The Search For Mycotoxins In Pleural Effusion

A Thesis

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By

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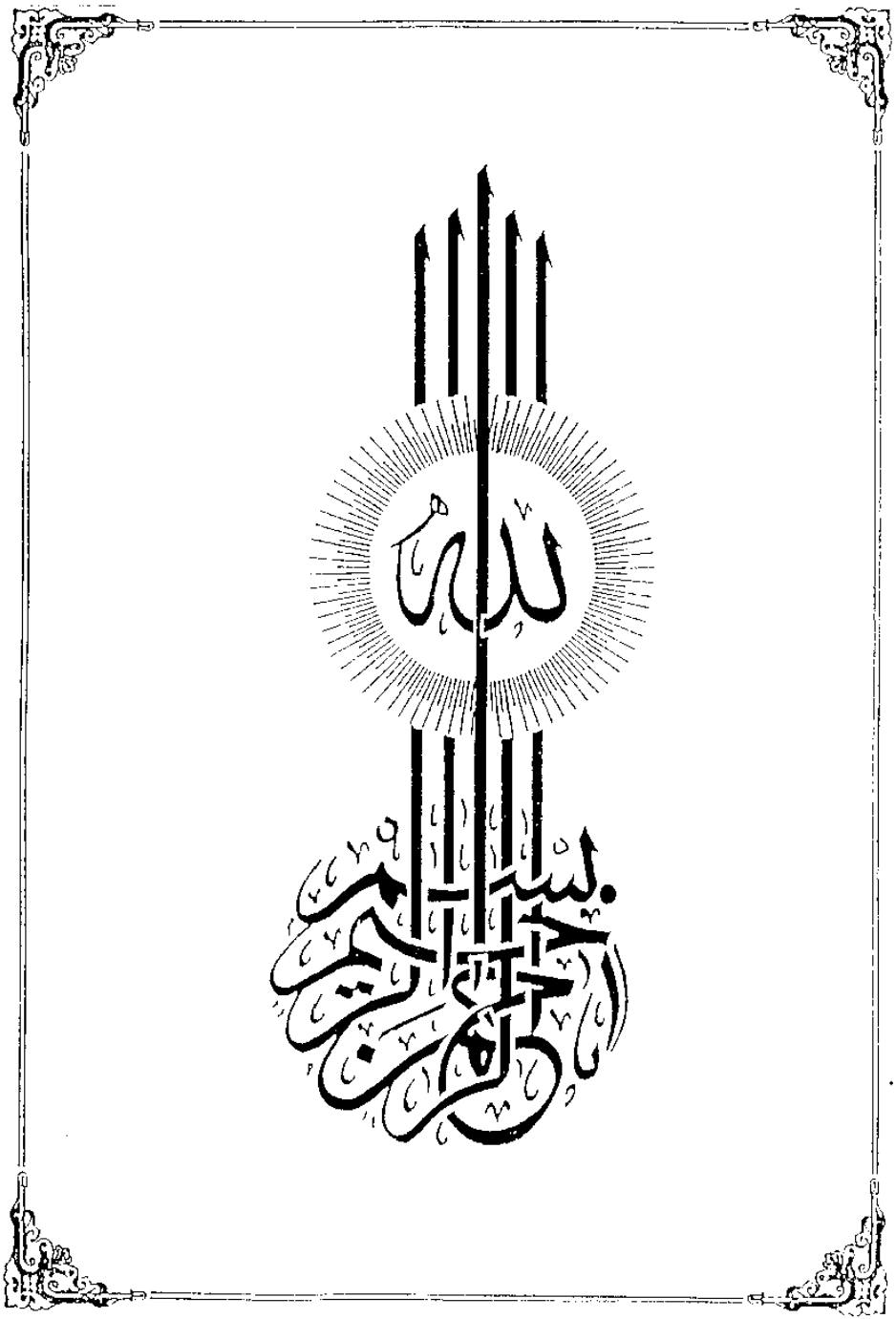

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Introduction

It is generally accepted that the protection of human health and well-being is the primary purpose of environmental pollution control. People under malnutrition would suffer even so much more from mycotoxins as the well cared ones. The international reports confirmed these facts which revealed that mycotoxicosis outbreaks involved wide spread area all over the world, Gechoslovakia (Rye's syndrome), India (cirrhotic children) and more than 9 countries in Africa and 4 countries in Asia (carcinogenic effects) due to mycotoxins. Human health has been dramatically affected in outbreaks of mycotoxins, but these tragic events may be only a part of the cost to society in terms of impaired health and productivity from the exposure to subclinical levels of mycotoxins.

Little is known, however, of health effects resulting from exposure to trichothecenes, but there are no available literatures dealing with human respiratory systems.

Aim of work:

This work aims to give a review on the subject of pleural effusion and to search for mycotoxins (Trichothecenes) in pleural fluid of 30 patients with different diseases.

Review Of Literatures

Pleural Effusion

A pleural effusion is an accumulation of fluid in the pleural space as a result of excessive transudation or exudation from the pleural surfaces. (Crofton and Douglas, 1981).

Pathophysiology:

The pleural space is lined by the parietal and visceral pleural membranes which are permeable to liquid and gas. In normal persons this space contains a small amount of liquid but on gas. (Black, 1972).

