A Huge Lung Mass and Fever in a 36-Year-Old Man

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Siriraj Med J 2008;60:96-101
E-journal: http://www.sirirajmedj.com

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Dr. Praditsuwan: A 36-year-old Thai man from Chumphon Province had been admitted at our hospital from 31 August to 1 October 2005. His chief complaint was progressive dyspnea for 1 week. Three months prior to admission he developed discomfort and pain at his left chest wall together with exertional dyspnea and occasional non-productive cough. He also lost 5 kg. during this period. No fever was noted. After having this ongoing illness for 1 month, he went to Chumphon Hospital. An abnormality of the lung was observed in a chest x-ray. Although acid fast bacilli in the sputum were not found, he had been treated with 4 anti-tuberculous drugs for more than 1 month. However, the illness was not improved. Two weeks prior to admission, in addition to the mentioned symptoms, jaundice, occasional cough producing sputum with blood streaks and low-grade fever were observed. At Siriraj hospital, an enlarged left supraclavicular lymph node was detected and a left upper lung mass was shown in chest x-ray. Acid fast bacilli in the sputum were not found on 3 consecutive days. Liver function tests revealed total bilirubin 3 mg/dL (0.3-1.2), direct bilirubin 2.9 mg/dL (0-0.5), AST (SGOT) 99 U/L (0-37), ALT (SGPT) 76 U/L (0-40), alkaline phosphatase 316 U/L (39-117) and GGT 76 U/L (7-50). A lymph node biopsy was performed and he was told to stop taking the anti-tuberculous drugs. While waiting for the pathological diagnosis of the lymph node biopsy (which turned out to be only reactive change), he developed hoarseness of voice, edema of his left arm and both legs, more dyspnea with orthopnea, high-grade fever and more cough. On the day of his second visit for follow-up, all of these symptoms were worsening and hypotension was detected (blood pressure of 90/50 mmHg), so he was admitted.

In the past, he had never been so ill that required any admission. He earned his living by working in an orchard for the last 7-8 years. He had been divorced for 3 years. His only child was 5-year-old and healthy. He used to smoke 3-4 cigarettes/day for 18 years; he had quit smoking for 3 years. He drank alcoholic beverages at times. His father died of liver cancer; otherwise there was not any significant family history. Except for the recent medications for treatment of tuberculosis, he had never taken any drug. No history of drug hypersensitivity was noted.

Physical examinations at the medical ward revealed a temperature of 38.0°C, a respiratory rate of 30/min, a heart rate of 130/min (regular) and a blood pressure of 120/80 mmHg. Significant findings included mild respiratory distress, mild pallor, mild jaundice, pitting edema (2+) at his left arm and both legs, clubbing of fingers, dilated superficial veins at the anterior chest wall, trachea shifting to the right, decreased chest movement at the left upper portion together with percussion dullness, decreased breath sound and decreased vocal resonance, fine crepitation at the left lower lung field, hepatomegaly (3 fingerbreadths below right costal margin) and enlargement of his left supraclavicular and left axillary lymph nodes with hard consistency (2 and 3 cm. in maximal diameters, respectively). Laboratory investigations included hemoglobin 7.7 g/dL, hematocrit 24.6%, MCV 88.5 fL, wbc 50,200/ cu.mm., neutrophils 89%, band forms 5%, lymphocytes 6%, platelets 209,000/cu.mm., BUN 8 mg/dL (7-20), creatinine 0.6 mg/dL (0.5-1.5), fasting blood sugar 96 mg/dL (76-110), serum sodium 127 mmol/L (135-145), potassium 3.8 mmol/L (3.5-5.0), bicarbonate 18 mmol/L (21-34), chloride 99 mmol/L (105-115), total calcium 7.6 mg/dL (8.1-10.4), phosphate 3.6 mg/dL (2.2-5.0), total bilirubin 1.9 mg/dL (0.3-1.2), direct bilirubin 1.5 mg/dL (0-0.5), AST (SGOT) 61 U/L (0-37), ALT (SGPT) 24 U/L (0-40), alkaline phosphatase 453 U/L (39-117), GGT 310 IU/L (7-50), albumin 1.7 g/dL (3.5-5.5), globulin
4.0 g/dL (1.5-3.5), cholesterol 87 mg/dL (100-200), triglyceride 179 mg/dL (50-200), HDL 12 mg/dL (35-100) and LDH 505 U/L (225-420). Urinalysis revealed pH 6.0, specific gravity of 1.015, wbc 0-1/HP and rbc 0-1/HP. Chest x-ray demonstrated haziness at his left upper lung field and patchy infiltration at his left lower lung field.

