

CASE STUDY

Superior vena cava syndrome caused by encapsulated pleural effusion

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Superior vena cava syndrome caused by encapsulated pleural effusion. C-L. Lai, T-T. Tsai, S-C. Ko, K-Y. Yang, R-P. Perng, Y-M. Chen. ©ERS Journals Ltd 1997.

ABSTRACT: Pleural effusion is often a manifestation of the superior vena cava (SVC) syndrome. However, pleural effusion has never been reported to be a cause of the SVC syndrome.

We report the case of a 68 yr old male patient who presented with SVC syndrome and respiratory failure, both attributable to an encapsulated pleural effusion over the right upper mediastinum. Simple drainage was performed as a diagnostic and therapeutic procedure.

The pleural effusion was confirmed to be a tuberculous empyema. Chest computed tomography (CT) scan is the most appropriate initial diagnostic procedure for superior vena cava syndrome.

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Nonmalignant aetiologies account for approximately 12% of all cases of superior vena cava (SVC) syndrome, and have been comprehensively reviewed [1–6]. However, SVC syndrome resulting from an encapsulated pleural effusion has never been reported. We report a case of SVC syndrome due to an encapsulated pleural effusion over the right upper mediastinum.

Case report

A 68 yr old man was admitted to our hospital because of progressive orthopnoea. He had been well until a week prior to admission, when he experienced right-sided upper back pain. Shortly afterwards, he experienced exertional dyspnoea, which progressed to intolerable orthopnoea within a week, prompting an emergency admission.

Physical examination in the emergency room showed that the patient was in respiratory distress. He was afebrile. His face was puffy and flushed, and the neck and upper trunk were swollen, but displayed no visible signs of a collateral circulation. There was no palpable cervical lymphadenopathy. No lower leg oedema were present. Chest radiography showed cardiomegaly, right-sided pleural effusion, widening of the upper mediastinum, and left-sided displacement of the trachea (fig. 1). Chest computed tomography (CT) scan disclosed a right-sided encapsulated pleural effusion, compressing the superior vena cava (SVC), lower trachea, and main bronchi (fig. 2). There was no evidence of pericardial effusion, mediastinal or hilar lymphadenopathy, or dissecting aneurysm.

Diagnostic and therapeutic drainage of the effusion was performed and 1.5 L of bloody effusion was removed.

Cell counts showed 60×10^9 red blood cells $\cdot L^{-1}$, and 2.8×10^9 white blood cells $\cdot L^{-1}$, with a neutrophil lymphocyte ratio of 78/22. The patient was hospitalized. On the day after admission, he developed respiratory distress necessitating mechanical ventilation. A pigtail catheter was inserted to drain the pleural effusion. Electrocardiographic changes were noted 24 h after resuscitation, and subsequent elevation of cardiac enzyme confirmed the occurrence of an acute myocardial infarction. The patient recovered uneventfully from the cardiac event after resuscitation, and was successfully weaned from the ventilator. Symptoms of the SVC syndrome resolved completely after most of the effusion had been drained. Follow-up chest CT scan showed a minimal residual encapsulated pleural effusion, good patency of the SVC and airways and a normal mediastinum (fig. 3).



Fig. 1. – The initial chest radiograph shows severe widening of the upper mediastinum, with displacement of the trachea to the left (arrows), cardiomegaly, and right-sided pleural effusion.

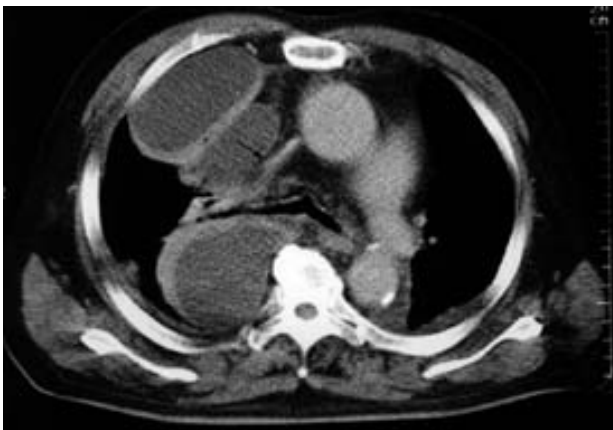


Fig. 2. – The chest computed tomography (CT) scan shows no evidence of dissecting aneurysm but discloses encapsulated pleural effusion in the right mediastinal pleural cavity, which compresses the superior vena cava (arrow), lower trachea and main bronchi.



