Pneumonitis Associated with Reiter's Disease

Report of a Case

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INTRODUCTION

REITER IN 1916 REPORTED A CASE OF arthritis, conjunctivitis, and urethritis in an army officer having diarrhea and spirochaetemia. Since that time, many reviews and case reports of the disease bearing his name have appeared in the literature noting involvement of various organ systems including the eye, skin, respiratory tract, locomotor system, lymphatics, gastrointestinal tract, heart, and central nervous system. The following case is reported representing what appears to be pneumonitis as a manifestation of Reiter's disease.

CASE REPORT

E. E., a 29-year-old white printer, was admitted to Philadelphia General Hospital on October 19, 1960 with the following history: on October 6, 1960, he noted a white urethral discharge. Three days later, he visited his family doctor who prescribed penicillin tablets for him and for his wife. No laboratory examination of the discharge was performed. The discharge continued and on October 11 he experienced sharp bilateral inguinal pain radiating to the lumbar areas. The following day his physician added chloramphenicol orally to the regimen. He remained on both of these medications until admission to the hospital without notable change in the discharge. On October 16, there developed numbness and pain of the right elbow and injection of the left bulbar conjunctiva. Three days later, on October 19, he again developed discomfort in the inguinal areas and in the low back. This was associated with right scapular aching, decreased ability in moving his legs, and excessive perspiration. He became anxious at this time, and subsequently noted weakness of his voice, trismus, numbness of the face, dysphagia, sore throat, weakness of the arms, and further weakness of his legs. No pruritus or rash had been noted. There were no known allergies and no previous history of arthritis or conjunctivitis. Nine years before he had been treated with penicillin on two separate occasions while in the Armed Forces, once for pneumonia and once for gonorrhea. There had been no recurrence of urethral discharge since that time until the present illness. He was married two weeks prior to the onset of his symptoms.

Physical examination revealed a pale, anxious, man with blood pressure 138/85, pulse 80/min., respirations 30/min., and temperature 100° F. orally. The bulbar conjunctivae were injected bilaterally; more on the left. The fundi were clear. The oropharynx was erythematous, and small injected, hemorrhagic ulcerations were present on the soft palate. The gums contained no abnormal pigmentation. Nuchal rigidity was absent. Skin and nails were devoid of rash, excoriation or thickening. Soft, small, movable nontender lymph nodes were noted bilaterally in the posterior cervical, axillary, and superficial inguinal areas. The heart, lungs, and abdomen revealed no abnormality. The musculoskeletal examination revealed tenderness of the masseter muscles and the area over the spinous process of T11. There was painful limitation of motion, synovial thickening and increased warmth of the right elbow. There was no other joint involved. On genital examination, a white urethral discharge was present. The prostate was normal to palpation. Neurologic findings were within normal limits.

Clinical Course: He was put at bed rest. The initial complete laboratory studies were within normal limits except for a white blood count of 12,240 per cmm. with 8 band forms; urine +1 protein, trace of sugar, innumerable white blood cells and 10 to 12 red blood cells; and fasting blood sugar of 117 mg. per cent. The uric acid was 6.6 mg. per cent. Urethral smear was negative for Neisseria gonorrhoeae and the culture grew Staphylococcus albus, coagulase negative. The following day, the right knee was noted to be warm, painful, and swollen. The aspirate was cloudy yellow and contained 16,000 white blood cells per c.c. with 87 per cent neutrophiles and protein of 4.5 gm. per cent. No organisms was seen on smear of this fluid and the culture was sterile. Two blood cultures were sterile. Pharyngeal culture produced Diplococcus pneumoniae.

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and a Neisseria species. Latex agglutination for rheumatoid arthritis, febrile agglutinins, C-reactive protein, sheep-cell heterophile agglutination, and the urinary Watson-Schwarz test were within normal limits. His oral temperature ranged from 100 to 101°F. Aspirin, 600 mg. every four hours, and tetracycline, 250 mg. every six hours, were administered orally.

