Case Report

Pathological Confirmed Diagnosis of Asbestosis: The First Case Report in Thailand

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Asbestosis is an occupational lung disease defined as pulmonary fibrosis caused by asbestos. Asbestosis was previously reported in Thailand based on radiologic findings, which demonstrated interstitial lung with calcified pleural plaques, and the patient worked in a fiber cement factory. However there was some doubt about the diagnosis because clinical and radiological findings are nonspecific; there was no data support of asbestos exposure in the patient and no histologic confirmed diagnosis. Histologic diagnosis is most useful when an equivocal of a history of asbestos exposure in patients with interstitial lung diseases take place. The authors report a patient presenting with progressive dyspnea for 2 years. She worked in an electric, wire, mesh fan cover factory to check quality of protective wire mesh for 10 years until the factory was closed 6 years ago. This type of factory had never officially reported asbestos use. Her clinical manifestations and radiologic findings are compatible with interstitial lung disease. She subsequently underwent thoracotomy with wedge lung resection. Pathology revealed interstitial fibrosis with honeycombing. Asbestos bodies were found more than 10 per cm^2 in the fibrosis. She was diagnosed asbestosis. The patient is suffering from dyspnea, severe hypoxemia and cor pulmonale. The patient is put on waiting lists for heart lung transplantation. The authors thus confirmed that asbestosis exists in Thailand. A policy to protect workers and people who may have risk of asbestos exposure is necessary, since diseases related to asbestos are incurable, but preventable.

Keywords: *Asbestos, Asbestosis, Asbestos related disease, Interstitial lung disease*

J Med Assoc Thai 2015; 98 (3): 314-9
Full text. e-Journal: *http://www.jmatonline.com*

Asbestosis is interstitial pulmonary fibrosis caused by inhalation of asbestos[1,2]. Asbestos is a crystalline mineral fiber. It is widely used commercially because of its flexibility, durability, resistance to heat and chemical corrosion and high electrical resistance. Products containing asbestos are used in the shipbuilding, construction and textile- and insulation-manufacturing industries such as flooring, roofing materials, vinyl tile, cement pipe, brake pads, automobile clutches and paint[1,3]. Asbestos fibers can cause malignant (malignant mesothelioma and lung cancer) and non-malignant (asbestosis and asbestos-related pleural diseases such as benign pleural effusion, diffuse pleural thickening and pleural plaque)[1-3]. Asbestosis was previously reported in Thailand based on radiological findings and working history in a fiber cement factory[4]. However there was some concern about the diagnosis because the clinical and radiological findings were non-specific and there was no data support of asbestos exposure in the patient[5]. Histological diagnosis is needed in case of an equivocal of a history of asbestos exposure. The authors report the first case of histologically confirmed diagnosis of asbestosis in Thailand.

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Case Report

A 57-year-old female was referred to Siriraj Hospital for management of interstitial lung disease. She had progressive dyspnea and productive cough for 2 years. She was a non-smoker and had no other

symptoms. She was a farmer and then went to work in an electric wire, mesh fan cover factory 16 years ago. Her duty was checking the quality of protective wire mesh fan covers. She did not use any personal protective equipment. She worked nearby the part of the wire mesh coat where there was a heating process for 10 years until the factory was closed 6 years ago because of financial problems. After that, she sewed the clothes for cleaning lubricant oil at home.

On physical examination, the patient had body temperature 37.4°C, heart rate 110/minute, blood pressure 134/80 mmHg, respiratory rate 22/minute and SpO_2 86% at room air. Heart sounds were regular, with tachycardia and loud P_2. Lung revealed generalized coarse crackles predominating at both lower lung zones. Both legs were edematous. There was no clubbing of fingers. The rest of physical examination indicated no detectable abnormality.

Her complete blood count, blood chemistry, liver function test and urine analysis were within normal limits, except bicarbonate level which was 32 mmol/L. Anti-HIV antibody and antinuclear antibodies (ANA) were negative. The portable CXR (Fig. 1) shows cardiomegaly with diffuse interstitial opacity of both lungs. The prominent of both hili were also found. Sputum stain for acid fast bacilli (AFB) was not found. Pulmonary function test revealed forced expiratory volume in 1 second (FEV_1) 0.60 L (30.4% predicted), forced vital capacity (FVC) 0.67 L (28.4% predicted), FEV_1/FVC ratio 89.6% and total lung capacity (TLC) 23% predicted compatible with severe restriction.

