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Meningococcal Meningitis and Meningococcemia Associated with Pulmonary Infiltrates and Hemoptysis*

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A case of meningococcemia, meningitis and a pulmonary infiltrate with hemoptysis has been presented. The significance of the infiltrate has been discussed and a brief review of the literature on meningococcal pneumonia has been included. A brief discussion of the therapy of meningococcal pneumonia based on pathophysiologic evidence is included.

Pulmonary complications of meningococcal pneumonia are rarely discussed and seldom present significant clinical problems. Culture proved primary meningococcal pneumonia is uncommon and in several reports has not been associated with classic meningitis. Hemoptysis has not been reported in the literature in association with meningococcal pneumonia. The mechanisms responsible for a possibly bleeding diathesis in meningococcemia are not fully under-

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A recent case of meningococcemia, meningococcal meningitis and a pulmonary infiltrate with hemoptysis will be reviewed and a brief discussion of meningococcemia and some of its complications and therapy will be undertaken.

CASE REPORT

The patient was a 20-year-old white military recruit in the early weeks of basic training who had been confined at another military hospital for five days with a severe upper respiratory tract infection characterized by fever, cough and sore throat. Symptomatic treatment and possible antibiotics were rendered and he was discharged asymptomatic five days before his admission here.

Eighteen hours prior to admission, he developed general malaise and headache which was followed shortly thereafter by dizziness, pain in both legs and a temperature of 100° F (37.8° C). He was examined by a physician who noted no nuchal rigidity or rash. A course of tetracycline was started. Ten hours prior to admission, he became ataxic and vomited several times. His speech became confused and petechial rash ware noted by a parent shortly before admission. There was a questionable history of penicillin allergy offered by the parent.

Physical examination at the time of admission revealed a normally developed, well nourished, disoriented white man who appeared acutely ill and who had no palpable radial pulse and no recordable blood pressure. Temperature was 97° F (36.1° C), respiration 24 per minute and apical cardiac rate 130 per minute and regular. The pupils were equal and reacted to light. Disc margins were blurred bilaterally; tympanic membranes were markedly hyperemic. The mouth and pharynx showed dry mucous membranes. There was moderate nuchal rigidity. The chest was clear and the heart was of normal size and no abnormal sounds were heard. The abdomen showed diffuse mild tenderness to direct palpation, but no rebound was elicited. Bowel sounds were normal; genitalia and rectum were normal. The skin was pale, cool and diaphoretic, and there was a generalized petechial eruption. There was no significant adenopathy.

Neurologic examination revealed depressed but equal deep tendon reflexes; there was no pathologic reflex. Kernig's and Brudzinski's signs were positive.

Lumbar puncture upon admission revealed milky fluid containing 15,200 WBC, 63 per cent polymorphonuclear leukocytes and 37 per cent lymphocytes. The glucose was 12 mg/100 ml and the protein was 156 mg/100 ml. Gram stain of the cerebrospinal fluid (CSF) showed several white blood cells, many containing organisms morphologically resembling Neisseria meningitidis. Cultures of the CSF, blood and petechiae were subsequently reported positive for N meningitidis, group B, resistant to 1.0 mg/100 ml sulfadiazine but sensitive to 2.0 mg/100 ml. Hematocrit was 40; WBC count 41,400 with 47 neutrophils, 2 metamyelocytes, 29 bands, and 22 lymphocytes. Blood urea nitrogen (BUN) 48, sodium (Na) 137 mEq/liter, potassium (K) 4.7 mEq/liter, chloride (Cl) 99 mEq/liter, and carbon dioxide (CO₂) 27 vol. per cent. Electrocardiogram showed a sinus tachycardia of about 130 per minute but was otherwise normal.

Original therapy consisted of erythromycin 1 gm, sulfisoxazole (Gantrisin) 3 gm, hydrocortisone sodium succinate (Solu-cortef) 500 mg which was administered intravenously in the admission room. Intravenous therapy with erythromycin 4 gm, chloramphenicol (Chloromycetin) 2 gm, sulfisoxazole 8 gm, hydrocortisone 800 mg daily was instituted.
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upon arrival on the ward. Metaraminol (Aramine) was originally used as required to hold the blood pressure at 90 to 100 systolic. Six hours after admission, vasopressor treatment was changed to levarterenol (Levophed), which was required for the next 30 hours. The patient was semicomatose during this period of time.

On the second day, the petechial rash had become more confluent; subconjunctival hemorrhages were noted and moist rales were present throughout the right lower lung field anteriorly, laterally and posteriorly. Portable chest x-ray examination at this time demonstrated an infiltrative density in the region of the right middle lobe with smaller areas of infiltration demonstrated throughout both the left and right lung. There was also evidence of pleural fluid (Fig 1). We feel the x-ray film revealed right middle lobe pneumonitis with consolidation associated with diffuse bronchopneumonic changes in both lungs.