The next day, October 21, a supine chest x-ray film was taken and originally reported as "several areas of linear infiltration in the right upper lung field," but later review suggested the possibility that these were artifacts. Thoracic and lumbar spine x-ray films were negative. The following day, he developed dry cough, although physical examination of the chest revealed no abnormality. Tetracycline was increased to 500 mg. every six hours. At this time, the previously noted conjunctivitis had diminished greatly although he continued to be extremely weak and listless. Two days later, the seventh hospital day, his cough became productive of grossly bloody sputum and dullness and decreased breath sounds were present at the right base. His temperature showed low-grade elevation as before. Subjectively, he felt stronger, the only complaint being stiffness of the right knee. The following day, October 25, he experienced severe pleuritic chest pain in the right midaxillary line over the seventh rib. Rales appeared in addition to the previous findings at the right base. Supine chest x-ray film taken on this date revealed a minimal pleuroneumonic process with pleuritis at the right base (Fig. 1). Two sputum cultures were taken several hours apart, one growing Streptococcus pyogenes and the other Streptococcus viridans, Pneumococcus species, Neisseria species, and Monilia species. The next day his ankle became tender, warm, and swollen. The right knee was again greatly enlarged and was aspirated with laboratory results similar to those previously reported, being negative for tubercle bacilli on smear and negative for Neisseria on culture. Sputum smears were negative for acid-fast bacilli at this time although intermediate strength PPD skin test was positive. On October 28, aqueous penicillin 600,000 units every six hours intramuscularly was added to his regimen, as an abortive form of gonococcal arthritis could not be ruled out despite the negative cultures. By this time, his cough had diminished though his temperature was unchanged.

Five days later, on November 2, left elbow pain and right bulbar conjunctivitis developed. A culture from the right eye revealed Staphylococcus albus, coagulase negative. Posteroanterior and lateral chest x-ray films taken on November 2 showed definite increase in the pleuroneumonic process with infiltration of the mesial and posterior segments of the right lower lobe (Fig. 2). At this time, there was no cough or rales, although the breath sounds continued to be depressed at the right base. One week later, on November 9, the left elbow and right ankle had returned to normal and the conjunctivae were clear. The right knee continued to be slightly
swollen and had limitation of motion. The chest remained unchanged on physical examination. His temperature still showed a low grade elevation. All medications were discontinued on this, his 22nd hospital day.

Four days later, fusiform swelling of the proximal interphalangeal joint of the fourth finger of the right hand developed. His sedimentation rate was 26 mm./hr. On November 14, an x-ray film of the chest was within normal limits and x-ray films of the right knee showed soft tissue swelling. Thickening of the skin of the soles of the feet was noted and was consistent with early keratoderma blenorrhagicum. He was discharged on November 17, after 29 days of hospitalization, to be followed as an outpatient.

He was seen two weeks later at which time he had painful swelling of the right knee and ankle. Both joints were aspirated and injected with hydrocortisone. His sedimentation rate was 42 mm./hr. He stated aspirin had helped the pain and that he had been having night sweats. Two months later, he was asymptomatic and had returned to work. Chest x-ray film at this time was negative. Nine months after discharge, he continued to be asymptomatic.

**DISCUSSION**

Active pulmonary infiltration in the absence of another apparent etiology has been described previously in reports of Reiter's disease. 14,15 "Dry pleurisy" has been noted in up to 10 to 22 per cent of cases. 2,3,16,17 However, in other series, no mention of pleurisy has been made. 4,6,18,19 Three of 23 cases of Reiter's disease reported by Hall and Finegold 19 were noted to have cough. One of them had pleuritic chest pain and two had audible moist rales. Only one of the three had pulmonary lesions demonstrable by x-ray. This consisted of a transient peribronchial infiltrate in the right lower lobe consistent with bronchopneumonia. Lafon et al. 11 in 1955, reported a case of Reiter's disease having x-ray evidence of multiple bilateral pulmonary infiltrates which cleared spontaneously in ten days. There was no clinical finding on examination of the chest.

