the left parietal pleura were noted as well as lymphatic neogenesis. The right effusion was not treated, but fluid was not noted. The author did not find any explanation; the same was observed in our patient and we speculate about the role of lymphatic neogenesis.

In animals, thoracic radiation causes, at an early stage, actinic pneumonia and a small pleural effusion; later on, lung changes regress completely and a large pleural effusion appears, probably due to lymphatic block. No histologic proof was presented, but we think our patient reproduced this experimental obstruction.

In 1971, Whitcomb and Schwarz described three patients with pleural effusion several months after thoracic radiation. Based on autopsy, a different mechanism was ascribed to each patient: constrictive pericarditis, cavai obstruction, and mediastinal fibrosis, as in our patient.

Recently Rodrigues-Garcia et al. described a similar patient, with bilateral serohemorrhagic pleural effusion detected 8 years after thoracic irradiation. The authors considered presumptive the diagnosis and ascribed it to impaired lymphatic drainage; no histologic proof was presented.

However, we believe that a lung biopsy specimen demonstrating enlarged lymphatic vessels is a strong suggestion to this diagnosis and should be seen as diagnostic in a compatible clinical set.

REFERENCES

Metastatic Carcinoma of the Prostate Presenting as a Superior Vena Cava Syndrome* 

Carlos Montalbán M.D.; María A. Moreno, M.D.; José P. Molina, M.D.; Isabel Hernanz, M.D.; and Carmen Bellas, M.D.

A 75-year-old patient presented with a superior vena cava syndrome (SVSC) lasting 3 years. A prostatic carcinoma was found and a supraclavicular lymph node biopsy specimen disclosed metastasis of the prostatic carcinoma. Antiandrogen and luteinizing hormone-releasing hormone analogue therapy produced a marked improvement. Prostatic carcinoma, although a very rare cause, must be considered in the diagnosis of cases of SVSC with a protracted course, since it is a treatable disease. (Chest 1993; 104:1275-80)

LHRH = luteinizing hormone-releasing hormone; SVCS = superior vena cava syndrome

Carcinoma of the prostate is a very common neoplasm in men. On the other hand, superior vena cava syndrome (SVSC) is also common and in most cases due to lung neoplasms. We present the unusual case of a patient presenting with a SVCS due to metastatic carcinoma of the prostate.

CASE REPORT

A 75-year-old patient was referred with a history of dyspnea. He had a long history of chronic pulmonary airways disease and a prostatic adenoma had been diagnosed 7 years previously. Three years before, he had been studied in another hospital where supraclavicular lymphadenopathy and collateral venous circulation were found. Computed tomographic (CT) scan disclosed upper mediastinal lymph node enlargement and superior vena cava and left subclavian and jugular venous obstruction that was confirmed in a venogram. Superior vena cava syndrome was diagnosed, but lymph node biopsy specimen was not obtained and no further studies were performed. The patient came to the hospital due to increased dyspnea in the previous 3 months. Physical examination disclosed raised bilateral venous jugular pulse, facial swelling and cyanosis, venous engorgement of the head, neck, and upper chest, and very large supraclavicular masses, averaging in the largest diameter 7 cm on the left side and 4 cm on the right side (Fig 1). Nodular structures suggesting lymph nodes along with dilated varicose veins were felt within the masses. Thoracic auscultation disclosed tracheal stridor. No lymph node enlargement was found in other areas. The thyroid gland was not palpable and ophthalmologic examination, heart, and abdomen were normal. Digital rectal examination disclosed a nodular and firm prostatic gland. In the analytical examinations, results of blood and routine biochemical determinations were normal. Immunoglobulins and coagulation test were normal. Hypercoagulability could not be demonstrated.

Prostatic acid phosphatase level was 554 ng/ml and prostate-specific antigen was 4.154 ng/ml. Chest radiograph showed an enlarged upper mediastinum and a CT scan lymph node enlargement in the supraclavicular, infracavicular, upper mediastinal (Fig 2, top), obturator, pelvic, and retroperitoneal areas. The prostate gland was enlarged with heterogeneous density. Thyroid scan, serum thyroid...

