STUDY OF T-LYMPHOCYTES IN PLEURAL EFFUSIONS

THESIS

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In Chest Diseases and Tuberculosis

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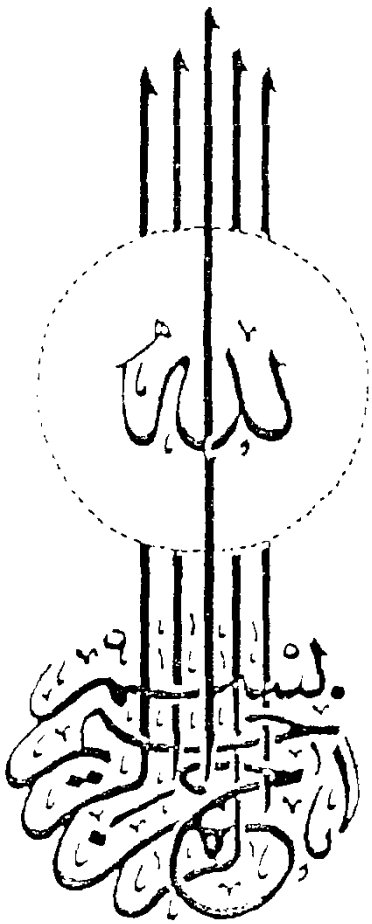

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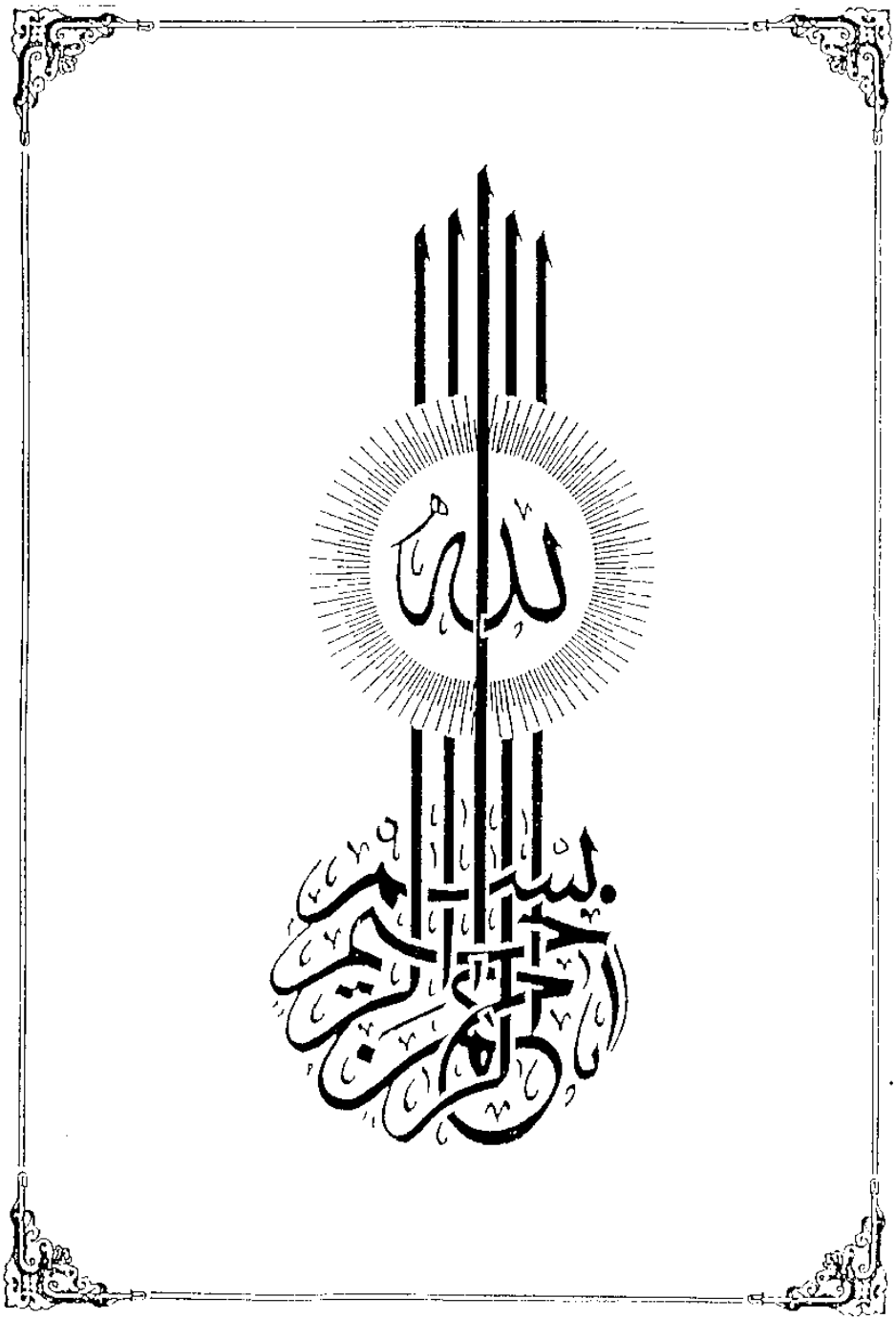