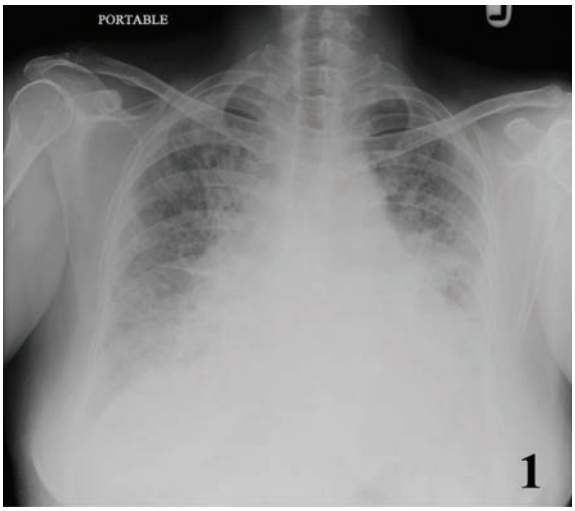

Fig. 1 The portable CXR shows cardiomegaly with diffuse interstitial opacity of both lungs. The left costphrenic angle is obscured by the left silhouette.

The axial view of chest CT scan showed diffuse areas of pulmonary fibrosis, which predominate at both lower lobes (Fig. 2). The evidence of traction

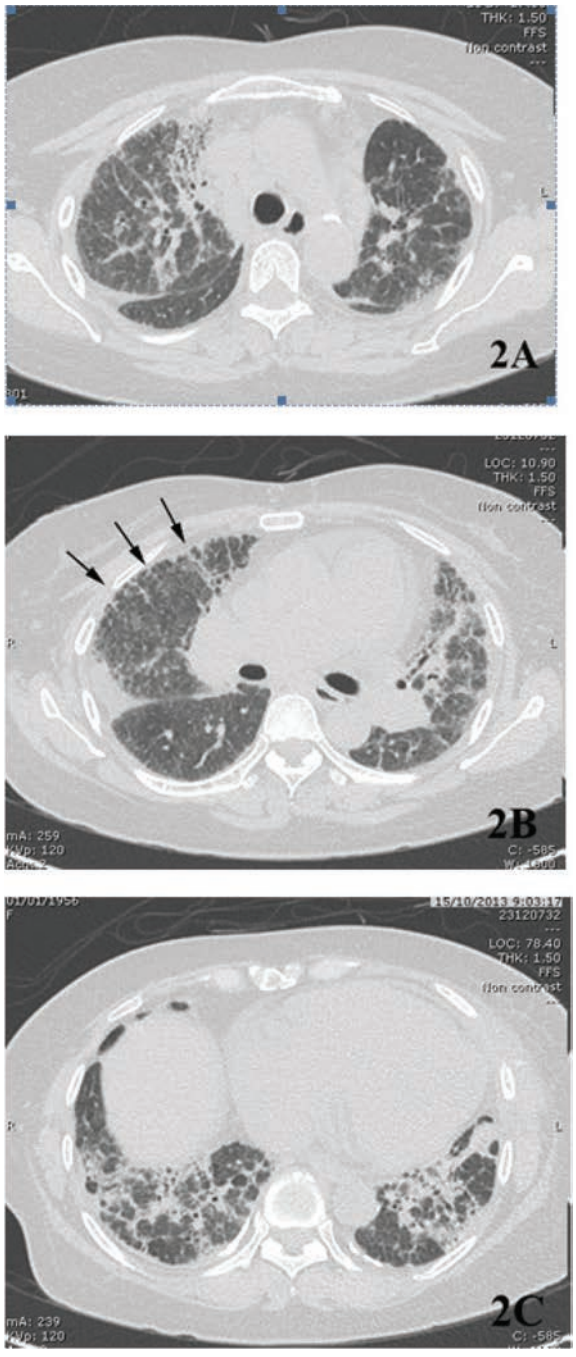

Fig. 2 The serial axial CT scan of the chest (lung window) shows diffuse pulmonary fibrosis at upper (A), middle (B), and lower (C) lung zones. Honeycombing lung is also demonstrated at the peripheral aspect of the right middle lobe (B, arrows).

bronchiolectasis was noted within the area of pulmonary fibrosis. Honeycombing was noted at the anterior aspect of the right middle lobe. Calcified pleural plaques (Fig. 3) were seen bilaterally at lower costovertebral junction. Also noted was enlargement of pulmonary trunk (Fig. 4), measured at about 3.7 cm, which represented some degree of pulmonary hypertension. Multiple mediastinal lymphadenopathies were seen.