The problem list in this patient was shown. (Table 1) Since there are many problems, I will use the key feature approach. First, about the left upper lobe mass, (Fig 1) one needs to consider whether it is neoplastic or infectious in nature. Based on the solitary large lesion, primary lung cancer is the most likely. Primary malignant lymphoma of the lung with involvement of both supraclavicular and axillary lymph nodes is another possibility. From the chest x-ray findings, the mediastinum may also be involved. Mediastinal germ cell tumor should be considered in this patient whose age was only 36. Other differential diagnoses include infectious process and other uncommon entities such as benign tumor, inflammatory mass, vascular mass and other extrapulmonary masses. Lung abscess is possible and one should consider fever. However, the location of his left upper lobe is unusual for lung abscess following aspiration pneumonia. In addition, fever occurred later in the clinical course of this particular case. Pulmonary tuberculosis may present as mass (tuberculoma) but the size here is too big. Moreover, no clinical improvement was noted despite anti-tuberculous therapy for at least 1.5 months. Clubbing of fingers has been described in malignant tumor of the lung, infectious lesions, and mediastinal mass.1

Hard consistency of the lymph nodes highly suggests malignancy. Metastasis to axillary lymph node is quite uncommon for CA lung so that lymphoma is more likely to be the case just based on this aspect. Regarding pleural effusion, pulmonary infiltration and fever for 2 weeks, primary lung cancer with obstructive pneumonitis is considered. Pleural effusion is most likely to be exudate. Hoarseness of voice implies that the mass involved the left recurrent laryngeal nerve. Edema of the left arm and both legs can be caused by malignancy that may locally occlude the venous return or lymphatic ducts. Associated deep vein thrombosis of localized vessels can also occur. Hypo-albuminemia partly plays role in pleural effusion and leg edema.

Hematologic abnormalities, including anemia and neutrophilia, may be caused by inadequate erythropoiesis (secondary to malnutrition and chronic disease) and reaction to infection, respectively. Data prior to this illness about any underlying cause of neutrophilia such as chronic myeloid leukemia or myeloproliferative disorder are not available. Other problems seem to be responsible for the late consequences. As an internist, I think this patient had a primary lung malignancy with lymph node metastasis and its consequences including pleural effusion and obstructive pneumonitis. Next, I would like to ask Dr. Sompradeekul, a chest physician, to approach the problems in this patient.

Table 1. Problem list

<table>
<thead>
<tr>
<th>Problem list</th>
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<tr>
<td>Progressive left-sided chest tightness, dyspnea, cough, weight loss</td>
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<td>for 3 months; not responding to anti-TB treatment</td>
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<td>LUL mass with pleural effusion and LLL infiltrates</td>
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<td>Jaundice for 2 weeks</td>
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<td>Fever for 2 weeks</td>
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<tr>
<td>Hoarseness of voice for 1 week</td>
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<tr>
<td>Edema of left arm and both legs for 1 week</td>
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<td>Hypotension for 1 day</td>
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<tr>
<td>Clubbing of fingers</td>
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<tr>
<td>Hepatomegaly</td>
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<tr>
<td>Markedly increased alkaline phosphatase and GGT</td>
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<tr>
<td>Left supraclavicular and left axillary lymph node enlargement</td>
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<td>Anemia, normal MCV</td>
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<tr>
<td>Marked leukocytosis with neutrophilia</td>
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<td>Hyponatremia</td>
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<tr>
<td>Marked hypoalbuminemia, reversed A/G ratio, hypocolesterolemia, low serum</td>
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<td>uric acid</td>
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Fig 1. Huge mass at left upper lobe (chest x-ray). A) Note the shifting of trachea to the right, pulmonary infiltration in the left lower lobe and pleural effusion. B) The mass occupies superior, anterior, posterior, and middle mediastinum.

