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A CASE OF SEVERE ALLERGIC PULMONARY REACTION TO GOLD

BY

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This report describes a case of severe pulmonary reaction due to gold therapy of rheumatoid arthritis. The severity of the reaction almost caused the death of the patient. ACTH brought dramatic and lasting relief. The unusual type of the reaction and the importance of recognizing similar cases seem to justify this communication.

The case. S. K., a 61 year old woman. She had been essentially healthy until her 57th year. Then she began to suffer from a moderately severe rheumatoid arthritis particularly affecting the knees. She was treated conservatively at home but when after four years a contracture of the left knee developed she was admitted to a rheumatoid arthritis hospital.

Here she was given a course of intramuscular gold injections. Having received 0.30 gms of one preparation her eosinophil count rose to 10 per cent. Treatment was then switched over to a different gold preparation of which she received a total amount of 2.6 gms. The course was given during two months. Her rheumatoid symptoms improved. In order to prevent allergic complications she was given antihistamines. Possibly because of that she complained a little of

dryness of the mouth. Her eosinophils under the later period of treatment did not exceed 8 per cent. She was discharged home on October 8, 1952.

The day after her return home she became acutely ill with chills, temperature of 40° C. and severe prostration. She went to bed but having had a temperature for two more days she summoned a physician who gave her penicillin. She now received penicillin by injection for five days but without any improvement. Instead she developed dyspnoea and a dry non-productive cough. She was then admitted to an infectious disease hospital.

On arrival there she had a temperature between 38 and 39° C. Her white count varied between 7-11000 per cu.mm. with a normal differential. The eosinophils did not exceed 4 per cent. A chest x-ray showed bilaterally blotchy densities which were more marked towards the lung bases. Treatment with penicillin, streptomycin and terramycin in adequate dosage did not improve her condition and she grew steadily worse. The temperature climbed higher, her dyspnoea increased and fine pulmonary râles were heard over both lung fields. An electrocardiogram showed "signs of myocardial damage".

With a history of three weeks undiagnosed febrile illness after her dismissal from the rheumatoid arthritis hospital she was transferred from the infectious disease hospital to the medical department. She appeared to be severely ill with a stertorous type of dyspnoea and a respiration rate of 36 per minute. Her face had an ashen pallor and her lips were cyanotic. A new chest x-ray showed increasing pulmonary infiltrations now occupying most of both lungs. No asthmatic wheezing sounds were heard. There was slight edema of the lower extremities.

The laboratory data showed a normal hemoglobin and red cell count and a white count between 8-9000 per cu.mm. The total eosinophil count amounted to 1200 per cu.mm. The sedimentation rate was 93 mm. an hour. Urine contained a small amount albumen. Blood culture and cold agglutinin test

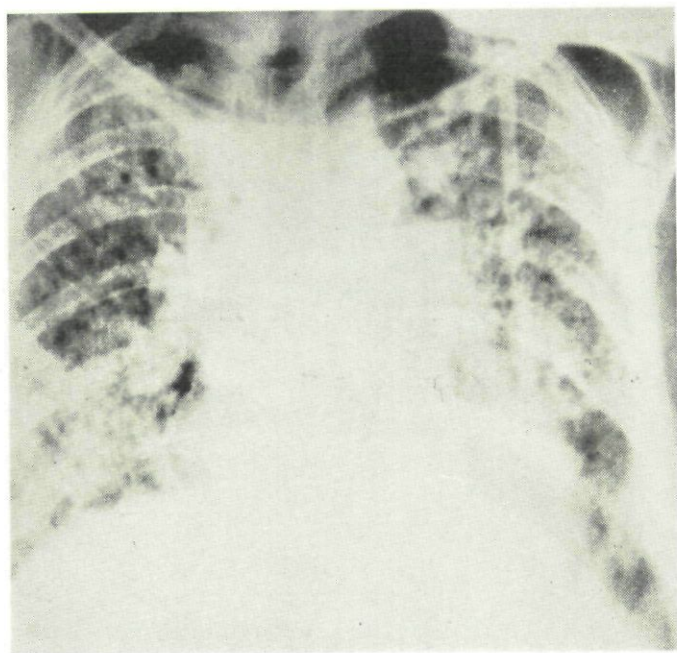


Fig. 1.

