TRACHEAL OBSTRUCTION SECONDARY TO EXTRAVASATION OF INTRAVENOUS FLUIDS FROM A CENTRAL CATHETER PORT

The use of central venous catheterization has gained widespread acceptance in the management of critically ill patients. Complications involving central line use are related to embolization and thrombosis [1], infection [2], vascular perforation and laceration [3], nerve injury [4], thoracic duct laceration [5], pneumothorax [6], and retained catheter fragments [7]. We report an unusual case of a patient who had acute respiratory decompensation secondary to tracheal compression as a result of the accumulation of extravasated intravenous fluids.

This patient is a 27-year-old woman with a history of insulin-dependent diabetes mellitus and migraine headaches who was admitted to University Hospital for further evaluation of a refractory migraine headache and mild diabetic ketoacidosis. The patient was managed with narcotic analgesics, intravenous fluids, and insulin with initial improvement in clinical status. Five days after admission, she developed a fever to 38.5°C with a new right-sided pulmonary infiltrate. Due to poor peripheral venous access, a triple-lumen central catheter was placed in the right internal jugular vein. The patient was managed with intravenous fluids and antibiotics. Two days after the placement of the central line, the patient complained of an "odd sensation in her throat" and intermittent dyspnea. Arterial blood gas analysis revealed no evidence of hypoxemia or carbon dioxide retention. The ear, nose, and throat service was consulted and performed laryngoscopy, which revealed prominent right arytenoid and aryepiglottic folds that were believed to be edematous. The following morning, the patient was transferred to the intensive care unit for management of worsening diabetic ketoacidosis. Antibiotics and fluids were infused through the distal and proximal ports of the central catheter. A chest radiograph revealed that the central line had migrated proximally since insertion (**Figure 1**). Although a blood return with aspiration was obtained through the distal port, there was no blood return from the proximal port. The fluids appeared to be infusing without difficulty and the catheter was not adjusted. Several hours after transfer to the intensive care unit, the patient experienced acute respiratory distress followed immediately by respiratory and cardiac arrest. It was noted at this time that the right side of the patient's neck appeared swollen. The patient was intubated with some difficulty and successfully resuscitated.

Subsequent computed tomography of the neck revealed a low-density fluid collection involving the right paratracheal area with marked left shift of the trachea with the endotracheal tube in place (**Figure 2**). The patient was taken to the operating room, where marked edema of the right side of the neck was noted without evidence of blood or pus collection. A tracheostomy was performed and a drain placed. Mechanical ventilation was discontinued 3 days later.

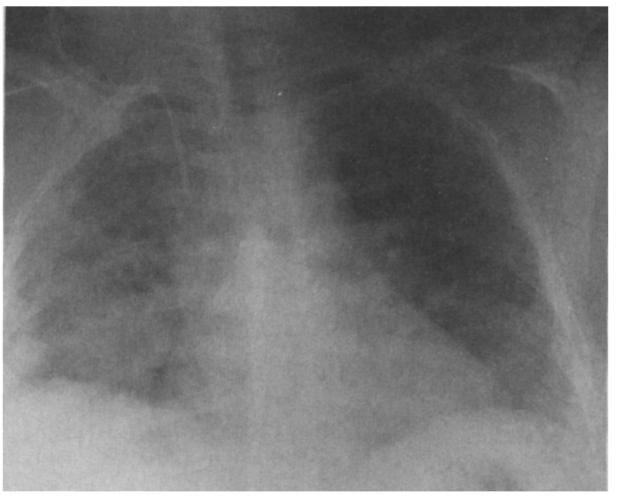

Figure 1. Chest radiograph showing position of right internal jugular catheter.

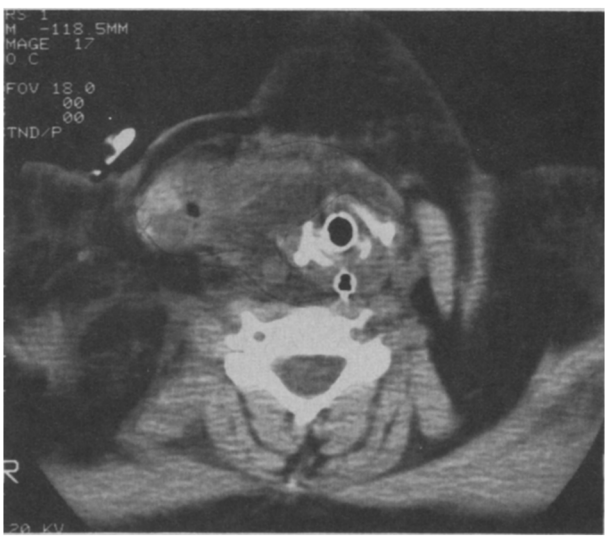

Figure 2. Computed axial tomographic scan of neck showing right paratracheal fluid accumulation with shift of trachea to the left.