Dr. Sompradeekul: In fact, my approach is quite similar to the foregoing one given by Dr. Praditsuwan. However, I would like to add on the chest x-ray taken 10 days earlier than the one just shown by Dr. Praditsuwan. (Fig 2) The left upper lobe mass is obviously evident while the degree of pleural effusion and pulmonary infiltration is much less. The trachea has already been displaced to the right by this mass that involves the mediastinum. Therefore the LUL lesion may have existed for a while and the patient developed more symptoms due to increasing effusion leading to this admission. From the CXR findings, the LUL density could have been a tumor mass or a dense consolidation. Decreased breath sounds as well as vocal resonance obtained by physical examination have already indicated that this is a mass lesion – not pneumonia. The mass at this location could represent lung cancer with rib invasion, anterior mediastinal tumor or a large metastatic cancer to the lung. Hard consistency of the lymph node highly suggests metastasis. Clubbing of fingers favors CA metastatic cancer to the lung. Hard consistency of the lymph node highly suggests metastasis. Clubbing of fingers favors CA.
lung over others. Fever is most likely caused by secondary obstructive pneumonia since primary CA lung hardly presents with fever. Anterior mediastinal mass is less likely to be the case because accompanying clubbing of fingers is seldom described. Clinical management needs both investigation for a definite diagnosis and empirical treatment to alleviate the patient’s suffering, especially from obstructive pneumonia. Investigation should include CT scan to determine the nature and extent of the tumor as well as pleural involvement. Left laryngeal nerve lesion should be suspected by tumor as the location of the tumor mass at LUL area would explain the patient’s hoarseness. Tissue diagnosis is certainly needed. Review of the lymph node biopsy and repeat biopsy are recommended should the first biopsy be actually negative for malignancy because it might fail to obtain representative tissue.

Dr. Praditsuwan: Since CA lung is mostly concerned, I would like to ask Dr. Keerativitayanant, an oncologist, to comment on this case.

Dr. Keerativitayanant: Generally, supraclavicular lymph node enlargement is hardly caused by reactive process. Repeat biopsy is mandatory should the result of the first biopsy turn out to be negative because the hard consistency of the lymph node at this site highly indicates malignancy. Solitary lung mass sometimes can be metastatic sarcoma; however, there is not any history of previous mass lesion or any other evidence supporting tumor mass outside the lung. The patient’s age of 36 is not against CA lung since it can occur at times in this age group. This patient used to smoke; both non-small cell lung cancer (NSCLC) and small cell lung cancer can occur in smokers but NSCLC is more common. Clubbing of fingers is also more commonly seen in NSCLC. Metastasis to the axillary lymph node is possible in CA lung. Primary lymphoma of the lung is not commonly seen. Mediastinal germ cell tumor should be considered since this is a case of a mediastinal mass because of a dramatic response to treatment. Investigation for tumor markers is highly recommended, including alpha-fetoprotein (AFP) and beta-hCG (HCG). Superficial vein dilatation over the anterior chest wall should result from left brachiocephalic venous obstruction. I agree with the assumption about secondary obstructive pneumonia being the cause of fever. Lymphoma is common to have fever, but it should occur earlier in the clinical course of the illness.

Dr. Praditsuwan: Based on the hemoculture result and a melioidosis titer of 1:600, intravenous ceftazidime was given. At this time, the results of CT scan were received as follow: inhomogeneous lung mass, 14x12 cm., from root of neck to hilar region; enlarged lymph nodes at posterior chest wall, pectoris major muscle and left axilla; left massive pleural effusion; left brachiocephalic venous obstruction with collateral veins; heterogeneous enhancing of caudate lobe of liver and lobulated outlined mass at the pancreas. No enlargement of intra-abdominal lymph node was noted. Thoracocentesis revealed 300 mL of serosanguinous fluid, wbc 420/cu.mm., lymphocytes 93%; cytology failed to detect any malignant cells. Dr. Sompradeekul, what is your opinion?

Dr. Leelarasamee: We can exclude contaminant and colonizer in any case if _Burkholderia pseudomallei_ (B. pseudomallei) is isolated from blood even from one specimen because both situations are unlikely to occur – that means it was a true pathogen in this case. Once melioidosis is diagnosed, one should look for underlying conditions including diabetes mellitus, malignancy, and occupational risk such as farmer. Melioidosis can be the most common community-acquired infection, especially in the northeastern region of Thailand. Clinical setting is generally diversified and imitates other common bacterial infections. Chest X-ray findings can be similar to those already shown in this patient. In this particular case, sputum examination may be helpful and malignant cells should be searched for. Ceftazidime is the antibiotic of choice and the given antibiotics in this case are effective enough since this patient survived and had been treated for more than one month. Therefore, it is unlikely for melioidosis to be the cause of death – but advanced carcinoma can.