X-ray picture of lungs on the twelfth day of disease (top) and on the twentyninth (below). On the thirtieth the ACTH was administered.

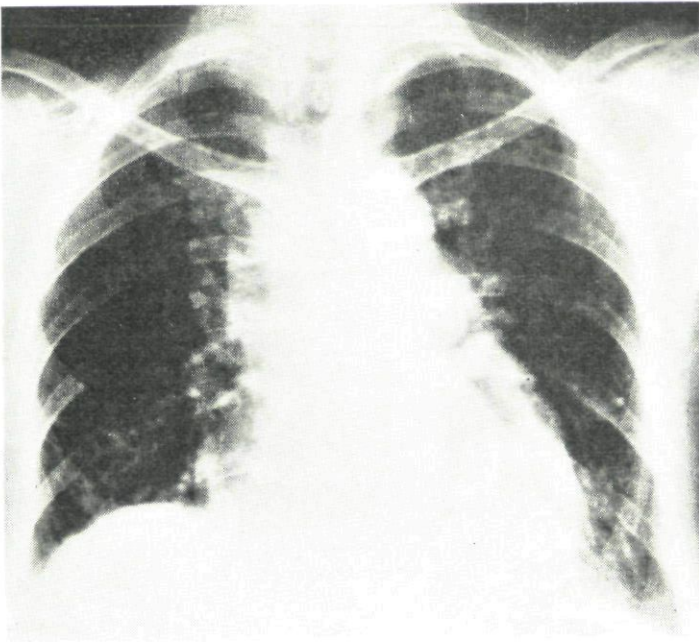
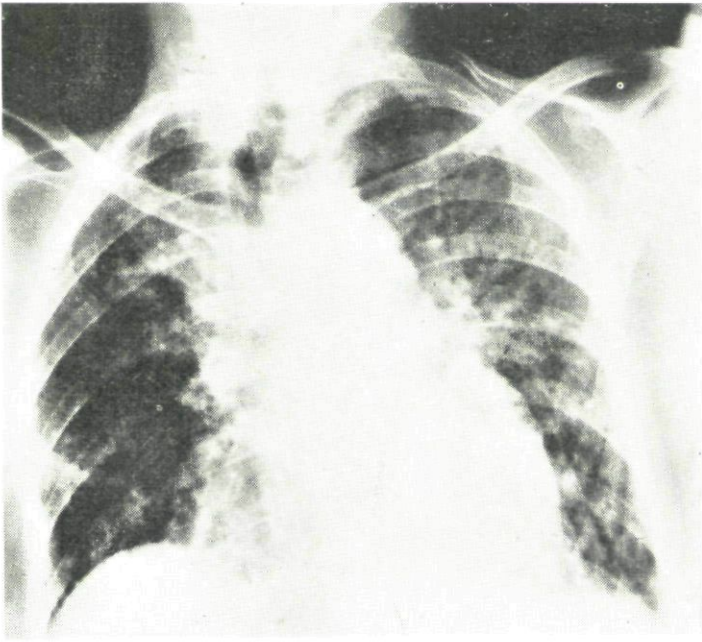


Fig. 2.

X-ray picture on the fourth day of ACTH-therapy (top) and on the eleventh.
It is evident that the infiltrations are disappearing.

were negative. Electrocardiograms showed low voltage and changes of S-T and T that might suggest a pericarditis. The venous pressure was within normal range.

Treatment was continued with intravenous digitalization, oxygen by nasal catheter and aureomycin. In spite of this her condition deteriorated rapidly and within a few days there was little doubt that a fatal outcome could be expected within 24 hours.

It was annoying that no definite etiologic diagnosis had been made for the pulmonary consolidations which apparently were the cause of the patient's distress. Many suggestions, however, were offered by the staff. It was obvious that this was not a case of common pneumococcal pneumonia, nor was it that of an atypical pneumonia. No evidence of tuberculosis was found. A rapidly progressing carcinomatous invasion of the lungs from some undiscovered and silent primary focus was seriously considered. The signs of heart failure were, though, so conspicuous that a cardiac disease was commonly thought to be the most likely explanation. A coronary failure or a pericarditis with chronic increasing pulmonary edema was the diagnosis that had most advocates. The failure of response to cardiac therapy did not contradict this opinion.