Pulmonary manifestations have been noted as a part of the disease entity in patients with systemic lupus erythematosus, scleroderma, and rheumatoid arthritis. Thus, it should not prove surprising to find pulmonary lesions in patients with Reiter's disease, long considered by some a variant of rheumatoid arthritis. An interesting relationship also has been suggested linking Reiter's disease with Stevens-Johnson syndrome. 9 Roentgen-ray findings similar to those seen in primary atypical pneumonia, ranging from minimal involvement to nearly complete consolidation, have been reported in 30 to 83 per cent of cases of Stevens-Johnson syndrome. 11 Stanyon and Warner 5 have suggested that the pulmonary findings are due to mucosal changes in the tracheobronchial tree. Although no gross ulceration was seen in the tracheobronchial mucosa in their cases, microscopic inflammatory reaction was seen in the bronchi and pleura. Bronchial ulcerations have been reported elsewhere in Stevens-Johnson's syndrome. 5 In the case now being reported, as in many cases of Reiter's disease and Stevens-Johnson syndrome, there were lesions of the oral mucosa. The hemorrhagic appearance of the palatine lesions in this patient as well as the subsequent effortless hemoptysis suggest the possibility that lesions similar to those seen on the oral mucosa were present in the tracheobronchial tree.

It would be of interest in the future to note the frequency of pulmonary manifestations in patients with Reiter's disease demonstrating oral mucosal lesions.

Although it cannot be stated categorically that the pulmonary lesion noted in this case had a direct relationship to the Reiter's disease, the course of events seems to make this a reasonable assumption. The patient, a previously healthy young man, developed transient lung findings coincidental with the onset of Reiter's disease. These changes occurred while he was on a broad-spectrum antibiotic and the pulmonary manifestations roughly paralleled the general course of his disease. There was no predominant or consistent organism isolated on sputum cultures to support a bacterial etiology for the pneumonitis. Viral etiology is possible, but associated hemoptysis is uncommon. 8 The presence of similar cases in the literature supports the conclusion that a primary
relationship existed in this patient between the pneumonitis and the remainder of his clinical picture.

REFERENCES


CARDIOVASCULAR MANIFESTATIONS OF DERMATOMYOSIS AND POLYMYOSITIS

Cardiovascular manifestations of dermatomyositis and polymyositis are considered rare; more often there are only histologic modifications of the heart muscle in the absence of any clinical manifestation apart from tachycardia. Two cases are reported: a polymyositis with attacks of angina pectoris and high blood pressure ending in an irreducible angina; a dermatomyositis with attacks of angina pectoris leading to a fatal myocardial infarct. In both cases, there existed evidence of arteriosclerosis associated with collagenosis, but in one of these patients, myocardial lesions related to polymyositis were also noted. The interaction of these two diseases renders the course of coronary disease more rapid and severe.


PAROXYSMAL VENTRICULAR TACHYCARDIA

Three cases of paroxysmal ventricular tachycardia in the absence of demonstrable heart disease are presented. Diagnosis of this condition becomes certain only after its demonstration by the electrocardiogram. The identification of P waves during the paroxysm, at a slower rate than the ventricular complexes; the presence of a paroxysm of abnormal ventricular complexes occurring during auricular fibrillation; the onset of tachycardia with an abnormal ventricular complex or a close resemblance, in the same lead, of isolated ectopic QRS complexes to the complexes of the tachycardia are the established criteria for diagnosis. The esophageal lead may be of great help.

Therapy is directed toward removal of precipitating factors and use of procaine amide or quinidine to suppress the ectopic factors. Established bouts require vigorous attention, i.e., parenterally administered procaine amide or quinidine.