Dr. Sompradeekul: I have reviewed the CT scan with a radiologist and we developed some different findings. (Fig 3) In addition to the huge left upper lobe mass, mediastinum is involved. The left side of the anterior chest wall is much different from the right side by abnormal soft tissue thickening and heterogeneous appearance of the lesion. Enlarged left axillary and left supraclavicular lymph nodes with central necrosis are also seen from the chest CT. Hypodensity areas, indicating tumor necrosis, are observed both in the left upper lobe mass and the enlarged lymph nodes. There is not any obstruction of SVC or main arteries except for the left brachiocephalic venous obstruction already described in the radiological report. There were no abnormalities seen in the liver or pancreas. For tissue diagnosis, one can perform lymph node biopsy or transthoracic needle aspiration or biopsy guided by

![Fig 3. Huge mass at left upper lobe (CT scan). Note the thickening of the left anterior chest wall caused by tumor invasion through the intercostal spaces. The heterogeneous appearance of the tissue is similar to that in the mass and in the left axillary lymph node (arrow). The hypodensity reflects necrosis.](image)

Dr. Praditsuwan: On the fourth admission day, the patient still had fever despite the empirical treatment with intravenous ceftaxione and clindamycin, leading to substitution of piperacillin and tazobactam. Serum tumor markers for AFP and HCG were negative. Bone marrow aspiration and biopsy revealed only granulocytic hyperplasia without any evidence of metastatic tumor. Five days later, the patient developed high-grade fever and signs of acute inflammation over the left anterior chest wall despite some resolution of edema at his left arm and both legs. One specimen of hemoculture yielded _Burkholderia pseudomallei_ (B. pseudomallei). Can this be the cause of fever in this patient or just contamination by the organism? I would like to ask Dr. Leelarasamee to discuss on this issue.

Dr. Leelarasamee: Based on the empirical treatment with intravenous ceftazidime was given. At this time, the results of CT scan were received as follow: inhomogeneous lung mass, 14x12 cm., from root of neck to hilar region; enlarged lymph nodes at posterior chest wall, pectoris major muscle and left axilla; left massive pleural effusion; left brachiocephalic venous obstruction with collateral veins; heterogeneous enhancing of caudate lobe of liver and lobulated outlined mass at the pancreas. No enlargement of intra-abdominal lymph node was noted. Thoracocentesis revealed 300 mL of serosanguinous fluid, wbc 420/cu.mm., lymphocytes 93%; cytology failed to detect any malignant cells. Dr. Sompradeekul, what is your opinion?

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ultrasonography or CT. Open lung biopsy is always the last choice. Since the patient had *B. pseudomallei* identified from the hemoculture, one could think of this LUL lesion as a typical presentation of pulmonary melioidosis since radiological findings in melioidosis are varied and sometimes difficult to distinguish from malignancy. Thus, tissue diagnosis is very important in this particular case.

**Dr. Praditsuwan:** On the 16th admission day, antibiotics were changed to intravenous imipenem due to unresolved high-grade fever. Thoracotomy with decortication of pleura at the left lower lobe and open drainage of the left upper lobe was performed. Five days later, the patient did not have any fever; oxygen was given via mask with reservoir bag due to some ongoing dyspnea. The pathology result showed extensive necrosis with presence of atypical large cells. On the 26th admission day, the patient’s consciousness deteriorated and more dyspnea was noted. Endotracheal intubation along with ventilator was introduced for respiratory support. Another lung node biopsy was then performed on the following day and metastatic undifferentiated carcinoma was confirmed. On the 30th admission day, repeated episodes of hypoglycemia developed as well as some coffee-ground content in the NG tube and hypotension. Supportive treatment, including 10% dextrose solution, normal saline solution, and dopamine, was given. The patient passed away peacefully on the 32nd admission day.

**Dr. Keerativitayanant:** Repeated episodes of hypoglycemia can occur in tumor producing insulin (such as insulinoma) or insulin-like substance (such as some sarcomas). However, hypoglycemia should occur earlier in the course of disease. In this particular case, this phenomenon should be caused by sepsis and poor liver reserve for glycogen. I still wonder about melioidosis playing some role in the patient’s death.

**Dr. Leelarasamee:** As I mentioned earlier, melioidosis is not a culprit for the cause of death. Advanced carcinoma and, probably, superimposed opportunistic infection are responsible for that. Candidiasis, other fungal infection or even CMV infection may be found at autopsy.

