PULMONARY EMBOLISM AND HEMOLYTIC ANEMIA IN A PATIENT WITH COLD AGGLUTININ DISEASE

AKIRA MURAYAMA, M.D., TAKAO AYABE, M.D., KEICHI TOYAMA, M.D. AND YOSHIRO NAKAMURA, M.D.

We report a case of multiple pulmonary embolism with hemolytic anemia. The patient was a 48-year old woman who suffered from cold agglutinin disease with evidence of a recent mycoplasma pneumonia infection. Multiple pulmonary embolism was diagnosed on the basis of her symptoms, serial blood gas studies and $^{99}$Tc-MAA perfusion lung scan. A recent mycoplasma infection was evidenced by both a 1:64 rise in mycoplasma complement fixation titers and a 1:2048 rise in cold agglutinin titers. The pulmonary embolism was thought to be due to massive intravascular hemagglutination taking place in the presence of cold agglutinin, and the hemolytic anemia with an elevation of IgM immunoglobulin was thought to be autoimmune in nature.

MYCOPLASMA pneumonia infection is known to occur with various clinical manifestations besides primary respiratory tract infection. We report a case of cold agglutinin disease secondary to mycoplasma infection complicated with multiple pulmonary embolism and severe autoimmune hemolytic anemia.

CASE REPORT

A 48-year old Japanese woman was admitted to our hospital because of severe dyspnea. She developed cold-like symptoms with dry cough and fever 2 weeks prior to admission. A week later she began to notice increasing dyspnea and became orthopneic. There was no history of cardiac disease, thrombophlebitis or a recent

Key Words:
Pulmonary embolism
Cold agglutinin disease
Mycoplasma pneumonia infection

(Received October 1, 1980; accepted June 24, 1981)
Department of Internal Medicine, School of Medicine, Keio University, Tokyo, Japan
Address for correspondence: Akira Murayama, M.D., Cardiopulmonary Division, Department of Internal Medicine, School of Medicine, Keio University, 35 Shinanomachi, Shinjuku-ku, Tokyo 160, Japan

pelvic operation. On physical examination, she was found to be a moderately obese woman who was pale and orthopneic. She had a regular pulse rate of 108/min. Her blood pressure was 120/90 mmHg and respiration rate 32/min. Her neck veins were flat at 30°. Moist rales were heard at both lung bases. The apical impulse of the heart was not felt. The heart sounds were normal, and neither murmurs nor gallop sounds were audible. Hepatomegaly, thrombophlebitis and pretibial edema were not noted. The urinalysis was normal. The complete blood count revealed microcytic hypochromic anemia with a hemoglobin of 9.2 g/dl and a reticulocyte count of 5%. The white cell count was 20,200/mm³ and the platelet count 950,000/mm³. The chest roentgenogram showed an elevation of the left diaphragma and no infiltrate in the lung fields (Fig. 1). The electrocardiogram showed ST segment deviation suggestive of acute coronary insufficiency (Fig. 2). The arterial blood gas analysis revealed a PaO₂ of 50 mmHg, a PaCO₂ of 28 mmHg and a pH of 7.57. The lung scan using ⁹⁹mTc-MAA showed bilateral multiple perfusion defects (Fig. 3). The anticoagulant therapy was instituted with intravenous heparin and followed by oral warfarin. The hemoglobin was 6.5 g/dl on the 4th hospital day. On the 5th hospital day, there was a rise in her mycoplasma complement fixation titer to 1:64 and in her cold agglutinin titer to 1:2048. The IgM erythrocyte antibody was detected in her serum. Oral administration of prednisolone was started. She responded well to the therapy, and her anemia and dyspnea improved. She was discharged on the 35th hospital day. Her cold agglutinin and mycoplasma complement fixation titers subsequently returned to their normal ranges (Fig. 4).

DISCUSSION

Mycoplasma pneumonia infection is known to be associated with complications of the central nervous system or the skin.¹ ² Besides these complications seen during acute phases of mycoplasma pneumonia infection, the postinfectious cold agglutinin disease sometimes occurs giving rise to autoimmune hemolytic anemia and/or intravascular hemagglutination.³ ⁵ However, the manifestations of intravascular agglutination in the peripheral circulation have been largely described. Although pulmonary embolism in sickle cell anemia is thought to occur because of sickling

Fig. 2. Electrocardiogram on admission.

Fig. 3. ⁹⁹mTc-MAA perfusion lung scan on admission. Upper left: anterior view. Upper right: right lateral view. Bottom left: left lateral view.

Fig.4. The clinical course of the illness. Heavy arrows indicate the time when the blood samples were drawn. Mycoplasma CF titer = mycoplasma complement fixation titer, BT = body temperature, Hb = hemoglobin.

of erythrocytes in the pulmonary vasculature, there has been, to our knowledge, no case report regarding massive pulmonary embolism caused by cold agglutinin disease following mycoplasma pneumonia infection. Therefore, the present case is unique in that multiple pulmonary embolism took place during the course of cold agglutinin disease. And its mechanism was thought to be massive intravascular agglutination in the pulmonary vasculature. The presence of cold agglutinin in the patient’s serum with severe autoimmune hemolysis lends support to this consideration. Moreover, our patient did not have ordinary causes of pulmonary embolism such as deep vein thrombophlebitis, valvular heart disease or sickle cell anemia. It is also of interest that there were no peripheral signs of intravascular agglutination.

Thus our experience suggests pulmonary embolism due to intravascular agglutination should be always taken into consideration in a patient with mycoplasma pneumonia who is suffering from cardiorespiratory distress.

REFERENCES

2. HORSTMANN D, TATLOCK H: Cold agglutinins: A diagnostic aid in certain types of primary atypical pneumonia. JAMA 122: 369, 1943