Clinical and other Notes

A CASE OF PULMONARY TUBERCULOSIS AND ADDISON'S ANÆMIA.

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The patient, a soldier, aged 20, stated that his previous health had been excellent. He played football, and had won medals for long-distance running. He admitted, however, that since January, 1929, when appendectomy was performed, he had not felt very well. He had been noticeably pale since coming to India early in 1930, and when transferred in April to Jalapahar, a hill station, at an altitude of about 8,000 feet, he began to get breathless and weak. He had no cough or night sweating, but lost weight slightly.

In May, 1930, he was admitted to hospital in Jalapahar for "tachycardia," and he was slightly anaemic at this time. In July he was readmitted with a mild pyrexia, and had an erythrocyte count of 4,480,000; on August 7 he was transferred to Lebong.

His family history furnished no suggestion of haemopoietic disorders or of tuberculosis.

The conditions found by clinical and laboratory examinations were briefly as follows: No emaciation, extreme lemon-yellow waxy pallor, teeth sound, tongue not inflamed but its edges slightly indented by the teeth.

The heart was not enlarged; a soft systolic murmur was audible over the whole precordium, maximal at the pulmonary area, and not conducted in any direction.

Blood-picture: Red cells 2,090,000, haemoglobin 45 per cent, colour index 1.07, leucocytes 5,000, with relative increase of lymphocytes. A blood-film was not grossly abnormal; it showed poikilocytosis, anisocytosis, and slight polychromatophilia, but no nucleated red cells. A platelet count was not made, but the film contained very few platelets. The spleen was not enlarged.

The central nervous system was normal, no evidence of postero-lateral sclerosis. There were harsh vesicular breath sounds with medium pitched crepitations at the apex of the lower lobe of the left lung; some mucopurulent sputum, microscopic examination of which was negative. Blood-culture was sterile, and no haemolytic streptococci were found in swabs from the throat and nasopharynx. Whilst the fasting juice of the stomach showed a low acidity, the acidity after a test meal was within normal limits. There was nothing to suggest sprue, nor any evidence of helminthic infection. A tentative diagnosis of Addison's anæmia was made, but treatment with raw and lightly-cooked liver and with liver extract produced no improvement, and his blood-picture steadily became more typical of true Addison's anæmia, until, on October 14, 1930, the red cells numbered 1,720,000, the colour index was 1.4, and a film contained
three megaloblasts and one normoblast, the changes in the red cells, already noted, being much more marked, punctate basophilia being added to them.

Meanwhile, his pulmonary condition had also progressed unfavourably, physical signs of pulmonary tuberculosis being present in both lungs, and tubercle bacilli in the sputum. On October 16, as he was becoming steadily more ill, and his blood-picture showed no signs of improving, despite treatment with liver, liquor arsenicalis, and graduated doses of direct sunlight, he was given a transfusion of whole blood. The patient stood the procedure very well, and within three days his temperature, which twelve days previously had been very high, was normal, and remained so, except for a few days of low pyrexia, until he left India three months later. He gained in weight, and improved sufficiently to be transferred to Calcutta for ultimate transfer to England. His red cells, however, never numbered more than 2,250,000, and his last count before leaving India was 1,160,000.

In this case there was no doubt as to the correctness of the diagnosis of pulmonary tuberculosis, but it is interesting to consider if the findings justified also a diagnosis of Addison's anaemia.

In the available medical literature no mention has been found of tuberculosis producing anaemia of a pernicious type, and the only grounds for doubting that this was a case of true Addison's anaemia appear to be:

(1) The normal gastric acidity, and (2) the absence of any response to liver treatment, not even an increase of the reticulocytes being observed.

As regards (1), although achlorhydria is almost invariably a feature of Addison's anaemia, a normal acidity need not negative a diagnosis of this disease in the presence of pulmonary tuberculosis, in the early stages of which condition a hyperchlorhydria has been described by many writers. (2) The comparatively slight morphological changes in the erythrocytes at first and the late appearance of nucleated forms might suggest that the anaemia tended to be of aplastic type, in which case treatment would be unavailing. In favour of this is the age of the patient—Addison's anaemia being rare before the third decade of life—but strongly against it are the facts that nucleated and distorted forms were eventually prominent, that the leucocytes were of normal granular type, and that the patient was alive six months after the onset of the condition.

I consider, therefore, that pulmonary tuberculosis and Addison's anaemia co-existed in this patient, and that in all probability the anaemia was the primary condition, rendering him more susceptible to tuberculosis.

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