**Dr. Sompradeekul:** After endotracheal intubation for respiratory support, another chest x-ray showed some patchy infiltration in the right lung. This may represent superimposed infection or uncontrolled previous infection which may have caused the patient’s clinical deterioration. Hypoglycemia in this case can be caused by advanced tumor, sepsis, or adrenal insufficiency if there is any metastasis to both adrenal glands (CT scan does not show adrenal lesion). All these causes of hypoglycemia suggest poor prognosis.

**Discussants’ diagnosis**

**Primary CA lung with lymph node metastasis; melioidosis and sepsis probably by opportunistic infection**

**Dr. Chuangsawanich:** This autopsy was performed by Dr. Piyarat Omeapinyan and Dr. Panthep Suttinont; Dr. Omeapinyan is our pathology resident who helped me to prepare this powerpoint presentation as well. The last lymph node biopsy demonstrated sheets of large cells with pleomorphic nuclei; some had multiple nuclei. Many neutrophils infiltrated amidst the tumor cells. No mucin or particular growth pattern was detected. The tumor cells are marked with AE1/AE3 cytokeratins, an epithelial marker, confirming the nature of carcinoma. (Fig 4) The first lymph node biopsy did not have any evidence of metastatic tumor, possibly due to a sampling problem.

At autopsy, pitting edema of the left arm, left axillary and supraclavicular lymph node enlargement, 200 mL of straw-colored right pleural effusion and 1,200 mL of straw-colored ascitic fluid were noted in addition to the huge left upper lobe mass that adhered with the parietal pleura. (Fig 5) The cut surfaces of the lung mass and the enlarged lymph nodes were similar to one another – heterogeneous in texture varying from greyish white to dark reddish brown in color. Multiple areas of hemorrhage and necrosis were noted. The morphology of the tumor was similar to that found in the lymph node biopsy. In addition to the large cells and some multinucleate giant cells, there were some small areas of spindle cells that looked sarcomatoid. Immunostaining for vimentin, a marker for mesenchymal cells, demonstrated many more vimentin+ tumor cells than cytokeratin+ ones. Only rare tumor cells mark with HHF-35, a marker for muscle specific actin. This additional information indicated that this is a giant cell variant of sarcomatoid carcinoma. There were liquefactive necrotic areas and a definitely walled-off abscess at the apex of the left upper lobe.

At the apex of the right upper lobe, a 2 cm. mass with central necrosis was noted; it was not radiographically mentioned during admission. Microscopically, in addition to those described in the left upper lobe mass, sheets of tumor cells with definite gland formation and mucin+ tumor cells were shown; thus confirming a more differentiated component of adenocarcinoma. (Fig 6) For academic interest, we performed an EM study from a formalin-fixed paraffin-embedded tissue block of the last lymph node

**Fig 4.** Metastatic undifferentiated carcinoma in the lymph node biopsy. A) Many large cells show loose cohesiveness. No specific growth pattern is noted. B) Large tumor cells have pleomorphic nuclei and occasional binucleation. Many neutrophils admix with tumor cells. C) A number of tumor cells mark with AE1/AE3 cytokeratins, confirming the nature of carcinoma.

**Fig 5.** Primary carcinoma of the lung. A) The heart-lung en bloc showing tumor destroying left upper lobe. Note metastatic tumor to left supraclavicular lymph node (arrow) and invasion of the surrounding tissue by tumor. A walled-off abscess is also noted at the apex of left lung (arrow head). B) Multinucleate giant tumor cells admix with neutrophils and macrophages. C) Spindle-shaped tumor cells are focally noted. D) Vimentin+ tumor cells are noted.
biopsy that was originally diagnosed as metastatic undifferentiated carcinoma. We found perinuclear clusters of intermediate filaments corresponding to vimentin in many tumor cells in addition to some microvilli, poorly developed desmosomes, and some tonofilaments. These ultrastructural findings once again confirmed the diagnosis of a giant cell variant of sarcomatoid carcinoma. Since there were giant cells more than 10% and a definite area of poorly differentiated adenocarcinoma, the diagnosis in this case according to the current WHO classification should be pleomorphic carcinoma, a subgroup of sarcomatoid carcinoma.7 We thank Associate Professor Klephant Thakerngpol for the EM study. Tumor metastasis was confirmed in the left supraclavicular and axillary, mediastinal, subcarinal, and para-aortic lymph nodes, both adrenal glands (especially the left one), a small focus at the lower pole of the right kidney, and small hemorrhagic foci in the cerebral cortex (areas that did not produce any localizing sign). The latter exhibited clearly isolated large tumor cells amidst numerous neutrophils, similar to the histologic features of giant cell carcinoma of the lung.

