A BRIEF COMMUNICATION

EOSINOPHILS AND THE LUNG IN TROPICAL PULMONARY EOSINOPHILIA

Tropical pulmonary eosinophilia (TPE) is commonly seen in areas endemic for filariasis. It must be remembered that the respiratory manifestations are part of a systemic disease characterised by marked increase in the blood eosinophil counts, malaise, fever and weight loss in addition to the respiratory symptoms. The clinical and laboratory features of the disorder have been extensively reviewed.[1-4]

The disorder has been known for over 50 years now, but the association with filarial disease began to be recognised with the demonstration of antifilarial antibodies and the findings of microfilariae in the tissue. Also of interest is the occurrence of high levels of IgE in the sera of these patients. Both animal and human filarial parasites have figured as etiological agents of the syndrome. Currently it is believed to represent an abnormal response to human parasite rather than sensitization to animal parasites[5].

The most significant haematological abnormality in this disorder is the intense eosinophilia which is an integral part of the syndrome. Before we consider the role of eosinophils in this disorder, a brief review of our current state of knowledge of these cells is in order. The immunobiology of the eosinophils[6] and eosinophilia[7] have recently been well reviewed.

The eosinophil was first described by Ehrlich over a hundred years ago. The cell is easily recognised by the characteristic granules that it possesses. The structure and content of these granules have been extensively studied during the last decade. There are at least two types of granules within the eosinophil. The large granules contain an electron dense core and a matrix. Four major proteins with diverse biological functions have been identified within the large granule. The most abundant of these which forms the crystalloid core is the major basic protein (MBP). MBP has been shown to be toxic to several parasites. Like other granulocytes, eosinophils also have a peroxidase which is capable of killing microbes by virtue of its ability to generate oxidants. Eosinophil cationic proteins (ECP) have profound effects on the coagulation system besides being toxic to parasites. The fourth protein is the eosinophil derived neutrotoxin which can produce extensive damage in experimental animals referred to as the "Gordon phenomenon".

The small granules contain a variety of enzymes such as aryl sulfatase and collagenases. The plasma membrane of the eosinophil contains lysophospholipase which crystallises to form the familiar Charcot-Leyden crystals.

Several abnormalities of the eosinophils have been identified in TPE. These include appearance of cytoplasmic vacuoles[8] and degranulation.[9] It must be noted that there is no relationship between the eosinophil counts and the clinical or radiological features of this disorder.

It has now been clearly established that eosinophils play an important part in immune mediated injury to parasites. However, of intereset is the growing body of information that these cells may be injurious to tissues, particularly the lung. Their role in the various allergic reactions was hitherto believed to be one of controlling the immediate hypersensitivity reactions. This view was based on the observation that the eosinophil possessed enzymes that could neutralise mediator activity. Recently Gleich et al[6] have convincingly shown that the eosinophil is an effector cell in immediate hypersensitivity reactions and may be responsible for a significant portion of the damage caused by such reactions. Extensive damage to the architecture of the bronchi with pathological changes similar to bronchial asthma have been traced to MBP activity. Eosinophils have been shown to be capable of releasing leukotrienes which are potent bronchospastic agents. Moreover, Davis et al[10] have demonstrated the presence of collagenases which are capable of cleaving lung collagen which could play an important role in the cytotoxicity of eosinophilia to normal tissue.

Weller and Goetzl[11] recognised the lung as the prime target organ of eosinophil injury. The findings of Cantin et al[12] that the lung has poor anti-oxidant potential suggest that this may be one of the factors which renders the lung susceptible to eosinophil mediated injury.

The relationship of eosinophils to lung injury in TPE has not been well established. It is not clearly known what stimuli attract eosinophils to the lung in TPE. Also unexplored is the possibility that eosinophils are responsible for the bronchospasm seen in this disorder either directly by their ability to release leukotrienes or by their capacity to activate basophils or mast cells. Such questions await examination of the issue by adopting techniques which can further probe into the functions of eosinophils within the lung.

V. KUMARASWAMI
P. R. NARAYANAN
Tuberculosis Research Centre, Chetpet, Madras-600 031

Madras
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