Malignant Pleural Mesothelioma with Pleural Plaques: A Case Report

Narongpon Dumavibhat, MD, PhD1; Ruchira Ruangchira-urai, MD2; Nisa Muangman, MD3; Punnarerk Thongcharoen, MD4; Sitthiphon Bunman, MSc1; Apinut Jaroonpipatkul, MD1

Diffuse malignant mesothelioma (DMM) is an aggressive tumor of the mesothelium associated with a poor prognosis. Asbestos exposure is the major cause of the disease. Furthermore, pleural plaques are also caused by asbestos exposure. The authors present a case with malignant pleural mesothelioma and pleural plaques.

Abstract
Diffuse malignant mesothelioma (DMM) is an aggressive tumor of the mesothelium associated with a poor prognosis. Asbestos exposure is the major cause of the disease. Approximately 90% of DMM cases occur in the pleura and are associated with poor prognosis due to delay in diagnosis. Most cases of the cancer are caused by asbestos exposure. Asbestos can not only cause DMM but also many diseases. Asbestos-related diseases (ARDs) include non-malignant disorders and malignancies. The aim of the present study was to present a patient with malignant pleural mesothelioma (MPM) and pleural plaques (PPs) to raise concern about health problems arising from asbestos.

Case Report
A 66-year-old English male, staying in Chiang Rai province, presented with a 2-month history of pleuritic chest pain. Decreased breath sounds with dullness to percussion were detected at the lower part of the left hemithorax. Chest radiograph (CXR) revealed diffuse thickening of the left pleura with a small amount of left pleural effusion. There was a focal extra-pulmonary opacity at lateral aspect of the right middle lung field (Figure 1).

Figure 1: CXR (PA upright) shows diffuse thickening of the left pleura (white arrows) with a small amount of left pleural effusion. The focal extra-pulmonary opacity is found at the lateral aspect of the right middle lung field (black arrows).
Computed Tomography (CT) of the chest from another hospital revealed circumferential pleural thickening at left chest wall and mediastinal sites (not shown). His previous pleural biopsy from another hospital was reported as benign pleura. Based on the chest CT findings, DMM was highly suspected. A definite diagnosis was necessary for the thoracic surgeon’s decision to select an appropriate operation. However, the patient refused a repeat biopsy because he had suffered from pleuritic chest pain for quite a long time. Although an intraoperative consultation is not recommended for the first diagnosis of DMM, one was performed after discussion between the thoracic surgeon and the pathologist with the relevant and supportive clinical information and radiographic findings.

Two pieces of fresh, thickened pleural tissue, measuring 1x0.7x0.6 and 1.5x1.3x0.7 cm, were received for intraoperative consultation (Figure 2). One frozen section showed diffuse infiltration of cytologically bland spindle cells with repetitive storiform growth pattern into deep adipose tissue. These findings, despite a definitive confirmation still being required with an immunohistochemical study, were highly suggestive of desmoplastic malignant mesothelioma.

A left extra-pleural pneumonectomy was performed on the patient. The lung was encased by diffuse, sheet-like thickening of pleural mass (Figure 3). Microscopic examination demonstrated a pleural tumor with a prominent paucicellular fibrotic pattern of spindle mesothelial cell proliferation with repetitive storiform appearance similar to those seen in the frozen section. The tumor invaded into the visceral pleura including fissures, lungs, pericardium, endothoracic fascia, chest wall adipose tissue and the diaphragm. The diagnosis of desmoplastic malignant mesothelioma was made after immunohistochemical staining which was positive for AE1/AE3, vimentin, as well as D2-40, but tested negative for BerEP4, MOC31 and desmin. Focal staining for calretinin, WT-1 and thrombomodulin was also present (Figure 4). Hyaline pleural plaques were detected (Figure 5). The remaining lung showed a few scattered asbestos bodies adjacent to bronchovascular bundles (Figure 6). No lung fibrosis was found.

His CXR following surgery demonstrated total opacity of the left hemithorax with evidence of left lung volume loss. A faint extra-pulmonary opacity was also detected at the lateral aspect of the right middle lung field (Figure 7). Chest CT showed multiple calcified pleural plaques which corresponded to the extra-pulmonary opacity on the CXR (Figure 8).

All of the data were collected from retrospective chart reviews. The patient worked as an engineer and has been married for 30 years. It was noted that he had a history of 10-year asbestos exposure while he had worked in England. There were no records about asbestos exposure or his occupation while he stayed in Thailand. He had a history of colon cancer and underwent surgery 2 years ago at a hospital in Chiang Rai.

**Figure 2:** Pleural tissue received intraoperatively is thickened due to a fleshy grey white tumor infiltrating thoroughly to deep tissue (A). The pleural tumor shows storiform fascicles of bland spindle cells in eosinophic collagenized background (B).