The patient underwent fiberoptic bronchoscopy. Bronchoalveolar lavage (BAL) fluid revealed white blood cells 316/mm^3, alveolar macrophages 79%, lymphocytes 16%, neutrophils 4%, and monocytes 1%. BAL fluid cultures for mycobacterium, fungus and bacteria indicated no growth. BAL fluid for cytology was negative for malignancy cells. Papanicolaou-stained smears of BAL fluid displayed many extracellular ferruginous bodies (Fig. 5). The ferruginous bodies showed beaded appearance of hemosiderin coating a thin, translucent core, which is characteristic of asbestos bodies. Transbronchial biopsy was obtained showing only benign, non-diagnostic bronchial tissue without alveolated lung. The patient subsequently underwent thoracotomy with a wedge lung resection at the lingula of left upper lobe. The lung tissue revealed diffuse patchy subpleural intersititial fibrosis with honeycomb appearances, resembling those of usual interstitial pneumonia (UIP) (Fig. 6). The fibrosis displayed mainly dense, hyalinized, eosinophilic collagen. Fibroblastic foci, typical features of UIP, were not distinctly observed. Asbestos bodies were identified within the fibrosis, ranging from 2-5 bodies per high power field (more than 10 per cm^2) (Fig. 7). The asbestos bodies were easily recognized by Perls iron stain, which highlighted the iron-coated particles (Fig. 8). These histopathological findings corroborated diagnosis of asbestosis.

The transthoracic echocardiography revealed RA and RV dilatation, pulmonary hypertension with mean PAP of 37.67 mmHg, systolic D-shaped LV due to RV pressure overload and moderate tricuspid and pulmonic regurgitation. She was diagnosed with asbestosis with cor pulmonale and chronic respiratory failure. She had to be admitted due to worsening of right heart failure and severe hypoxemia. Arterial

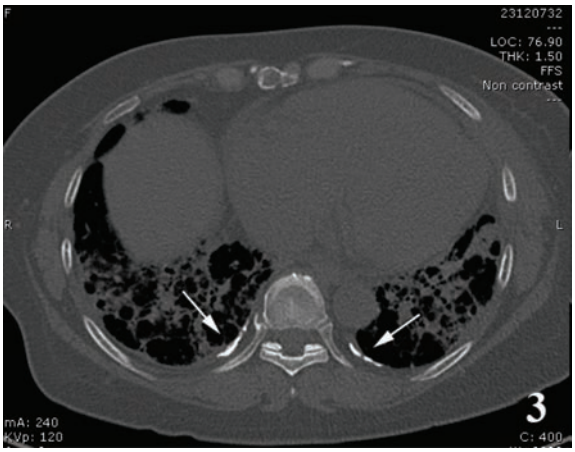

Fig. 3 The axial CT scan of the chest (bone window) shows calcified pleural plaques at bilateral costovertebral junction (arrows).

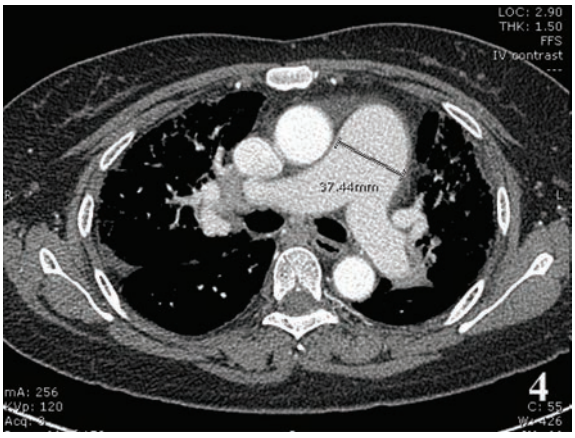

Fig. 4 The axial CT scan of the chest (mediastinal window) shows enlargement of pulmonary trunk, measured about 3.7 cm, which indicates pulmonary hypertension.

