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A Biermer's Disease Hidden Under an Iron Deficiency Anemia

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Biermer's disease (BD) also called pernicious anemia is conventionally associated with macrocytic anemia. We report the case of a BD revealed by a microcytic iron deficiency anemia which can in many cases be responsible for a diagnostic wandering and reinforces the idea of not ignoring this BD revelation mode.

A 65-year-old woman admitted for anemic syndrome exploration, the symptomatology evolves for 1 year and a half with palpitation, asthenia and dyspnea. She has been menopausal for 15 years, with a medical history of high blood pressure under losartan, osteoporosis under bisphosphonates. No externalized hemorrhage was found at the anamnesis and the patient diet was normal. She was not taking long-term NSAIDs and was never followed for diabetes, dysthyroidism, vitiligo or other autoimmune diseases. Clinical exam found pallor; the patient had no splenomegaly or lymphadenopathy.

A first biological assessment showed a microcytic iron deficiency anemia, the hemogram showed: hemoglobin at 8.5 g/dl (NR: 12-16), mean globular volume at 66 fl (NR: 80-100), white blood cells at 7500 elements/mm^3 (NR: 4000-10000), platelets at 151000 elements/mm^3 (NR: 150000-450000) and ferritin level at 4 µg/l (NR: 30-300).

Erythrocyte sedimentation rate and C-reactive protein were normal. The lipid, phosphocalcic, protein and prothrombin levels were normal, which can rule out a possible malabsorption syndrome. Anti-endomysium, anti-gliadin and anti-transglutaminase antibodies were negative. The blood smear found no abnormality excepting microcytosis. Gynecological examination as well as the pelvic ultrasound excluded a gynecological origin of the anemia. The colonoscopy was normal. Esogastroduodenal endoscopy showed a hiatal hernia with fundic atrophy and discreet erythematous antritis. Histological study was in favor of atrophic type A gastritis at the fundic level without duodenal atrophy and Helicobacter pylori-negative gastritis.

In view of these endoscopic and histological data, BD was suspected, the assessment was completed by the dosage of vitamin B12 which was lower than 85 pmol/l (NR: 133-675), the folic acid level was normal. Parietal cells antibodies and intrinsic factor antibodies were highly positive. Fasting gastrinemia was at 416 pg/ml (NR<100). The reticulocyte rate was 45000 elements/ml (non-regenerative anemia). Subsequently, a myelogram was performed which showed isolated medullary megaloblastosis with nucleo-cytoplasmic asynchrony. The diagnosis of BD was then retained and the patient was treated with intramuscular injection of hydroxocobalamin at a rate of 1000 µg/day for seven days, then 1000 µg/week for four weeks, then 1000 µg/month for life associated with an iron substitution by ferrous sulfate.

BD is characterized by atrophic fundic gastritis of autoimmune origin with achlorhydria resistant to stimulation and a collapse of intrinsic factor secretion leading to malabsorption of cobalamin. Generally, vitamin B12 deficiency leads to late onset megaloblastic macrocytic anemia. Iron deficiency can, however, appear early. It is explained by the non-transformation of food ferric iron into ferrous iron within the gastric cavity due to hypochlorhydria, the gastric pH becomes greater than three and consequently the ferric iron becomes insoluble and precipitates [1].

The relationship between iron deficiency anemia and BD was first discussed by Dickey et al. [2]. In their study, out of 41 patients with microcytic anemia without associated bleeding, seven of them presented hypergastrinemia associated with positive parietal cell antibodies and or intrinsic antifactor antibodies. A study by Lagarde et al. [3], confirms the non-exceptional nature of the association which exists between BD and iron deficiency. This cohort study identified 95 patients with BD, 16 of whom had iron deficiency and 9 of whom had associated microcytosis. Thus, the contribution of the mean globular volume as a main marker in the etiological reasoning of anemia must, therefore, be more relativized especially in the elderly, as is the case with our patient. Vitamin B12 deficiency is extremely common in the elderly (10-24%), but because of its crucial role in all hematopoiesis, their deficit is the cause of anemia which is not always macrocytic [4,5].

The aim of this clinical case is to reinforce the idea of not ignoring this BD revelation mode. This therefore incites to perform, in addition to the duodenal biopsies, fundic and antral biopsies in search of an isolated fundic atrophy during an esogastroduodenal endoscopy made as part of an etiological assessment of iron deficiency with absence of associated documented bleeding.

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