We believe this patient had acute airway occlusion secondary to the extravasation of intravenous fluids from the proximal port of a right internal jugular central catheter. This port was likely located superior to the internal jugular lumen leading to extravascular fluid accumulation. We suspect the fluid perfused under low pressure through a fascial plane to form a paratracheal compressive mass. To

our knowledge, this complication of central venous catheterization has not been previously reported. This should reemphasize the importance of periodically assessing blood return prior to the infusion of fluids.

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DIGITAL VASCULITIS ASSOCIATED WITH INTERFERON THERAPY

Several drugs have been shown to cause necrotizing vasculitis [1]. When occlusion of the digital arteries of the hands occurs, the cause is usually small vessel disease rather than thromboembolic events [1,2]. We report a patient who developed digital vasculitis after treatment with interferons. We add this form of toxicity to those known to be caused by interferons and emphasize such toxicity may be seen in patients who are receiving long-term interferon therapy. This complication is considered clinically important because long-term interferon therapy is used in the treatment of chronic hepatitis B infection [3], inflammatory and viral dermatoses [4], rheumatoid arthritis [5], and with increasing frequency in hematologic malignancies such as chronic myelogenous leukemia, myeloma, and non-Hodgkin's lymphoma.

A 42-year-old man was diagnosed with chronic myelogenous leukemia in August 1988. He was a nonsmoker and denied any history of vascular or neurologic disorders. He participated in a Cancer and Leukemia Group B protocol using recombinant α-interferon (Hoffmann-La Roche) and γ-interferon (Genentech). The interferons were alternated weekly, and each was given at a dose of 5×10^6 IU/m^2/d by subcutaneous injection for 7 days.

Interferon-related toxicities were transient and mild and included arthralgia, fever, sweats, and nausea. Fifteen months after beginning interferon therapy, the patient noted paresthesias of the distal fingers bilaterally, more prominent on the right hand. Within 4 weeks, this progressed to numbness in all fingertips. He noted discoloration and diminished temperature of the right index finger that were not precipitated by temperature changes. The patient was taking no other medications. He denied fevers, arthralgias, myalgias, dysphagia, history of frostbite, toxin exposure, or trauma.

Physical examination revealed a cold blue, swollen right index finger with a small necrotic area at the distal tip. The remaining fingers were pale and cool. There was no sclerodactyly. Radial and ulnar pulses and Allen's test were normal bilaterally. There was diminished sensation to pinprick in all fingertips extending to the proximal interphalangeal (PIP) joint in the right index finger. The lower extremities were not involved. Results of the remainder of the physical examination were normal.

The chest radiograph, electrocardiogram, and echocardiogram were normal. A complete blood count showed a leukocyte count of 2.7×10^9/L, hemoglobin level of 11.5 g/dL, and a platelet count of 221×10^9/L. Serum chemistries, prothrombin time, activated partial thromboplastin time, thrombin time, fibrinogen, protein C, antithrombin III, protein S, and complement levels were all normal. The sedimentation rate was 26 mm/h. Rheumatoid factor, antinuclear antibody, VDRL, antibody to the two interferons, and hepatitis B serologies were all undetectable. Results of indirect and direct Coombs' tests and blood cultures were negative.

Digital plethysmography showed an absence of waveforms in the right index finger. Arteriography revealed a normal aortic arch and great vessels. Digital subtraction angiography (DSA) of the hands showed that each finger in both hands had at least one digital artery completely occluded (**Figure 1**), and the vascular profile suggested endothelial proliferation. Both digital arteries in the right index finger were occluded. Warfarin therapy was initiated, and the interferons were continued. After 4 weeks the symptoms were unchanged, and a new necrotic lesion developed on the distal aspect of the left second toe. The warfarin and both interferons were discontinued. After 2 weeks, the right index finger remained cool, assumed normal color, and demonstrated healing. The other fingers were warm. The paresthesias, numbness, and sensory loss had resolved. Plethysmography showed partial return of arterial waveforms in the right index finger.

The DSA in our patient showed evidence of a beaded pattern in the digital arteries, consistent with vasculitis [6]. The improvement in his hand