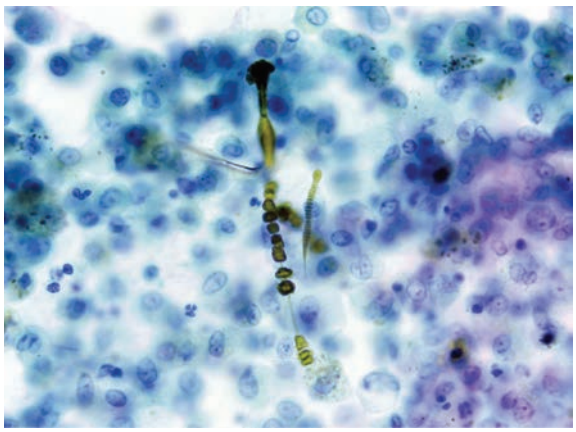

Fig. 5 Pap-stained smear of bronchoalveolar lavage fluid (x600). Asbestos bodies are identified as a thin translucent core of asbestos fiber coated by beadlike, golden brown hemosiderin. They are present extracellularly among background of alveolar macrophages.

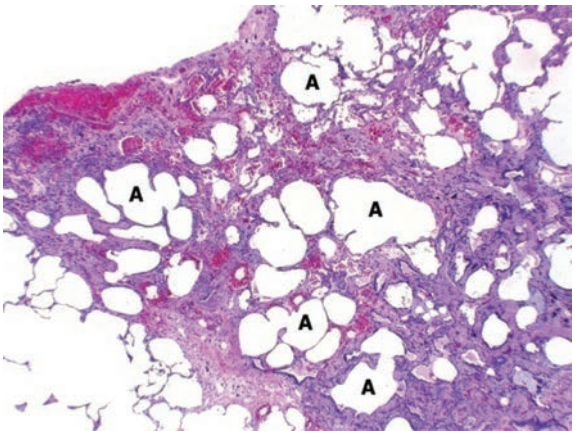

Fig. 6 Low magnification of H&E-stained slide (x40). The lung shows subpleural intersitial collagenous fibrosis with dilated airspaces (A) resulting in honeycomb appearance.

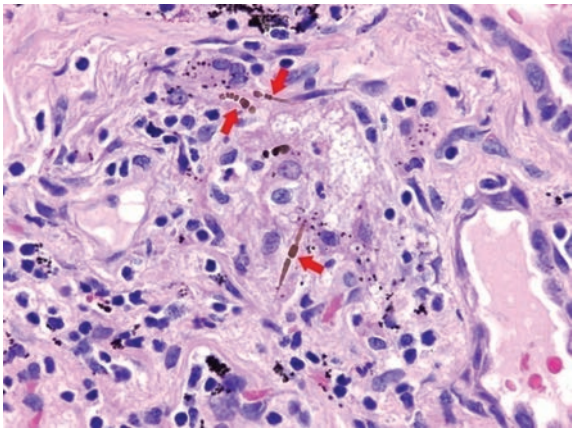

Fig. 7 H&E-stained slide (x600). Three asbestos bodies (arrow)are present in the fibrosis as beaded, golden brown structures with a thin, translucent core.

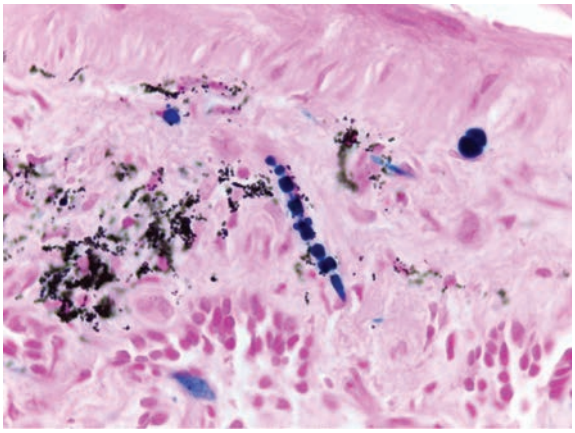

Fig. 8 Perls iron-stained slide (x600). The beaded iron-coated asbestos fiber are highlighted in blue by Perls iron stain.

blood gas analysis showed pH 7.44, pCO_2 51 mmHg, PO_2 35 mmHg and serum bicarbonate 32 mmol/L. She received long-term oxygen therapy and put on waiting list for heart-lung transplantation. The patient information was sent to the regional social security office for the compensation; unfortunately the source of asbestos cannot be investigated since the factory was closed. The patient must continue to suffer from occupational related-asbestosis without any compensation.