Hemoculture taken from heart blood and culture from the lung abscess yielded a few colonies of *B. pseudomallei*. We discovered some tentative bacteria in the histologic sections stained for bacteria (Brown-Hopps stain and Warthin-Starry stain) which compared to the *B. pseudomallei* colony obtained from the other case. We also performed an EM study to seek for bacteria in sinus histiocytes from the spleen and macrophages in the lung abscess to compare with the known *B. pseudomallei* infected in macrophages from a previous experimental study of melioidosis by Dr. Sunee Korbrisate and colleague6 for comparison. The size of *B. pseudomallei* from that experiment varied from 310-680 nm. in diameter and 420-1,520 nm. in length.7 The average size of bacteria recovered from this patient was 510 nm. in diameter and 1,330 nm. in length. We also asked Dr. Chanwit Tribuddharat from our microbiology department to perform a PCR for detection of 16S rRNA gene amplification of *B. pseudomallei*. Unfortunately, no amplification product was recovered from formalin-fixed tissue taken from the lung abscess, spleen and liver. I would like to thank Dr. Tribuddharat for his kind help and to ask him to comment on this issue.

**Dr. Tribuddharat:** Formalin-fixed tissue certainly hampers the amplification product because the quality of recovered DNA is suboptimal. In this situation, negative PCR result cannot exclude the possibility of disseminated melioidosis.

**Dr. Chuangsuwanich:** The other findings included some degree of hemosiderosis of liver with mild hepatomegaly and spleen of normal weight, leukemoid reaction (granulocytic hyperplasia of the bone marrow) and early antibiotic-associated pseudomembranous colitis. There was not any opportunistic infection detected. [Editorial note: The infiltration of all tumor areas by neutrophils highly suggests that the tumor cells may induce neutrophilic response; there have been studies showing tumor cells secreting granulocyte-colony stimulating factor (G-CSF) or other cytokines attracting neutrophils. This explains leukemoid reaction as one of paraneoplastic syndrome in malignancy. This phenomenon was also observed in a CPC case previously published in Siriraj Medical Journal.]

**Pathologic Diagnosis:**
- Sarcomatoid (pleomorphic) carcinoma of the lung with:
  - Predominantly giant cell component involving the entire left upper lobe and a portion of left lower lobe
  - Poorly differentiated adenocarcinomatous component at right upper lobe (2 cm. in diameter)
- Direct extension into anterior and middle mediastinum and thymus
- Invasion of the anterior chest wall to the overlying skin
- Metastasis to multiple lymph nodes at left supraclavicular area (7x6x4 cm.), left axillary, mediastinal, subcarinal and para-aortic areas, left diaphragm (2 cm. in diameter), both adrenal glands, right kidney (0.2 cm. in diameter), and brain (multiple small foci)
- Melioidosis (suppurative inflammation and positive postmortem left upper lung tissue culture)
- Early antibiotic-associated pseudomembranous colitis
- Hemosiderosis of liver and spleen
- Leukemoid reaction

**Dr. Leelarasamee:** Based on the autopsy findings, only localized melioidosis is left as a lung abscess. The only few colonies of *B. pseudomallei* found in the postmortem cultures implies at least some effectiveness of the given antibiotics to control melioidosis. The tentative bacteria in the splenic sinus macrophage is doubtful for *B. pseudomallei*; hence ongoing bacteremia by this organism is not confirmed. About the size of bacteria in the EM study, it cannot be used to determine the type of bacteria unless we apply an antibody specific for *B. pseudomallei* to confirm melioidosis in this particular case.

**Dr. Panutsaya Tientadakul** (Department of Clinical Pathology): In the case of increased serum alkaline phosphatase alone, GGT will be helpful to distinguish the hepatic cause from the others such as bony cause. However, there is no need to order GGT at all in this patient who had already had jaundice.

**Dr. Parvinee Suwanagool** (Head, Department of Pathology at that time): Today we have learned a great deal from this CPC. There are 275 medical students participating in submission of their diagnoses. A majority of them concern various types of malignancy. We have found one medical student who gave the diagnosis of primary CA lung with metastasis to lymph nodes and other organs and accompanying melioidosis. That is the closest diagnosis!

**Keywords:** Pleomorphic carcinoma; sarcomatoid carcinoma; giant cell carcinoma; lung cancer; neutrophilia; leukemoid reaction; melioidosis; cytokeratins; vimentin; EM
REFERENCES