Fig. 3. – The follow-up chest computed tomography (CT) scan shows minimal residual encapsulated effusion with patency of the superior vena cava (SVC) (arrow), resolution of the airway compression, and normal mediastinum.

Microscopic examination of the pleural effusion failed to reveal bacteria, *Nocardia* or malignant cells, but staining was positive for acid-fast bacilli. Pleural biopsy showed a granulomatous inflammation. The level of adenosine deaminase in the pleural fluid was 121 U·L⁻¹. Moreover, smears of the caseous material obtained from a subcutaneous abscess over the right posterior auricular area revealed 1–9 acid-fast bacilli per high power field.

Antituberculosis medication was prescribed, and the pulmonary condition of the patient improved. Unfortunately, 3 months after discharge, he died from acute congestive heart failure secondary to ischaemic heart disease. There was no evidence of recurrence of the SVC syndrome. An autopsy was refused by the patient's family.

Discussion

The SVC syndrome can result from a multitude of aetiologies, the most common being malignant tumours [1–3, 5–11]. Massive pleural effusions have never been reported to cause SVC syndrome. On the contrary, pleural effusion has been reported to result from SVC

syndrome [2, 9, 12–15], as a consequence of increased hydrostatic pressure and decreased lymphatic return [14]. In a sheep model, by increasing the SVC pressure to 15 mmHg for 24 h, it was possible to induce the formation of pleural effusion [14]. However, this order of events did not seem to be the situation in the present patient, in whom the encapsulated fluid clearly compressed the SVC, lower trachea, and main bronchi (fig. 2). Moreover, after drainage of the effusion, the symptoms of SVC syndrome resolved. Thus, the encapsulated pleural effusion was probably the cause rather than the consequence of the SVC syndrome.

Two conditions are necessary for the SVC syndrome to manifest itself: SVC flow should be compromised; and collateral circulation should not be established. Once collateral circulation compensates for the obstruction of venous flow, symptoms resolve even without relief of the primary obstruction [1, 10]. There are no data indicating how long collateral circulation takes to become established. In the present patient, the rapidity of volume expansion of the encapsulated fluid precluded the formation of an adequate collateral network.

Pleural effusion has rarely resulted in the SVC syndrome. In this patient, local fluid accumulation with rapid volume expansion resulted in a high pressure build-up, mechanically compressing the SVC, the membranous portion of the lower trachea and the main bronchi. Massive pleural effusion culminates in a pressure build-up over the entire mediastinum, whereas the pressure build-up of encapsulated effusion is limited to the upper mediastinum.

SVC syndrome is rarely fatal [10], and the respiratory distress can be attributed to tracheal compression rather than laryngeal oedema [3, 10, 16]. In this patient, intubation was necessary to reverse the upper airway compression documented radiologically.

Tuberculous lymphadenopathy or mediastinal fibrosis used to be the most common nonmalignant causes of SVC syndrome prior to the era of antituberculosis chemotherapy [7]. However, tuberculous empyema has not been reported as a cause of SVC syndrome [17, 18]. Since this patient had neither lymphadenopathy nor mediastinal fibrosis, tuberculous empyema may have resulted in the clinical picture. Treatment of SVC syndrome by drainage and antituberculosis drugs was effective. The definitive diagnosis of tuberculous empyema was made only after relief of the SVC syndrome. This case also documents the value of the CT scan in determining the cause of SVC syndrome. CT scan can detect and exclude other causes of SVC syndrome, such as dissecting aneurysm, tumour mass, lymphadenopathy and goitre. In addition it helps to establish the most appropriate treatment.

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