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CASE STUDY

A case of superior vena cava syndrome caused by *Klebsiella pneumoniae*

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A case of superior vena cava syndrome caused by Klebsiella pneumoniae. *J.Y. Kim, C-M. Lim, Y. Koh, K.H. Choe, W.S. Kim, W.D. Kim. ©ERS Journals Ltd 1997.*

ABSTRACT: A 27 yr old man presented with productive cough, fever and manifestations of superior vena cava syndrome. He was an alcoholic but had been in good health until 3 days prior to admission.

The physical examination, the chest radiograph and the results of the sputum culture were compatible with *Klebsiella pneumoniae* pneumonia of the right upper lobe. The superior vena cava scintigram using technetium-99m showed near total occlusion of the superior vena cava, while sputum cytology, chest computed tomography, and bronchoscopy were all negative for malignant aetiology.

Antibiotic therapy brought about slow resolution of the pneumonia and also of the superior vena caval obstruction. The follow-up scintigram showed normalized venous flow of the superior vena cava. To our knowledge, this is the first case of superior vena cava syndrome developed in probable association with *Klebsiella pneumoniae* pneumonia.

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Superior vena cava syndrome develops when the venous return of blood from the head and upper thorax to the heart is interrupted. Malignant tumours are the most frequent cause of this syndrome [1]. However, the obstruction can result from a benign process in a small number of patients. Among benign aetiologies are tuberculosis, retrosternal thyroid, aortic aneurysm and thymoma [2]. We present the case of a patient with *Klebsiella pneumoniae* pneumonia of the right upper lobe, complicated by superior vena cava syndrome. Antibiotic therapy resulted in resolution of not only the pneumonia but also the superior vena cava syndrome.

Case report

A 27 yr old man presented to the Asan Medical Center with facial flushing and shortness of breath. He had been in good health until 3 days before admission, when he noticed fever, cough productive of thick, red phlegm, and pleuritic chest pain. He had a history of 10 pack-year cigarette smoking and used to drink 5 days per week, consuming on average 50–75 g of alcohol each time.

The vital signs at presentation were: blood pressure 110/70 mmHg; cardiac frequency 120 beats·min^{-1}; respiratory frequency 30 breaths·min^{-1}; and temperature 38°C (100.4°F). On initial physical examination, the patient appeared acutely ill-looking, with the face and both arms oedematous. In the sitting position, the jugular veins were distended up to the angle of the jaw. Chest auscultation disclosed decreased breath sounds and increased vocal fremitus on the right upper lung field. There was no organomegaly, lymphadenopathy or clubbing of the digits.

The initial laboratory evaluation showed: white blood cell 10,400 cells·mm^{-3} (5% metamyelocytes, 48% band forms, 38% segmented neutrophils, 5% lymphocytes); haemoglobin 12.6 g·dL^{-1}; platelet 40,000 platelets·mm^{-3}; erythrocyte sedimentation rate 85 mm·h^{-1}; prothrombin time 13.5 s (normal control 12.4 s); activated partial thromboplastin time 38.0 s (normal control 32.4 s); and fibrinogen 1,114 mg·dL^{-1}. Fibrinogen degradation products were negative. The results of arterial blood gas analysis were: pH 7.46; arterial carbon dioxide tension (P_{a,CO_2}) 3.9 kPa (30 mmHg); arterial oxygen tension (P_{a,O_2}) 9.4 kPa (70 mmHg); bicarbonate 21.4 mEq·L^{-1}; and arterial oxygen saturation (S_{a,O_2}) 95% on room air.

The initial chest radiograph showed homogenous airspace consolidation of the right upper lobe and part of the right lower lobe with contralateral displacement of the trachea, but air-bronchogram was not observed (fig. 1a). Computed tomographic scan (CT) of the chest showed mediastinal involvement of the necrotizing pneumonia, and poor enhancement of the right brachiocephalic vein due to surrounding inflammation (fig. 1b). Antibiotic therapy with second-generation cephalosporin and aminoglycoside was initiated under the suspicion of community-acquired pneumonia after specimens were taken from blood and sputum for microbiological studies.

In order to confirm the clinical diagnosis of superior vena cava syndrome, superior vena caval scintigraphy was performed with technetium-99m. It showed stasis of the right brachiocephalic vein at its proximal portion with reflux flow into the right internal jugular vein and faintly visible superior vena cava *via* a collateral vein (fig. 2a). The Gram stain of the expectorated sputum revealed Gram-negative bacilli, which were later identified to be *Klebsiella pneumoniae*.