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Introduction
&
AIM OF WORK

INTRODUCTION AND AIM OF WORK

Pleural effusion often pose diagnostic difficulties despite the use of routine diagnostic methods. A variety of diseases may be associated with pleural effusion and several techniques are employed to determine an etiologic diagnosis in order to reach a proper management.

In the course of clinical, cytologic and histologic study of groups of patients with pleural effusions, various cellular patterns are found, in particular the degree of lymphocytosis was very informative often offering a clue to or leading to the possible underlying pathogenesis (Yam, 1967).

It was found that human peripheral blood and pleural fluid T-lymphocytes can bind with sheep erythrocytes to form a characteristic rosette configuration (Herscowitz, 1978).

The aim of this work is to throw some light on E-rosette forming cells in both pleural fluid and peripheral blood in patients with pleural effusion. This may help to reach an etiologic diagnosis in idiopathic cases.

Review of Literature

[CHAPTER 1]

MORPHOLOGY OF THE PLEURA

Each lung is invested by a serous membrane arranged in the form of closed invaginated sac termed the pleura. The visceral pleura convers the surface of the lung and lines the fissures between its lobes, while the parietal pleura lines the inner surface of the hemithorox and is subdivided into mediastinal pleura, diaphragmatic pleura and costal pleura. (Warwick and Williams, 1973).

Structure of the Pleura:

The pleura consists of a thin superficial layer known as endapleura and a dense deeper layer known as the chief layer. The free surface of the pleura is smooth and moistend by serous fluid, it is coverd by a single layer of flattened cells which form a mesothelium and rest on a basement membrane beneath which are networks of yellow elastic and white fibers, embeded in ground substance, containing also fibroblasts, macrophages and other cell types which characterize loose connective tissues. Blood vessels, lymphatics and nerves are distributed in the substance of the pleura (Fraser and Pare, 1985).

The electron microscope has shown that the surface of the mesothelial cells is not smooth but bears microvilli

varying in length up to 3 um. The number of microvilli varies from cell to cell but increases in the visceral more than in the parietal pleura. They may have an absorbtive function, but may also increase surface mucopolysaccharide and so, counteract frictional forces (Corrin, 1980).

Blood Supply:

The blood supply of the costal part of the parietal pleura is from the intercostal arteries, while the mediastinal and diaphragmatic parts are supplied from the pericardiophrenic branch of the internal mamary artery (Crofton and Douglas, 1981a), while the blood supply to the visceral pleura is mainly from bronchial arteries. However venous drainage of the visceral pleura is via the pulmonary veins (Agostoni, 1972), and that of the parietal pleura is via the intercostal veins.

Lymphatic Drainage:

The lymphatics of costal part of parietal pleura drain into glands along internal mamary artery (sternal glands) and to the intercostal glands near heads of the ribs. The lymphatics of diaphragmatic part drain into the sternal, anterior and posterior mediastinal lymph glands, the lymphatics from the mediastinal part accompany the pericardiophrenic artery and drain into the

posterior mediastinal glands (Simer, 1952).

The lymphatic drainage of the visceral pleura is via a small subpleural network of lymphatics within the interlobular connective tissue which communicate with the deep lymphatic vessels within the lung (Simer, 1952).

Nerve Supply:

The visceral pleura is supplied by autonomic fibers only and is essentialy devoid of pain fibers while the partietal pleura is richly supplied with sensory nerve fibers from the intercostal nerves. The sensory supply to the central portion of the diaphragmatic pleura is via phrenic nerve (Robert and Robert, 1974).

Fig. (1): Pressures involved in transudation and reabsorbtion of pleural fluid (Agostoni and Mead, 1964).

