Acute Pulmonary Edema Caused by Venous Air Embolism After Removal of a Subclavian Catheter*

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A 17-year-old boy, who had received chemotherapy for a relapse of acute myelogenous leukemia, manifested a brief loss of consciousness and acute pulmonary edema immediately after the removal of a triple-lumen subclavian catheter. This complication was attributed to a venous air embolism.

Venous air embolism, a well-recognized risk of certain medical procedures, may on rare occasions be complicated by pulmonary edema. The reported cases occurred after air insufflation into a catheter inserted in a forearm vein and after neurosurgical operations. A young patient developed acute pulmonary edema likely related to a venous air embolism after the removal of a subclavian catheter. All clinical symptoms disappeared in two hours without specific treatment.

Case Report

Acute myelogenous leukemia was diagnosed in this 17-year-old boy in June 1985. Remission was induced with chemotherapy from June to September 1985. In May 1986, the patient was again hospitalized for chemotherapy after a relapse was diagnosed. The treatment induced severe agranulocytosis for four weeks. A right subclavian vein triple-lumen catheter (external diameter, 2.3 mm) was used for infusing cytotoxic drugs. On June 28, a chest roentgenogram was normal (Fig 1).

On June 29, the catheter was removed while the patient was in a half-sitting position. Immediately afterward, he complained of retrosternal oppression accompanied by dyspnea, and then lost consciousness for a few seconds. The systolic blood pressure was 80 mm Hg, the pulse was regular at 100 beats per minute, the respiratory rate was 40 per minute. Five minutes later, cardiopulmonary...
negative pressure of 5 cm H2O is applied. This can already represent a lethal volume.

Acute pulmonary edema is a rare complication after venous air embolism. Measurements of protein concentration in edema fluid and in plasma showed that this edema is due to hyperpermeability. In a sheep model, large numbers of polymorphonuclear leukocytes agglomerate around the air bubbles in the pulmonary capillaries. The pulmonary edema is less important after depletion of circulating leukocytes or addition of the enzyme superoxide dismutase, which inactivates the toxic superoxide anion released by activated leukocytes.

These results support the hypothesis that the microvascular injury that results in increased permeability of lung capillaries during venous air embolism is related to leukocyte production and release of toxic oxygen metabolites. The activation of the coagulation cascade does not seem to be important for the evolution of this acute pulmonary edema.

In our case, the pulmonary edema had rapidly resolved, and the patient was again well two hours after removal of the catheter. In other cases, the pulmonary edema was severe enough to require mechanical ventilation, and one case of adult respiratory distress syndrome was described. These observations show, once again, the importance of correct handling of intrathoracic catheters, particularly if the patient is not in the horizontal position.

REFERENCES
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