Gas does not accumulate in the pleural space because of the low total gas pressure in venous blood. At sea level the partial pressures of the various gases in venous blood are: P_{O2} = 40 mmHg, P_{CO2} = 46 mmHg, P_{N2} = 573 mmHg and P_{H2O} = 47 mmHg. The total of these partial pressures, 706 mmHg, is 54 mmHg (73 cm H_2O) less than atmospheric pressure. The pressure in the pleural space at resting lung volume is 5 Cm H_2O less than atmospheric pressure, therefore, there is a gradient of 68 cm H_2O favoring absorption of gas from the pleural space, and the pleural space remains gas- free.

There is a measurable amount of pleural fluid in healthy humans (Black, 1972).

Muller and Lofsted (1945) reported the roentgenographic demonstration of pleural fluid in 13 of 120 healthy subjects

(12.5%). They modified the lateral decubitus position by elevating the pelvis so that the most concave portion of the upper thorax was dependent.

They performed thoracentesis on some of their patients, and concluded that the smallest amount of fluid that could be indentified by this technique was 3 to 5 ml. and the greatest amount of fluid found in healthy subjects was 15 ml.

The concentration of pleural fluid protein in healthy men is similar to the protein concentration in interstitial fluid elsewhere in the body. (Black, 1972).

Pleural fluid transport in normal subjects:

Under normal conditions, fluid is filtered out of the parietal pleural capillaries. This fluid may be absorbed by the visceral pleural capillaries and by the lymphatics, the protein in the fluid is absorbed by the lymphatics.

There are extensive communications between the subserosal lymphatics on the superior and inferior surfaces of the diaphragm. Since the visceral pleura is more vascularised than the parietal pleura, the resistance to flow of fluid should be lower through the visceral membrane . The net pressures in the visceral pleural capillaries favour absorption. Therefore, one might expect all of the pleural fluid to be absorbed. To prevent complete absorption, the

absorption pressure of the visceral pleural capillaries must decrease as fluid is absorbed. The absorption pressure in the visceral pleural capillaries is - 10 cm H_2o. In order for the absorption pressure to be decreased as fluid is absorbed, either the colloid osmotic pressure of pleural fluid or the mean intrapleural pressure must increase. To decrease the absorption pressure to o, a colloid osmotic pressure of pleural fluid of 16 cm H_2o would be required. This is equivalent to a pleural fluid protien concentration of about 4 gm/100 ml. if the pleural fluid proteins are similar in type to the plasma proteins. This pleural fluid protein concentration is greater than that found in normal pleural fluid. (Black, 1972). As pleural fluid is absorbed from the pleural space, contact points develop between the visceral and parietal pleurae. Local stretching of the pleural membrane occur at these points, and the pressure in the fluid in the spaces between the contact points, (mean intrapleural pressure), is therefore decreased due to the adjacent pleural deformation. This increased negative pressure in the liquid between the contact points decreases the absorption pressure and prevents complete reabsorption of the pleural fluid. (Black, 1972).

Pleural fluid transport in disease states:

Several factors involved in the formation and removal of pleural fluid may be altered in various ways in disease states, leading to the production of a pleural effusion.

- Filtration coefficient:

In the presence of inflammation, the filtration coefficient increases. This change may be due to damage to the vascular basement membrane or to the action of substances such as histamine and kinins that may be released by the inflammatory process. If an increase in local blood flow accompanies the inflammation, capillary hydrostatic pressure may increase. Increased temperature will also increase the filtration coefficient. (Landis and Pappenheimer, 1963).

The increased permeability associated with the inflammation produces increased protein loss from the capillaries. Certain substances, such as thioureas, increase capillary permeability by a direct toxic effect on the endothelial cells, specially in the lung, and produce pulmonary edema and pleural effusion. It was suggested that the fluid loss from the capillaries in the lung exceeded the capacity of the lymphatics to remove it, and the fluid in some manner moved into the pleural space. (Black, 1972).

- Colloid osmotic pressure:

The electrophoretic pattern of pleural fluid protein is that; the pleural effusions have the same major protein fractions as plasma, albumin is usually present in slightly higher proportions in pleural fluid than in plasma, probably

due to the smaller size of the albumin molecule, the proportion of B-globulin and fibrinogen in pleural fluid is generally lower than in plasma (the decrease in fibrinogen is probably due to defbrination in the pleural space). With an inflammatory process, pleural fluid protein contentration may approach the plasma concentration.

The colloid osmotic pressure of the pleural fluid is a factor in determining how the fluid may be reabsorbed. With normal proteins, the absorption pressure of the visceral pleura is -10 cm H_2o. Since the pleural fluid protein profile is similar to that of plasma, a pleural fluid protein concentration of about 4 gm/100 ml. is necessary to reduce the absorption pressure to 0. When the pleural fluid protein concentration is this high, there will be no net absorption by the visceral pleura. In this situation, pleural fluid must be absorbed by the lymphatics, at least untill the protein concentration is decreased. In patients with hypoproteinemia the critical level for edema formation is a plasma albumin concentration of about 1.5 gm/100 ml., and edema usually is present when the plasma albumin concentration is 1 gm/100 ml. or less. (Black, 1972).

- Hydrostatic pressure:

The greater increase in pleural fluid during systemic venous hypertension may be due to a higher filtration