PARIETAL PLEURA		VISCERAL PLEURA	
Factors causing pleural fluid formation	Factors causing pleural fluid absorbtion	Factors causing pleural fluid formation	Factors causing pleural fluid absorbtion
1. Capillary hydrostatic pressure - 30 cm H_2O	1. Plasma colloidal osmatic pressure - 34 cm H_2O	1. Cappillary hydrostatic pressure - 11 cm H_2O	1. Plasma colloidal osmatic pressure - 34 cm H_2O
2. Pleural fluid colloidal osmotic pressure - 8 cm H_2O		2. Pleural fluid colloidal osmotic pressure - 8 cm H_2O	
3. Intra pleural pressure - -5 cm H_2O		3. Intra pleural pressure -5 cm H_2O	
TOTAL 43 cm H_2O	34 cm H_2O	24 cm H_2O	34 cm H_2O
RESULTANT 9 cm H_2O filteration into the pleural cavity.		10 cm H_2O absorbtion into visceral pleura.	

pressure (11 cm H_2O) plus the intra pleural negative pressure (-5 cm H_2O), the resultant is 10 cm H_2O causing pleural fluid absorbtion into the visceral pleura (Agostoni and Mead, 1964).

At equilibrium the flow of liquid through the two membranes must be equal (except for the drain through the lymphatics that under normal conditions should be nearly negligible). When a saline solution is introduced into the pleural space the equilibrium is disturbed, the pressure of the pleural liquid rises and therefore the net driving pressure across the visceral pleura increases while that across the parietal pleura decreases. So the outflow of liquid through the visceral pleura should become higher than the inflow through the parietal pleura. The volume of the liquid is then reduced until the deformation forces produced by the contact between the membranes lower the pressure of the pleural fluid to its original value, preventing further reduction of its volume, and the equilibrium is thus established (Green and Pride, 1980).

[CHAPTER 3]

DIAGNOSIS OF PLEURAL EFFUSION

Diagnosis of pleural effusion follows the same orderly for other forms of chest disorders. Simple noninvasive studies are carried out first, after which, when indicated, more complicated invasive procedures are performed. Accordingly, the initial evaluation consists of history and physical examination, roentgenographic studies and laboratory tests (Hamid and Robert, 1977).

The Clinical Picture:

Patients with pleural effusion are usually seen by physicians because of dyspnea, pleurisy or symptoms related to the underlying cause of the disorder. Some patients are found to have unsuspected pleural effusion when physical or roentgenographic examinations are performed for some other purpose (Hinshow and Murry, 1980).

Signs of pleural effusion include restriction of movement of the chest wall on the affected side, stony dullness on percusion, diminished to absent tactile vocal fremitus, and decreased to absent breath sounds. In pleural inflamation that is associated with little or no effusion, friction rub is most likely to be heard (Johnston and Dovnarsky, 1980).

Chest Roentgenogram:

Pleural effusion usually appear as dense homogeneous opacities. A very small effusion may be only represented by obliteration of a costophrenic angle and may be difficult to be distinguished from pleural thickening or adhesions between the diaphragmatic and lower costal pleural surface. Lateral decubitus film will distinguish free pleural effusion as the fluid gravitates to the lateral chest wall and casts a much more evident shadow than that in the upright position (Hinshaw and Murry, 1980).

The amount of fluid necessary to produce blunting of the costophrenic angle is usually 500 to 600 ml. On the chest radiograph, pleural effusion usually appears as a homogenous density with a concave upper border at the base of the hemithorax. The distribution of pleural fluid is determined principally by gravity and by the availability of a free pleural space (Conston and Dovnarsky, 1980).

Sometimes effusions are trapped or loculated and do not assume the characteristic upward curve. A common location is between the inferior margin of the lung and the superior surface of the diaphragm. It may be necessary to induce a pneumoperitonium to distinguish it from a raised hemidiaphragm. On the left side, the distinction

may be made by observing the position of the stomach gas bubble, created artificially by giving sodium bicarbonates (Crofton and Dougls, 1981b).

Pleural effusion often shifts the mediastinum to the opposite side. When an obstructing endobronchial lesion produces both atlectasis and pleural effusion, the mediastinum often remains in place. Massive pleural effusion without mediastinal shift suggests fixation of the mediastinum and is most often produced by bronchogenic carcinoma (Johnston and Dovnarsky, 1980).

If the fluid level is horizontal, air or gas has entered the pleural space, a common circumstance after thoracocentesis. If thoracocentesis has not been performed, the presence of an air fluid level indicates presence or former communication with the tracheobroncheal tree or infection with a gas forming organism (Hinshaw and Murray, 1980).

Ultrasonography:

Ultrasound is often helpful in determining the presence and location of the fluid in the pleural space. When the high-frequency sound wave is directed into the normal thoracic cavity, the sound is almost completely reflected by the "air-containing" lung at the surface. When pleural fluid is present, however, an