Hematology Quiz – Case 10

A 48-year old Greek worker in a small size iron foundry presented to our Department because of abdominal pain and teeth discoloration. His family history was unremarkable apart for an upper Gl bleeding that was attributed to peptic ulcer some years before. No other clinical signs or other symptoms were evident for a long time after he noticed teeth discoloration (figures 1, 2). He had visited many dentists before referred to us for further investigation. His present main complaint was abdominal pain, resembling that of an irritable colon. Physical examination was negative, but due to his upper GI bleeding history he was tested for H. pylori by a breathing test that proved to be positive. Accordingly he was put under a H. pylori eradication scheme consisting of proton-pump inhibitor plus antibiotic.

The blood count revealed mild anemia (Ht: 36.9%, hemoglobin 11.9 g/dL), WBC 7,500/μL (normal absolute numbers of poly- and lymphocytes), platelets 222,000/μL. Laboratory tests were as follows: BUN=0.58 mg/dL, creatinine 1.0 mg/dL, total bilirubin=0.95 mg/dL, SGOT=17 IU/L, SGPT=17 IU/L, alkaline phosphatase=162 IU/L, γ-GT=16 IU/L, LDH=293 IU/L, CPK 32 IU/L and total proteins 7.3 g/dL. Following a careful peripheral blood morphology examination (fig. 3), he was further tested for blood lead levels that were found to be abnormally high (602 μg/L, normal values being <100 μg/L). Following a three months’ follow up, abstinence of his occupation (occupational leave) on health grounds and chelation therapy for 20 days with succimer (DMSA), the lead blood levels are <20 μg/L, on repeated tests.

Comment

Lead intoxication is a reason for sideroblastic anemia because of heme synthesis inhibition. Lead inhibits the delta-aminolebulinic acid synthetase (change of delta-ALA to porphocholinogen), while it also acts on another enzyme of porphyrin synthesis. Lead is also related with iron metabolism, contacting the intracellular release in the place of ferrochelatase (decrease of iron supply in heme synthesis positions, absence of ring sideroblasts) as well as with decrease of hemoglobin chains synthesis and with increase of red cell destruction (inhibition of ATPase and perturbation of 5-nucleotidase). Hematological findings vary. Anemia is mild or moderate, mild hypochromia and microcytosis is present; reticulocytosis and sign of hemolysis are also present. A characteristic finding is a coarse basophilic stippling of red cells (ribsomal aggregations, abnormal RNA breakdown because of 5-nucleotidase insufficiency). In bone marrow a hyperplasia of erythroid series is usually present, while bone marrow may be hypocellular, with rare presence of ring sideroblasts. On examination, a decreased ALA synthetase activity, increase of urine ALA content as well as of erythrocytic protoporphyrin content are noted.

Lead poisoning events in Greece may be more common than officially recorded. Occupational health surveillance may be incomplete and most physicians or dentists may overlook important signs as teeth discoloration.

Laboratories also, performing regular blood examinations may oversee the classical basophilic stippling occurring in chronic cases. One reason for that may be that automated analysers are now replacing the routine red cell morphology testing by blood slide microscopy. Another reason may be that red cell changes in morphology may be confused with thalassemia changes, a common abnormality in some areas of the world.

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