The anaemia of lead poisoning

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SYNOPSIS Three cases of lead poisoning with a hypochromic anaemia, despite adequate iron stores, are presented and discussed.

The anaemia of lead poisoning is usually thought to be normochromic and normocytic, and punctate basophilia is regarded as a constant feature of the well-developed case. Amongst six proven cases of chronic lead poisoning seen in this Department in the last few years, two of the patients were not anaemic and in a third the degree of anaemia was very slight. We propose to comment here only on the remaining three patients, all of whom showed well-established anaemia. In all three the peripheral blood findings were those of iron deficiency; in only one case was punctate basophilia found.

Formerly the anaemia in lead poisoning was attributed to a shortened survival of cells in the circulation but more recently the importance of interference with haemoglobin synthesis has been recognized, and this is demonstrated in two of our three patients in whom an inability to use available iron despite active erythropoiesis was an important factor in the development of the anaemia. Only in the third did the haemolytic element predominate.

CASE HISTORIES

C.S., a lead burner, aged 41 years, who had twice previously been treated for plumbism, complained of increasing weakness of his hands. After investigation lead poisoning was again diagnosed. The blood findings initially were Hb, 8·7 g./100 ml. (59%), P.C.V., 30·5%, M.C.H.C., 28·5%, R.B.C.s, 4·2 m./c.mm., M.C.V., 73 cμ, M.C.H., 21 μg., reticulocytes, 7·5%. Punctate basophilia was not seen. The serum iron level was 137 μg./100 ml. and the total iron-binding capacity 260 μg./100 ml. Marrow examination showed normoblastic erythropoiesis with excess stainable iron. The uptake of $^{51}$Cr $T_1$ was 26 days; on electrophoresis of the serum there was no apparent reduction of the haptoglobin. Urinary urobilinogen levels were 3·4, 6·5, and 7·7 mg./24 hours on three different days. Faecal urobilinogen measured on two occasions was 265 mg. and 687 mg./24 hours. The M.C.H.C. remained between 28·5% and 29·5% until oral treatment with versene began. The patient then had a lead diuresis and the blood values gradually rose to Hb, 13·5 g./100 ml. (91%), P.C.V., 46·5%, and M.C.H.C., 31·25%.

R.C., a lead burner, aged 45 years, presented with abdominal pain and a gross blue line on the gums. Blood findings were Hb, 9·3 g./100 ml. (63%), P.C.V., 32·75%, M.C.H.C., 28·5%, R.B.C.s, 4·3 m./c.mm., M.C.V., 76 cμ, M.C.H., 21·75 μg., and reticulocytes, 5·5%. Punctate basophilia was not seen. The serum iron was 78 μg./100 ml. and the total iron-binding capacity 450 μg./100 ml. Urinary urobilinogen levels were 5·7 and 11·9 mg./24 hours on two occasions. Urinary coproporphyrins showed a daily excretion of over 1,000 μg. Stainable iron was present in the marrow in spite of the peripheral hypochromia and punctate basophilia was seen in red cell precursors. The uptake of $^{51}$Cr $T_1$ was 32 days and the plasma haptoglobin 145 mg./100 ml. (normal 45-150 mg./100 ml. as methaemoglobin).

M.S., a housewife, aged 30 years, had taken large quantities of lead and opium pills for diarrhoea for many years. The blood lead level was 80 μg./100 ml.; there was a blue line on the gums and the daily urinary coproporphyrin excretion exceeded 700 μg. Blood findings were Hb, 7·8 g./100 ml. (53%), P.C.V., 26·25%, M.C.H.C., 30%, and reticulocytes, 22%. Punctate basophiles were readily found only in buffy coat smears. The serum iron level was 160 μg./100 ml. and the total iron-binding capacity 335 μg./100 ml. The urinary urobilinogen level was 9·3 mg./24 hr., and the uptake of $^{51}$Cr $T_1$, 18 days. Marrow examination showed normoblastic erythropoiesis with a very great excess of stainable iron. The patient responded satisfactorily to versene which greatly increased the excretion of lead in the urine. The blood values rose to Hb 12·4 g./100 ml. (84%), P.C.V., 40%, and M.C.H.C., 31%.

DISCUSSION

More than one mechanism has been blamed for the production of the anaemia of lead poisoning. The red cells may be rendered more 'brittle' by the lead (Aub, Fairhall, Minot, and Reznikoff, 1925) and therefore may have a shorter life span than normal.

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Lead poisoning may interfere with attempts to compensate for this haemolytic state by inhibiting either the incorporation of iron into the protoporphyrin nucleus or the production of the porphyrin part of haem (Eriksen, 1955; Goldberg, Ashenbrucker, Cartwright and Wintrobe, 1956). The utilization of iron would therefore be prevented either directly or indirectly. In all our three cases it seems probable that increased red cell destruction was concerned in the genesis of the anaemia as shown by the raised reticulocyte count and, in the third case, a very active haemolytic process was the chief cause. In the other two cases, although neither the $^{51}$Cr T½ nor the haptoglobin concentration was reduced, the increased urobilinogen excretions confirmed the reticulocytosis in suggesting that some degree of increased blood destruction was present. More significant, however, was the finding of a peripheral hypochromia in all three cases despite adequate stores of iron in the bone marrow. This indicated that haemoglobin synthesis was selectively depressed and by thus lagging behind red cell formation materially contributed to the production of anaemia in each of these three cases of lead poisoning.

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REFERENCES