Discussion

Asbestosis is defined as interstitial pulmonary fibrosis caused by inhalation of excessive amounts of asbestos fibers. A dose of asbestos in the range of 25 to 100 fibers/ml-years is usually required for the development of asbestosis[2,6]. Most cases of asbestosis are diagnosable as a probability based on clinical and radiologic findings without histologic confirmation. A history of moderate to heavy asbestos exposure and reticular-linear diffuse opacities in the lower zones of the lung fields on radiologic examination are obligatory for the clinical diagnosis which is further supported by findings of parietal pleural fibrous plaques and/or diffuse pleural fibrosis[2]. Histological diagnosis of asbestosis is most useful when the clinical or radiologic features are atypical or non-diagnostic such as an equivocal of the history of asbestos exposure. Pathologic diagnosis of asbestosis requires a characteristic pattern of fibrosis (UIP-like pattern) and the finding of 2 or more asbestos bodies per cm^2 in the fibrotic lung[2]. The presented patient worked in the electric wire, mesh fan cover factory where has not been officially reported asbestos use in such this type of factory. However, the diagnosis of asbestosis was confirmed by pathologic findings of UIP-like pulmonary fibrosis with asbestos bodies more than 10 per cm^2 in the fibrosis.

Asbestosis manifests as progressive dyspnea, interstitial lung disease (ILD) with restrictive pattern on pulmonary function test. Clinical manifestation and radiologic findings of asbestosis are non-specific and must be distinguished from other ILD such as hypersensitivity pneumonitis, non-specific interstitial pneumonitis (NSIP), usual interstitial pneumonia (UIP) in connective tissue disease, drug-related fibrosis, sarcoidosis, idiopathic pulmonary fibrosis (IPF) and other pneumoconiosis[1,2,7]. Pleural plaques on radiologic findings provide useful corollary evidence that the parenchymal process is asbestos related[1].

Asbestosis is usually associated with prolonged exposure over 10 to 20 years and develops after initial exposure for 15 years or more[1,2,7]. However, asbestosis can develop early in the case of high exposures to asbestos, lasting from several months to 1 year or more[1,2], which such case shows small fibrotic lungs without honeycomb changes and is found numerous asbestos bodies in histological examination[2]. The pathologic examination in the presented patient demonstrated honeycombing suggesting the chronicity. The most likely source of asbestos is the protective mesh wire factory, since the patient worked in the suspected source of asbestos for 10 years, beginning 16 years ago and ending 6 years ago before the diagnosis of asbestosis. This latency period is usually found in asbestos related diseases[1,2]. In addition, none of her other jobs can explain clinical manifestations. Unfortunately, this hypothesis cannot be proved since the factory was closed. It may be helpful if surveillance at another same type factory can be done.

Thailand is the world's fifth and seventh largest asbestos user in 2009 and 2011, respectively[8]. However, asbestos related diseases are only found in sporadic case reports. The first case of pleural mesothelioma in Thailand had reported by Bovornkitti et al since 1954[9] followed by other cases of mesothelioma; however, none of them showed pathological evidence of asbestos etiology and only one of 57 patients had history of asbestos exposure in a factory[10]. Recently, Subhannachart et al[4] reported two cases of asbestos-related diseases: one malignant mesothelioma and the other asbestosis in patients who worked at a fiber, cement factory production line and as a mechanic, respectively. However, there was some doubt about these diagnoses because of no data of asbestos exposure in these patients and no pathological confirmation[5]. In the case of controversy, histopathological diagnosis of asbestosis is needed.

