is important to note that the metastatic lesions in our case were discovered only incidentally, the patient discovering them after undergoing a screening EBCT to assess his cardiac function. As EBCT is being used more often to evaluate cardiac function and disease, the conundrum of lung cancer screening is resurfacing.12
EBCT scans performed as screening procedures have the ability to provide high-resolution images and often present us with diagnostic dilemmas: to what extent is it cost-effective to pursue pulmonary irregularities incidentally discovered on cardiac screening? The findings of two previous Japanese nonrandomized studies13,14 on prevalence screening using low-dose CT with sputum sampling did not demonstrate clear decreases in mortality. A 1999 Mayo Clinic trial and the American College of Radiology Imaging Network have ongoing investigations, but no definitive evidence exists as to whether radiography for screening purposes has any impact on mortality.15 Our case highlights the efforts necessary to pursue the diagnosis entailed by the discoveries made available by these newer modalities; cost effectiveness remains to be elucidated.

REFERENCES
7 Vorzimer J, Perla D. An instance of adamantinoma of the jaw with metastases to the right lung. Am J Pathol 1932; 8:445–453

Venous Dilatation Seen on Routine Mammography*

A Clue to Superior Vena Cava Obstruction

Padmanabhan Krishnan, MD, FCCP; Lalith Uragoda, MD; Hemantha Rao, MD; and Santi R. Dhar, MD, FCCP

A diagnosis of superior vena cava obstruction (SVCO) generally is made on clinical grounds and can be confirmed by SVCO-specific diagnostic tests. When the obstruction is long-standing, clinical recognition may be compromised as venous drainage of the head, neck, chest, and upper extremity is diverted via collateral venous channels that bypass the obstructed superior vena cava. In such situations, only the visualization of this collateral flow will suggest the presence of SVCO. We describe a patient in whom the unanticipated diagnosis of SVCO was first suggested when routine mammography revealed grossly dilated superficial veins of both breasts, which were the result of collateral flow.

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Key words: mammogram; radionuclide venography; superior vena cava obstruction

Abbreviation: SVCO = superior vena cava obstruction

Clinical recognition of superior vena cava obstruction (SVCO) rests on the presence of a constellation of symptoms and signs that result from impaired venous drainage of the head, neck, chest, and upper extremities. SVCO-specific diagnostic tests such as radionuclide venography, contrast venography, and contrast-enhanced chest CT scan can be performed to confirm the diagnosis of SVCO. Obstruction of the superior vena cava results in collateral circulation diverted via the internal mammary, axysos, hemiazygos, lateral thoracic, thoracoepigastric, and vertebral veins.1 The improvement of venous drainage by collateral flow that bypasses the obstructed superior vena cava can allow SVCO to remain unrecognized. If these collateral venous channels are not visualized, SVCO is not suspected and SVCO-specific diagnostic tests are not performed.

This was the case in our patient in whom chronic SVCO

*From the Departments of Pulmonary Medicine (Drs. Krishnan, Uragoda, and Dhar) and Nuclear Medicine (Dr. Rao), Coney Island Hospital, Brooklyn, NY.
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Correspondence to: Padmanabhan Krishnan, MD, FCCP, Associate Director, Department of Pulmonary Medicine, Coney Island Hospital, 2601 Ocean Pkwy, Brooklyn, NY 11235
was not recognized until collateral flow was first visualized, in the form of grossly dilated superficial veins of the breasts, on routine mammography. A radionuclide superior venacavagram confirmed SVCO.

CASE REPORT

An 80-year-old woman was referred for the evaluation of possible SVCO after a routine mammogram revealed grossly dilated superficial veins of the breast (Fig 1). A radionuclide superior venacavagram (Fig 2) confirmed that the superior vena cava was obstructed. The superior vena cava was not visualized, and the collateral venous pathways were outlined clearly in the form of the internal thoracic vein on the left, the lateral thoracic vein on the right, and intercostal veins bilaterally.

A review of hospital records indicated that 8 years before, central venous catheter-induced superior vena cava thrombosis and obstruction were suspected during a prolonged and complicated postoperative course following resection of a rectal carcinoma. Since that time, the patient had noted facial and upper arm swelling, headaches in the supine position, and exertional dyspnea, all of which had decreased over the years.

An examination of the patient following the mammographic findings of venous dilatation revealed no facial or upper extremity swelling. The jugular veins were not distended and a few dilated veins were present over the upper anterior chest wall. The chest radiograph revealed no lung or mediastinal masses. The patient was thought to have chronic SVCO as a result of the thrombotic occlusion of the superior vena cava that had been induced by central venous catheter placement in the past.

DISCUSSION

Evidence of impaired venous drainage of the head, neck, chest, and upper extremities leads to the recognition of SVCO. Symptoms include dyspnea, headache, visual disturbances, epistaxis, hoarseness, dizziness, syncope, tongue swelling, and hemoptysis. Recumbency or bending over can exacerbate these symptoms. Physical signs include swelling of the face and upper extremities, jugular venous distension, facial plethora, chest or shoulder swelling, distended thoracic veins, proptosis, glossal or laryngeal edema, conjunctival suffusion, mental status change, impaired visual acuity, and Horner syndrome.2

Bronchogenic carcinoma remains the most common cause of SVCO. Benign causes include superior vena cava thrombosis and fibrosis following the insertion of central venous catheters, mediastinitis and fibrosis secondary to histoplasmosis and tuberculosis, and benign intrathoracic mass lesions.3

In acute SVCO, the presence of symptoms and signs of impaired venous drainage of the superior vena cava territory makes the diagnosis obvious. In patients with longstanding SVCOs, the development of collateral venous pathways can allow the SVCO to go unrecognized. These collateral pathways provide venous drainage to the head, neck, chest, and upper extremities, and they empty into the azygos vein and inferior vena cava. These pathways

Figure 1. A mammogram reveals the dilated superficial veins of the breast.