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hormones, and thyroid-stimulating hormone levels were normal. Bone scan showed multiple areas of abnormal contrast uptake in the cervical and dorsal spine and left rib cage. Prostatic ultrasonography demonstrated a hypoechoic lesion within the gland and an aspiration needle cytologic study demonstrated prostatic adenocarcinoma. Supraclavicular lymph node biopsy specimen showed metastatic adenocarcinoma (Fig 3, top) with a high positivity for prostatic acid phosphatase and prostatic antigen stains (Fig 3, bottom). The patient was started on a regimen of oral anticoagulants and antiandrogen (Flutamide) and luteinizing hormone-releasing hormone (LHRH) analogue (Triptorelin) treatment. He improved progressively and four months after the supraclavicular lymph node enlargement had disappeared on the right side and was markedly reduced on the left, although dilated veins persisted. A CT scan showed a decrease in the size of the mediastinal nodes (Fig 2, bottom) and a venogram disclosed partial stenosis of the right subclavia vein and total occlusion of innominate, left subclavia and axillary veins with patent collateral circulation. Serum levels of prostatic acid phosphatase and prostatic-specific antigen lowered to 2.4 ng/ml and 238 ng/ml, respectively.

**DISCUSSION**

The diagnosis of a prostatic carcinoma in this patient with the findings of a hard prostate, elevated acid phosphatase and prostate-specific antigen serum levels, hypoechoic lesions on ultrasonography, and a positive prostatic cytologic study was straightforward. However, the finding of the huge and long-lasting supraclavicular lymphadenopathy with a protracted SVCS raised the question of another disease. Although benign causes of SVCS are possible, such as superior vena cava thrombosis (primary or associated with hematologic disease, Behçet's syndrome, and central intra-venous catheters or devices), syphilitic aortic aneurysm, thyroid goiter, or tuberculosis, fungous, or pyogenic mediastitis, they amount only to 15 to 20 percent of all cases. The majority of cases are due to neoplasms, lung carcinoma representing 70 percent of cases, lymphoma 6 percent, and other primary or metastatic disease such as breast carcinoma and testicular seminoma 9 percent. In this patient, the diagnosis of either a nonmalignant cause of the SVCS or a second slowly growing malignancy such as low-grade lymphoma, thyroid carcinoma, or salivary gland tumor was considered, but the biopsy specimen of the supraclavicular node demonstrated the prostatic origin of the lymph node.

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**Figure 1.** Supraclavicular lymph node enlargement and venous engorgement of upper chest and neck.

**Figure 2.** A (top): Computed tomographic (CT) scan at diagnosis showing enlargement of mediastinal lymph nodes. B (bottom): CT scan performed 4 months after starting treatment, showing a marked reduction of the mediastinal lymph nodes.

**Figure 3.** A (top): Supraclavicular lymph node biopsy specimen showing a metastatic adenocarcinoma (hematoxylin-eosin, ×20). B (bottom): In the same biopsy specimen, malignant glands contain prostatic-specific antigen (PO, ×20).
Detection of Intrapulmonary Shunts in Schistosomal Cor Pulmonale*

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Two patients with schistosomal cor pulmonale and central cyanosis were studied by contrast-enhanced echocardiography, using indocyanine green injection. Intrapulmonary shunts were detected by this method. To our knowledge, this is the first report that proves the presence of intrapulmonary shunts in schistosomal cor pulmonale detected by contrast-enhanced echocardiography.

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The picture of schistosomal cor pulmonale is a composite one. In 1957, Zaki1 described the various vascular shunts in this disease, namely, (a) bronchopulmonary, (b) pulmonary arteriovenous, (c) portopulmonary, and (d) intrasplenic. In 1964, Zaki et al2 reported this type of pulmonary artery-

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Figure 1. Patient A (top): Indocyanine green injected intravenously and appearing in the right atrium and right ventricle. B (bottom): Dye appeared in the left atrium and left ventricle after five cycles denoting presence of intrapulmonary shunts. Cycles are counted from video tape record.

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