The authors report the first case of pathologically confirmed asbestosis in Thailand. This confirmed that asbestosis exists in Thailand. Asbestosis (including other asbestos-related diseases) may be under detected and under diagnosed. The diagnosis is not easy, needing both multidisciplinary team approaches and costly investigation. The authors are concerned about workers who work in factories or enterprises, which do not report asbestos use. These may be more dangerous than known asbestos-use factories. Physicians should be aware of occupational lung diseases and should inquire about occupational history in patients who present with interstitial lungs. In addition, a policy to protect workers and people who may be at risk of asbestos exposure is necessary, since diseases related to asbestos are incurable, but preventable.

What is already known on this topic?

Asbestosis is caused by asbestos.

What this study adds?

This case report is the first definite diagnosed asbestosis in Thailand, which confirms that asbertosis is exist in Thailand. Asbestosis including asbestos related diseases such as mesothelioma are debated in Thailand. The diagnosis is needed objective measurement amount of asbestos exposure or histology. Previous case reports were no definite diagnosis.

Potential conflicts of interest

None.

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โรคแอสเบสโตสิสวินิจฉัยจากผลพยาธิวิทยา: รายงานผู้ป่วยรายแรกในประเทศไทย

เบญจมาศ ช่วยชู, นิชฌา เหลืองด่านสกุล, นิธิพัฒน์ เจียรกุล, รุจิรา เรืองจิระอุไร, กาญจนา อมรพิเชษฐ์กูล, ปุณณฤกษ์ ทองเจริญ, นิศา เมืองแมน

โรคแอสเบสโตสิส เป็นโรคปอดจากการประกอบอาชีพชนิดที่มีพังผืดในเนื้อเยื่อปอดจากแอสเบสตอส ก่อนหน้านี้ได้มีรายงานโรคแอสเบสโตสิสในประเทศไทย โดยอาศัยภาพถ่ายรังสีทรวงอกและประวัติการทำงานในโรงงานผลิตกระเบื้องมุงหลังคาที่มีการใช้แอสเบสตอส แต่เนื่องจากอาการทางคลินิกและลักษณะภาพถ่ายรังสีทรวงอกไม่มีความเฉพาะเจาะจง ร่วมกับไม่มีข้อมูลที่ยืนยันว่าผู้ป่วยได้รับแอสเบสตอสจากที่ทำงาน ทำให้มีข้อโต้แย้ง ซึ่งในรายที่สงสัยการวินิจฉัยควรทำการตรวจยืนยันทางพยาธิวิทยา คณะผู้นิพนธ์ได้รายงานผู้ป่วยที่มาตรวจ ด้วยอาการเหนื่อยมา 2 ปี มีประวัติทำงานในโรงงานผลิตตะแกรงครอบพัดลมโดยทำหน้าที่ตรวจสอบซี่ตะแกรงพัดลมว่าเชื่อมติดกันดีเป็นเวลานาน 10 ปี จนโรงงานเลิกกิจการไปนาน 6 ปี ก่อนมาพบแพทย์ ซึ่งโรงงานประเภทนี้ไม่มีรายงานการใช้แอสเบสตอสอย่างเป็นทางการมาก่อน จากการตรวจร่างกายและภาพถ่ายรังสีทรวงอก ผู้ป่วยได้รับการวินิจฉัยโรคพังผืดในปอด ต่อมาผู้ป่วยได้รับการผ่าตัดเนื้อเยื่อปอดเพื่อช่วยในการวินิจฉัยสาเหตุของโรค ผลการตรวจทางพยาธิวิทยาพบว่ามีพังผืดในปอดร่วมกับตรวจพบแอสเบสตอสในเนื้อเยื่อปอดมากกว่า 10 ต่อตารางเซนติเมตร จึงได้รับการวินิจฉัยโรคแอสเบสโตสิส ผู้ป่วยมีอาการเหนื่อย ภาวะพร่องออกซิเจนอย่างรุนแรง ร่วมกับภาวะหัวใจด้านขวาล้มเหลว และรอรับการปลูกถ่ายหัวใจและปอด รายงานนี้จึงเป็นการยืนยันว่าโรคแอสเบสโตสิสมีอยู่จริงในประเทศไทย จึงควรมีนโยบายที่ป้องกันไม่ให้ผู้ที่ทำงานหรือประชาชนมีความเสี่ยงต่อการสัมผัสแอสเบสตอส ซึ่งเมื่อเป็นโรคแล้วไม่มีการรักษาให้หาย แต่เป็นโรคที่สามารถป้องกันได้