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Comparative Study Of Pleurodesis Using Iodopovidone (Betadine) and Bleomycin In Management Of Malignant Pleural Effusion.

Thesis

Submitted For Full Fulfillment Of Master Degree In Chest Diseases And Tuberculosis

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Introduction

Malignant pleural effusion (MPE) represents an important source of morbidity for patients with underlying cancer. It can occur as the initial presentation of cancer, as a delayed complication in patients with previously diagnosed malignancies, or as the first manifestation of cancer recurrence after therapy (**Heffner and Klein, 2008**).

Malignant pleural effusions can result from primary malignancies of the pleurae or from underlying intrathoracic or extrathoracic malignancies that reach the pleural space by hematogenous, lymphatic, or contiguous spread (Martinez-Moragon et al, 1997).

Patients with malignant pleural effusion have significantly short survival and the treatment is usually palliative. Not all malignant pleural effusions require treatment and the criteria for instituting therapy for malignant effusion are based on the frequency and pattern of reaccumulation in addition to the degree of symptomatic compromise (Lisa et al, 1994).

Pleurodesis is the process by which adhesions are induced between the visceral and parietal pleurae in order to obliterate the pleural space. It is commonly used method for palliative theraby of malignant pleural effusion (**Mourad et al, 2004**).

Chemicals such as bleomycin, tetracycline, povidone iodine, or a slurry of talc can be introduced into the pleural space through a chest drain. The instilled chemicals cause irritation between the parietal and the visceral layers of the pleura which closes off the space between them and

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prevents further fluid from accumulating (American Thoracic Society, 2001).



Aim of the Work

The aim of this work is to compare efficacy and possible complications of iodopovidone and bleomycin when used for pleurodesis in patients with malignant pleural effusion.

Acknowledgment

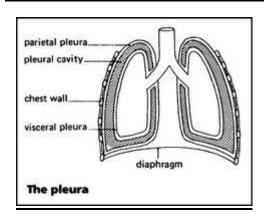
I thank **ALLAH** who granted me the ability to complete the work of this study.

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Anatomy of the Pleura



Fig(1):showing anatomy of the pleura.

Embryology of the pleura and pleural space:

The coelomic cavity in the embryo is a U-shaped system with the thick bend cephaled. The cephaled portion becomes the pericardium and communicates bilaterally with pleural canals, which in turn communicates with the peritoneal canals. With development, the coelomic cavity becomes divided into the pericardium, the pleural cavities and the peritoneal cavity. These newly formed pleural cavities are fully lined by a single layer of mesothelial cells (Arey, 1965).

The primordial bronchial buds and the trachea lie in a median mass of mesenchyme, cranial and dorsal to the peritoneal cavity. This mass of mesenchymal tissue is the future mediastinum and it separates the two pleural cavities. As the growing primordial lung buds bulge into the right and left pleural cavities, they carry with them a covering of the lining mesothelium which becomes the visceral pleura. As the separate lobes evolve, they retain their mesothelial covering which become the visceral pleura in the fissures, and the lining mesothelium of the pleural cavity becomes the parietal pleura (**Krahl**, 1964).

Gross anatomy:

The visceral pleura envelops the entire surface of the lungs, the parietal pleura covers the inner surface of the chest wall, mediastinum and diaphragm, the visceral pleura invests the lungs every where except at the hilum, where the bronchi, pulmonary vessels and nerves enter the lung substance (Von Hayek, 1960).

Below the merger of the visceral and parietal pleurae at the hilum, pleural reflections from the dorsal and ventral surface of the lungs usually extend to the diaphragm as a double layer of mesothelial tissue. In humans, the two pleural cavities are completely separate (**Kinasewitz**, 1998).

Microscopic anatomy:

The normal parietal and visceral pleural linings are smooth, glistening, semitransparent membranes. Beneath the single layer of mesothelium that covers the surface is a band of connective tissue containing abundant collagen and elastin. Mesothelial cells vary in size and shape from flat to columnar. Numerous mitochondria, rough endoplasmic reticulum and Golgi apparatus are prominent features of cuboidal and columnar cells suggesting that they are active in transport of substances across the pleural space (Wang, 1985).

Although the parietal and visceral membranes are similar in external appearance, important anatomic differences are found beneath the surface. Beneath the parietal surface, the arrangement of the connective tissue layer is straight forward; in contrast the sub mesothelial connective tissue layer of the visceral pleura gives rise to septae that permeate the lungs, creating subdivisions that enhance gas exchange while lending support to the lung parenchyma (**Kinasewitz, 1997**).

Mesothelial cells are frequently dislodged from the pleural surfaces and are present free in the pleural fluid; these may be transformed into macrophages capable of phagocytosis. Not all the macrophages of the pleural fluid evolve from mesothelial cells, and some may evolve from alveolar macrophages (Bakalos et al, 1974).

Blood supply of the pleura:

☒ Arterial supply:

The parietal pleura receives its blood supply from the systemic circulation. The costal pleura is supplied by small branches of the intercostal arteries and the internal mammary artery, whereas the mediastinal pleura is supplied principally by the pericardiophrenic artery; a branch of the internal mammary artery. The diaphragmatic pleura is supplied by the superior phrenic (from thoracic aorta) and the musculophrenic arteries (from internal thoracic arteries). The visceral pleura receives its blood supply from the systemic circulation (through the bronchial arteries) and the pulmonary circulation (Wang, 1975).

The visceral pleura on the costal surface of the lung and on most of the diaphragmatic surface are supplied by numerous twigs of the pulmonary artery and the bronchial artery. Most of the mediastinal pleura and the interlobular surfaces, receive blood from the bronchial arteries. The terminal branches of the arteries supplying the visceral pleura end in a loose network of large capillaries (Von Hayek, 1960). These capillaries allow the pressure in the entire visceral pleura to be that of the pulmonary circulation rather than that of the systemic circulation, which favours pleural fluid absorption (Wang, 1975).

▼ Venous drainage:

The visceral pleura is drained primarily by the pulmonary veins and

the parietal pleura by the intercostals. The mediastinal portion drains into the bronchial veins (Wang, 1985).

▼ Pleural lymphatics:

The parietal pleura is distinguished by the presence of the stomata, 2 to 12 micron, openings between mesothelial cells (Wang, 1985). These stomata are essential for the exit of the pleural fluid, protein and cells from the pleural space. These stomata communicate directly with lymphatic channels that drain to lymph nodes (Teklu, 1999).

The lymphatic vessels of the costal pleura drain ventrally towards sternal nodes along the internal thoracic artery and dorsally towards the internal intercostal lymph nodes near the heads of the ribs. The lymphatic vessels of the mediastinal pleura pass to the tracheobronchial and mediastinal nodes, whereas the lymphatic vessels of the diaphragmatic pleura pass to the parasternal, middle phrenic and posterior mediastinal nodes. All these groups of lymph nodes drain into the bronchomediastinal trunk which drain to the thoracic duct (on the left side) and the right lymphatic duct (on the right side). Both drain to the right and left innominate veins (Shanmugam et al, 1998). No stomata are seen in the visceral pleura and the lymphatic vessels of the visceral pleura are separated from the mesothelial cells by a layer of connective tissue. The lymphatic vessels over the visceral pleura form a plexus of inter communicating vessels that run over the surface of the lung towards the hilum and also penetrate the lung to join the bronchial lymph vessels by passing in the interlobular septa (Wang, 1975).