With antibiotic therapy, the patient showed slow but steady clinical improvement. Bronchoscopic examination was negative for any endobronchial lesion. Follow-up superior vena caval scintigraphy performed 20 days later showed normalized drainage of the right brachiocephalic vein into the superior vena cava (fig. 2b). The total duration of the antibiotic therapy was 29 days, and

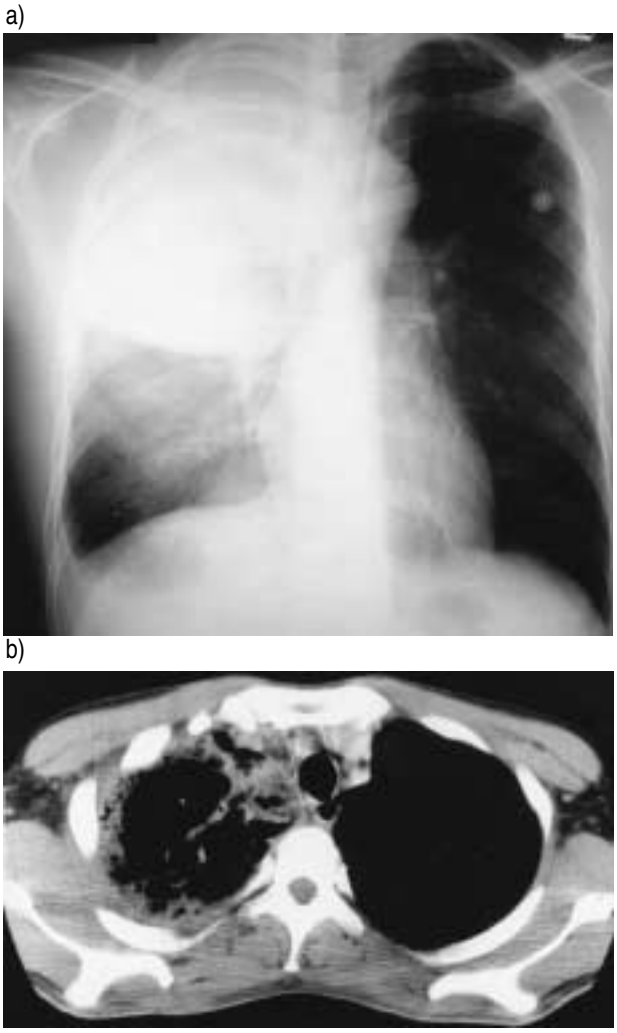

Fig 1. – a) Chest radiograph of the patient showing lobar consolidation of the right upper and partly lower lobe with bulging fissure. b) Computed tomographic scan showing necrotizing lobar pneumonia involving the superior mediastinum, and poor enhancement of the right brachiocephalic vein.

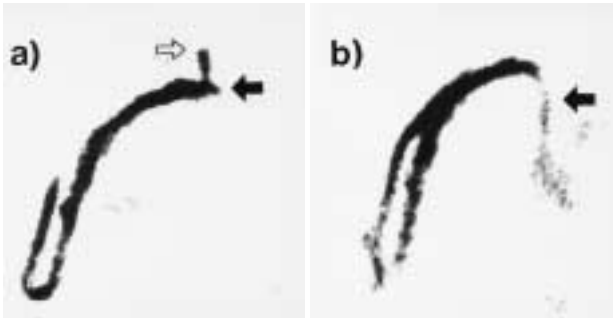

Fig 2. – a) Initial scintigraphy of the superior vena cava showing stasis of the right brachiocephalic vein at its proximal portion (closed arrow) with reflux flow into the right internal jugular vein (open arrow). b) Follow-up scintigraphy 20 days after hospital admission showing normalized drainage into the superior vena cava (closed arrow).

there was no other treatment for the superior vena cava syndrome. At 8 months of follow-up, the patient was in good health, without signs of superior vena caval obstruction, and chest radiography showed no active lesion other than some residual fibrosis.

Discussion

Superior vena cava syndrome is usually related to a malignant process, but many benign causes have also been described. The superior vena cava is a thin-walled, low-pressure system that is vulnerable to compression. Characteristic symptoms and signs may develop quickly or gradually when this thin-walled vessel is compressed, invaded, or thrombosed by various processes in the superior mediastinum.

The diagnosis of superior vena cava syndrome is usually based on clinical signs. The conscious patient may complain of hoarseness, shortness of breath, coughing, and visual disturbance, and advanced cases may show alteration in consciousness. Physical findings are proportional to the rapidity of development of the syndrome, and include dilatation of collateral veins and oedema of the upper chest, arms and conjunctivae. As might be expected, the most common radiographic abnormality is widening of the superior aspect of the mediastinum [1].

There have been reports of superior vena caval obstruction caused by pulmonary and mediastinal infection, such as tuberculosis [3], histoplasmosis [4], and nocardiosis [5]. In these patients, appropriate medical therapy led to resolution of the infection and complete relief of the symptoms of superior vena caval obstruction. The present case of superior vena cava syndrome of acute onset presented with lobar pneumonic consolidation of the right upper lobe. Malignant aetiologies were excluded by means of CT of the chest and bronchoscopy. The Gram stain and culture of the expectorated sputum revealed *Klebsiella pneumoniae*. Antibiotic therapy resulted in complete resolution of the consolidation and disappearance of the symptoms and signs of the superior vena cava syndrome. We suspect that the superior vena caval compression in this patient might have been caused by accompanying subclinical mediastinitis, as evidenced by the findings of the right upper lobe on CT (mediastinal involvement of the necrotizing pneumonia, poor enhancement of the right brachiocephalic vein due to surrounding inflammation).

To our knowledge, there have been no reports of cases of superior vena cava syndrome caused by *Klebsiella pneumoniae*. In the present case the superior vena cava syndrome was probably caused by *Klebsiella pneumoniae* pneumonia, which disappeared completely after treatment of the pneumonia with antibiotics.

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