Figure 2. A radionuclide superior venacavagram reveals no visualization of the superior vena cava. Clearly visible is the collateral flow via the lateral thoracic vein (oriented vertically on the right, small arrow), the internal thoracic vein (oriented vertically on the left, large arrow), and the intercostal veins (oriented horizontally on both sides, arrowheads).
include the following: the internal thoracic-superior-inferior epigastric veins on the left; the lateral thoracic-superficial epigastric veins on the right; the anterior and posterior intercostal veins; the azygos-accessory hemiazygos-hemiazygous veins; the jugular veins; the vertebral plexus; and the small tributaries of the thoracoabdominal wall and the breast.\(^5\) On radionuclide venography, the lateral thoracic veins, the internal thoracic veins, the accessory hemiazygous veins, and the azygos veins can be seen running vertically (with the latter two veins being medially located), while the intercostal veins run horizontally.\(^4\) The superficial collateral flow via the small tributaries of the chest wall can be visualized on chest inspection and by contrast chest CT scan as dilated veins over the anterior chest wall. Dilated superficial veins of the breast that provide collateral flow have been visualized by mammography.\(^5\)

In this patient, we have described the presence of dilated superficial veins of the breast, which were first noted on routine mammography, served to alert the physician to a possible SVCO. As a result, a radionuclide superior venacavagram was performed, and the diagnosis of SVCO was established.

In patients with chronic SVCO, the development of collateral venous pathways that bypass the obstructed superior vena cava can make SVCO clinically occult. In such cases, only the visualization of these collateral pathways will suggest the unanticipated diagnosis of SVCO. Our experience is unique in that a routine mammogram served this purpose.

REFERENCES

Pamidronate Results in Symptom Control of Hypertrophic Pulmonary Osteoarthropathy in Cystic Fibrosis*

Luke A. Garske, MBBS; and Scott C. Bell, MD

Hypertrophic pulmonary osteoarthropathy (HPOA) may complicate the advanced lung disease that is associated with cystic fibrosis, resulting in severe joint pain and early-morning stiffness. Symptoms are usually controlled with the administration of nonsteroidal anti-inflammatory drugs, physiotherapy, and, on occasions, oral corticosteroids. This report describes a case of refractory HPOA with complete remission following the administration of IV pamidronate, which is a potent inhibitor of osteoclastic bone resorption. Symptom relief resulted for up to 3 months, but repeated courses of pamidronate have been required to maintain symptom control.

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Key words: biphosphonates; cystic fibrosis; hypertrophic pulmonary osteoarthropathy; pamidronate

Abbreviations: CF = cystic fibrosis; HPOA = hypertrophic pulmonary osteoarthropathy; NSAID = nonsteroidal anti-inflammatory drug

Hypertrophic pulmonary osteoarthropathy (HPOA) is a well-recognized complication of cystic fibrosis (CF) and occurs more frequently in patients with advanced lung disease.\(^1\) Joint pain, stiffness, mastalgia, and gynecomastia may complicate HPOA in patients with CF and are usually controlled by therapy with nonsteroidal anti-inflammatory drugs (NSAIDs), physical therapy, and occasionally therapy with systemic corticosteroids.

CASE REPORT

We report the case of a 27-year-old woman with CF who presented with severe diffuse bone pain and HPOA, which responded to therapy with IV pamidronate. The patient had moderate bronchiectasis (FEV\(_1\), 65% of predicted), and chronic \textit{Pseudomonas aeruginosa} infection. The treatment of pulmonary disease included physiotherapy, therapy with aerosolized bronchodilators, therapy with intermittent nebulized aminoglycosides, and two previous hospital admissions for therapy with IV antibiotics. The patient was well-nourished (body mass index, 24.6 kg/m\(^2\)), but a liver biopsy had confirmed the presence of biliary cirrhosis, which was complicated by portal hypertension (ie, splenomegaly, thrombocytopenia, and esophageal varices, which had been controlled with sclerotherapy).

In June 1999, the patient presented with an 8-week history of severe bilateral ankle and knee pain, and right elbow and right wrist pain. The pain was being poorly controlled with simple analgesia and was impairing the patient’s sleep at night. The pain was associated with severe early-morning stiffness and required leave from full-time employment as a clerical officer. A clinical examination revealed bony tenderness proximal to both ankles, which was associated with minimal ankle edema. There was no clinical evidence of synovitis or of an inflammatory arthritis. The findings of chest and abdominal examinations were unchanged from those of earlier examinations, and the results of spirometry testing were consistent with earlier values. Rheumatoid factor was negative, and antinuclear antibody level was weakly positive (nucleolar titre 160); the normal reference is <40 titre). How-

*From the Adult Cystic Fibrosis Unit and University of Queensland, The Prince Charles Hospital, Brisbane, Australia.

Received January 26, 2001; revision accepted October 2, 2001. Correspondence to: Scott C. Bell, MD, Department of Thoracic Medicine, University of Queensland, The Prince Charles Hospital, Rode Road, Chermside, Brisbane, QLD, Australia 4032; e-mail: scott_bell@health.qld.gov.au