**Figure 3:** External surface of the left lung is almost entirely covered by thick pleural tumor (A). The lung is collapsed and diffusely encased by firm, grey white pleural tumor (red arrow). The tumor also involves fissures of the lung (yellow arrow).
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Figure 4: Microscopic findings of the pleural tumor (top image) show similar features with those seen in the frozen section (Figure 2B). The tumor cells are positive for calretinin, D2-40 and thrombomodulin, which are the markers that support mesothelioma.

Figure 5: Hyaline pleural plaque (*) coexists with and lines over malignant mesothelioma (*). It consists of layers of acellular hyalinized collagen (inset) arranged in “basket-weave” pattern.

Figure 6: An asbestos body is embedded in fibrous tissue around a bronchovascular bundle in the non-neoplastic lung parenchyma. It shows a golden-brown, beaded structure with a thin translucent core (A), which is more clearly demonstrated as a furrugeous body by iron stain (B).
**Figure 7:** CXR (AP supine) shows total opacity of the left hemithorax with evidence of left lung volume loss compatible with post pneumonectomy change. The faint extra-pulmonary opacity is also detected at the lateral aspect of the right middle lung field (white arrows). The absence of posterior left rib 7th and 8th (black arrows) is due to post thoracotomy.

**Figure 8:** CT chest (mediastinal window) in axial and coronal views show multiple calcified pleural plaques (arrows) which correspond to extra-pulmonary opacity on CXR and left pleural effusion from post-pneumonectomy (asterisk).
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Discussion

DMM is a rare cancer in which the highest annual incidence rate is approximately 30 cases per million observed in Australia, Belgium, and Great Britain. It is estimated that DMM is responsible for 43,000 deaths per year worldwide. Importantly, MPM is strongly associated with asbestos exposure. Concerning one study, the attributable risk for asbestos exposure among men with MPM was 88%. The latency of DMM from the time of first exposure until the onset of symptoms is variable, and is usually greater than 20 years. Due to the grave prognosis and poor response to treatment, most patients with DMM die within 2 years of diagnosis.

Asbestos is a group of naturally occurring fibrous silicate minerals (i.e. serpentine and amphibole). It is widely used due to its extraordinary tensile strength, poor heat conduction and relative chemical resistance. Asbestos can cause many ARDs including malignant and non-malignant diseases. DMM, lung cancer, laryngeal cancer, and ovarian cancer are the malignant diseases caused by asbestos, while pleural effusion, rounded atelectasis, pleural plaques, and diffuse pleural thickening are the non-malignant disorders caused by asbestos.

CXR is one of the most effective screening tools for ARDs. It is very important to look for the PPs (these may be calcified), as a marker of asbestos exposure. PPs are focal areas of pleural thickening with clearly demarcated edges. PPs themselves are harmless and can be recognized as a marker of previous asbestos exposure. Locations most commonly encountered include posterolateral, mediastinal as well as diaphragmatic pleura and most occurring in lower parts, sparing the apices. The latency period of PPs from the time of first exposure to asbestos until the appearance is typically reported is between 15–40 years.

In the present study, the authors reported a case of MPM with pleural plaques. To the best of our knowledge, there have only been two reported cases of PPs associated with other ARDs in Thailand. Both cases are patients with asbestosis and PPs. It was noted that the patient had a history of 10-year asbestos exposure while he had worked in England. Nevertheless, there was no record of the patient’s important medical history while he stayed in Thailand. A careful observation of occupational and environmental exposure to asbestos is necessary. A patient’s occupation, spouse’s occupation, hobbies, details of the house in which he/she lives are examples of significant details needed in an effective history-taking. The World Health Organization stated that there is no safe threshold for the carcinogenic effect of asbestos. The most efficient way to eliminate ARDs is to stop using all types of asbestos. More than 50 countries have banned the use of asbestos. Thailand used to be in the top 5 countries in the world as an importer and user of asbestos. The country still uses chrysotile (serpentine asbestos) although the number of these imports has declined.

This case report is an important reminder to chest physicians and general practitioners to be aware of this rare condition that needs careful history-taking especially discovering the patient’s previous occupations, conducting a complete physical exam and performing adequate tissues specimen analysis for definite diagnosis. An experienced multi-disciplinary team is vital and should include physicians, surgeons and pathologists. People in Thailand and the Asia Pacific region should be aware of ARDs due to the prevalent and wide use of asbestos in many industries especially construction-related industries.

Conclusion

The report demonstrates a case of ARDs that is MPM with PPs. Both MPM and PPs develop after a long latent period of exposure to asbestos. Careful history-taking is very important to show a link between asbestos exposure and ARDs especially MPM.

References