The patient exhibited no clinical evidence of congestive heart failure at this time. No ventricular diastolic gallop, hepatomegaly, peripheral edema or elevation of the jugular venous pressure was present. Sputum production was scanty, mucopurulent, and was streaked with bright red blood. On at least two occasions, the patient expectorated 15 to 20 ml of bright red blood. No definite source of bleeding was noted in the nasopharynx. Culture disclosed Micrococcus which was otherwise not classifiable. The patient had been on antibiotics for at least 24 hours at the time the sputum culture was obtained.

Bleeding, clotting, and prothrombin times, fibrinogen level, thromboplastin regeneration, activated partial thromboplastin time and platelet count were all subsequently shown to be within normal limits. Hematocrit fell from 40 to 32 in 48 hours.

Stool guiacs were positive and microscopic hematuria was present, but this was of questionable significance because a Foley catheter was in place and high doses of sulfonamide were being used. Forty-eight hours after admission when the patient was alert enough to be further questioned and any penicillin allergy was denied, penicillin, 40 million units daily was added. Antibiotic therapy was continued for seven days.

The patient was fully conscious and alert by the fifth hospital day. Expectorants were added and corticosteroids were reduced stepwise over the next two weeks. Serial chest x-ray films showed gradual clearing of the right lung infiltrative process until a stable x-ray picture was obtained by the 11th hospital day. Hemosiderin-laden macrophages were recovered from the sputum on the sixth hospital day and sputum stains for iron were positive for four consecutive weeks. Lung scan with 131I macroaggregates during the second week of hospitalization was normal.

Evaluation after the acute process had subsided revealed normal adrenal cortical function as measured by serial urinary 17 OH and 17 ketosteroid determinations of 6.0 mg, 7.7 mg/24 hours and 14.9 mg, 16.9 mg, 10.3 mg/24 hours respectively. Audiogram demonstrated minimal high tone loss in the left ear. A partial left median nerve palsy secondary to a flexion contracture at the left elbow was the only neurologic residual. This was gradually improving through the use of physical therapy. Bone marrow aspiration performed during the third week of hospitalization because of normochromic normocytic anemia, hematocrit 34 per cent, revealed normoblastic hyperplasia consistent with transient marrow depression secondary to drug therapy. The normoblastic activity was suggestive of recovery, and within an additional three weeks, the hematocrit had returned to the mid-40's.

FIGURE 1. Chest roentgenogram on second day of admission.

DISCUSSION

The exact etiology of the right middle lobe infiltrate is not known, but probably represents either an area of intrapulmonary hemorrhage or an area of primary meningococcal pneumonitis. The failure to demonstrate the Meningococcus in the sputum may well be due to the fact that the patient had been on antibiotic therapy for 24 hours prior to producing the specimen.

Ferguson and Chapman,3 in an autopsy series of 16 patients with fulminating meningococcal infection, noted the marked vascular damage resulting in thrombosis, hemorrhage or both in many organ systems. The lungs showed varying degrees of congestion and edema. Actual hemorrhage into interstitial tissue and alveoli varied from slight to marked in all cases. The authors make no mention of sputum cultures in this series.

Meningococcal pneumonia rarely has been described in literature. The vast majority of cases were reported as sequelae of the severe influenza epidemic of the World War I period and shortly thereafter. Jacobitz4 reported 12 cases of meningococcal pneumonia in one company of soldiers in 1919. Fletcher5 noted that Meningococcus was the predominant organism in the sputum of 11 of 36 patients who died with bronchopneumonia in an epidemic aboard a troop ship. Haemophilus influenzae was also present in the sputum of these 11 patients. Nineteen cases with positive lung cultures

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were reported by Kinnicutt and Binger; an additional five cases by Meader et al and 23 cases by Holm and Davidson were reported in 1919. Two cases were noted by Brick in 1948 and single case reports were made by Roberge in 1945 and Meltzer in 1957.

Autopsy studies reveal the pneumonia is usually of a lobular form, rarely of a lobar type and seldom involves the pleura. Patches of consolidated areas of a peculiar pale violet color have been noted. Since it would appear that the Meningococcus is no less prevalent today, it may be that the organism is overlooked in cultures other than CSF and blood.

The treatment of meningococcal disease has recently been investigated by Levin and Painter. They have pointed out that considerable physiologic data would support the thesis that the hypotension attendant to meningococcemia is due primarily to a reduced cardiac output rather than to a peripheral vascular collapse. They have attempted to combat this through the use of digitalis and a supplemental isoproterenol drip in patients not responding well to digitalis alone. It is their opinion that adrenal cortical steroids are of limited value in fulminating meningococcemia. Intravenous penicillin alone was used in their series.

REFERENCES

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