The last choice was that the lung disease should be connected with the gold medication given previously for rheumatoid arthritis. The increased value of the total eosinophils in spite of her high fever was an argument in favor of this assumption. It is well known that gold gives rise to different allergic phenomena although such a pulmonary reaction was unknown to us. It was, however, concluded that a course of ACTH should be given on the suspicion that her pulmonary infiltrations were due to an allergic gold reaction.

The strong possibility that she instead might have heart failure due to for instance myocardial infarction, did not make this decision too attractive or even popular.

ACTH was given in a dose of 10 mgms every six hour. In order to minimize the risk of sodium and fluid retention with consequently increasing pulmonary edema, one dose of a

mercurial diuretic was given simultaneously with the first dose of ACTH.

The response to therapy was dramatic. The following morning the temperature was normal, having dropped from 39.2° to 36.4° C. Her dyspnoea had decreased and the cyanosis had gone. X-ray examination which could not be performed until the fourth day of this regimen showed marked improvement. The consolidations were smaller and a return to the patchy type of infiltrations was discernible. The sedimentation rate dropped in a few days from 93 to 11 mm an hour. All other symptoms of a successful ACTH-course followed with increasing appetite and sense of general well-being. Most of the x-ray changes disappeared during the first week of therapy, as a matter of fact, during the first four days. See the roentgenograms. The total eosinophil count decreased to 78 per cu. mm. No fluid retention occurred. The ecg became normal.

She was treated with ACTH for three weeks and discharged after 7 weeks stay in the hospital. A follow-up made 4 months later found her in comparatively good health without any abnormal cardiac or pulmonary findings. Her sedimentation rate was still not more than 10 mm an hour but she was somewhat bothered by her bad knee.

DISCUSSION

The rapid response to ACTH and the subsequent smooth course of the disease excludes most of the discussed diagnoses except an allergic pulmonary reaction. This did not at all have the character of a bronchial asthma and the patient did not have any previous history of allergy. Not even during the most serious phase of the disease could any wheezing sounds be heard. It was quite apparent that the eosinophils were increased particularly when her high febrile condition is considered. The response of the eosinophils to ACTH excludes that the cause of this could be a disease of the suprarenals.

The absence of bronchial symptoms speaks in favor of the assumption that it was a vascular reaction to gold that had taken place. This possibility made it essential to find out whether she had any signs of periarteritis nodosa. A muscular biopsy from one deltoid muscle was done on the fifth day of ACTH therapy. The histological picture was quite normal. The subsequent course of the disease does not suggest this diagnosis.

In our opinion it must have been an allergic pulmonary edema, a reaction of the pulmonary capillaries with increased vascular permeability. The Loeffler syndrome with eosinophilic infiltrations is an essentially benign disease which also is of a transitory character. Moreover no intestinal parasites were found. The peculiar fact that she had no other signs of gold sensitization than these pulmonary findings is worth noting as well as the complete absence of any signs of asthma. The long duration, in all four weeks, with a slow but steady progress of the disease should also be pointed out. Finally the prompt recovery on ACTH indicates that this treatment is essential.

A similar case in an allergic female has been described by Vines and Clark (1951). Their case was also severely ill and responded well to ACTH when this finally was given. The cause of the allergy was unknown. Sante and Wyatt (1951) call attention to a special type of pulmonary edema which is localized to the central parts of the lungs leaving a marginal zone free. They found this in seven patients of which all were critically ill and some died. Three suffered from acute nephritis, two from sulfa drug sensitization and two had periarteritis nodosa. They believe this type of reaction to be characteristic of allergic response of the pulmonary vessels. They therefore coined the name antigenic or azotemic pulmonary edema. The present patient did not, however, have the type of x-ray picture as described by Sante and Wyatt.

No similar cases are reported in the literature. The serious condition and the rapid response to ACTH make it important to recognize them in order to institute a lifesaving therapy.

SUMMARY

A case of severe pulmonary disease apparently due to gold sensitization is described. It is assumed that the reaction took place in the lung vessels. ACTH brought the life-threatening disorder under immediate control.

REFERENCES

1. *Vines, R. W. & Clark, D.*: New Eng. J. Med. 244, 826, 1951.
2. *Sante, L. R. & Wyatt, J. P.*: 66, 527, 1951.

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