

CASE OF CHRONIC BERYLLIUM POISONING FROM ATOMIC ENERGY DEVELOPMENT

ROBERT J. HASTERLIK, M.D.
CHICAGO

THIS REPORT concerns itself with the treatment of a 36 year old physicist ill with beryllium poisoning since the spring of 1948.

During the period 1938 to 1941 this man worked in the physics department of an Eastern university and was specifically involved in the early studies on neutrons and neutron sources. On several occasions he made up neutron sources and in so doing weighed out in an open room, without ventilation, 10 Gm. samples of finely powdered beryllium metal. In 1941 he joined the staff of the University of Chicago Metallurgical Laboratory and was engaged in the building of the first nuclear reactor. The patient was exposed to beryllium oxide bricks which were stacked in his laboratory, often handled, and moved about.

A routine roentgenogram of the chest made on Jan. 6, 1946 was interpreted as follows: "The lung fields are diffusely granular in appearance and look subjectively like silicosis." At that time the patient was asymptomatic and so did not consult a physician.

This man had always been physically vigorous. However, in the spring of 1948 he noted that he was unable to play tennis without marked shortness of breath. By July 1948 he was unable to engage in any athletic activities and entered the University of Chicago Clinics for study.

A roentgenogram of the chest made at the clinics revealed numerous granulomas scattered throughout both lung fields, sparing the bases for the most part. The hilar lymph nodes were grossly enlarged bilaterally. Physical examination revealed a 20 lb. (9 Kg.) loss of weight, slight cyanosis, numerous moist rales throughout both lungs and other abnormalities in accord with the usual clinical picture.

The vital capacity in July 1948 was 3.6 l. and gradually fell to 2.2 l. during the next 18 months accompanied with progressive increase in the symptoms, further loss of weight, deepening cyanosis and clubbing of the distal phalanges of the fingers and toes. Progressive unfavorable changes were also noted in the chest roentgenograms. In 1949 and early in 1950 the patient was hospitalized on several occasions for rest in bed and other nonspecific therapy, with but slight improvement on each occasion. On March 16, 1950 he was hospitalized in the University of Chicago Clinics, and on March 23, 1950 treatment with the pituitary adrenocorticotrophic hormone (ACTH, corticotropin) was begun.

The vital capacity on the day of institution of therapy was 2.2 l., the venous pressure 19.8 cm. of water, and the patient's weight 128 lb. (58 Kg.). Therapy was begun with 100 mg. of ACTH, which was given in four doses at six-hour intervals. Certain ancillary studies, such as dextrose tolerance tests, determinations of the blood sodium and potassium levels, liver function tests and eosinophil counts were done but will not be described.

By the fourth day of therapy the patient for the first time felt much improved. On the twelfth day the dosage was reduced to 70 mg. daily. On this date the vital capacity had risen to 3.6 l. and the venous pressure had fallen to 7 cm. of water. The patient was given permission to leave the hospital for an hour and a half; he went to his laboratory, climbed several flights of stairs and was able to walk rapidly without dyspnea. Only an occasional rale was heard on auscultation of the chest.

Read at a meeting sponsored by the Occupational Medical Clinic at the Massachusetts General Hospital, Boston, Dec. 13, 1950.