by cell proliferation studies which revealed an attenuated ability of AR leukocytes to undergo an adequate blastogenic response when stimulated with the plant lectin phytohaemagglutinin. The results suggest a genetic basis of the disease despite the negative family history in all cases studied.

INTRODUCTION

Atrophic rhinitis is a chronic nasal disease characterized by progressive atrophy of the mucosa and underlying bone of the turbinates and the presence of viscid secretion which rapidly dries and forms crusts that emit a characteristic foul odour sometimes called ozaena (a stench). There is an abnormal patency of the nasal passages.

The aetiology of atrophic rhinitis is still controversial. In the past, numerous organisms have been cited as the cause; among which are Cocccbacillus (Loewenberg, 1894), Bacillus mucosus (Abel, 1895), Cocccbacillus foetidus ozaena, diphtheroid bacilli, and klebsiella ozaena (Henriksen and Gundersen, 1959). Indeed, these organisms may be found in cultures but there is little evidence that they cause the disease. Other factors have been regarded as possible causes such as chronic sinusitis, excessive surgical destruction of the nasal mucosa, and syphilis (Weir, 1987). It was postulated that any lesion preventing the formation or maturation of large numbers of motile cilia, or the production of mucus capable of forming confluent sheets suitable for continuous propulsion, might cause atrophic rhinitis (Gray et al., 1980). The authors described, in their EM study, the fundamental cilial lesions found in other low cilia motility diseases (Usher's and Kartagener's syndromes) in which the microtubules and dynein arms of the cilia are affected.