

ent clone of cells either secondary to the same etiologic agent that produced the WM or secondary to another etiologic factor, the simultaneous occurrence of the two processes being coincidence. In the case reported by Ligorsky *et al.*,⁸ both macroglobulinemia and leukemia cells contained the same immunoglobulin; a possible explanation for this could be that blast cells engulf the paraprotein secreted by the plasma cells. Finally, as in the case reported by Allen⁶ a decrease in the IgM levels was observed simultaneously with the reduction of blasts; the author speculates the possibility that this IgM was produced abnormally by blasts. This circumstance has not been confirmed in subsequent cases.

All subtypes of AML according to the FAB classification, except M3 and M5 have been reported. Although different degrees of cytopenia have been reported as preceding the development of AML in 9 cases, the simultaneous existence of myelodysplasia¹² was reported in only 2 cases. A decrease in the serum IgM levels could not predict the development of AML, as has been suggested by several authors in other secondary neoplasms following WM⁵ (only present in 1 of the 9 patients in which this data was available). Response to treatment is poor and the survival is very short.

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Key words

Waldenström macroglobulinemia, acute myeloid leukemia

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Idiopathic thrombocytopenic purpura associated with Crohn's disease

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Besides cytopenia related to treatment, several hematological disorders such as anemia, abnormal platelet activity, thrombosis, presence of anticardiolipin or anti-neutrophil antibodies, cyclic neutropenia, and myelodysplasia, have been reported in patients with Crohn's disease (CD).¹⁻⁴ The case we report here is the first one documenting the association of idiopathic thrombocytopenic purpura (ITP) with CD.

A 19-year-old woman was referred to our department for microcytic hypochromic anemia (Hb: 7.2 g/dL, MCV: 55 μm^3 , MCHC: 29%) and thrombocytopenia ($20 \times 10^9/\text{L}$).

At admission, physical complaints consisted of fatigue, diarrhea (3 to 4 stools a day) and weight loss (6 kg in 6 months); physical examination was normal. Biological findings, including bone marrow aspiration smears analysis, led to the diagnosis of ITP associated with iron-deficiency anemia. No antiplatelet antibodies were detected. Upper gastrointestinal endoscopy and full colonoscopic examination disclosed ulcerative inflammation of the duodenal cap, polypoid lesions and linear ulcerations on the terminal ileum. Histopathologic analysis of large bowel and ileum biopsies found a lymphocytic inflammatory infiltrate of the mucosa associated with granulomas composed of epithelioid and giant cells consistent with the diagnosis of CD. Successively, standard dose prednisolone, intravenous γ -globulins, and splenectomy failed to

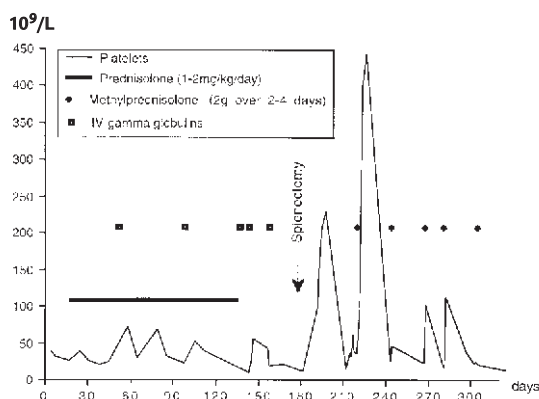


Figure 1. Platelet counts and treatments.

correct the platelet level (Figure 1). Finally, intravenous bolus of high dose methylprednisolone were administered, inducing a slight and transient increase of platelet rate. At that time, the patient refused any other treatment and did not have a follow-up. As for the CD, clinical improvement was observed regarding disappearance of diarrhea and a Crohn's disease activity index of less than 50, compared with 298 at diagnosis (CDAI < 150: quiescent phase; 150 < CDAI < 450: acute attack; CDAI > 450: very severe);⁵ ulcerative inflammation of the duodenal cap persisted and CD granulomas were found on gastric, ileal and colonic biopsies. Anemia responded to iron supplementation.

Anemia is a frequent finding in CD patients, mainly due to iron deficiency (as a result of chronic intestinal bleeding, iron malabsorption, or impaired dietary intake) and chronic inflammation, or to cobalamin and/or folate deficiencies or inadequate erythropoietin production.⁶ Humoral and cellular immune mechanisms contribute to the onset of chronic inflammatory bowel diseases (CD and ulcerative colitis). Chronic T-lymphocyte activation, abnormalities in the production of γ interferon and α tumor necrosis factor which affect B-cell proliferation and differentiation into immunoglobulin secreting cells, infiltration of plasma cells into mucosa with increased local production of IgG have been reported in CD patients.^{7,8} Association of chronic inflammatory bowel diseases with autoimmune cytopenias might be more than coincidental and account for the same immune dysregulation. At least five cases of ITP have been reported in patients with ulcerative colitis.⁹ Whatever the relationship between CD and ITP in our patient, co-existence of these two disorders complicated their respective clinical courses. Corticosteroids and γ -globulins have been shown to reduce bowel inflammation in some patients with CD;¹⁰ initially administered to treat severe

thrombocytopenia, they induced clinical improvement of CD but failed to correct platelet rate in our patient.

Key words

Idiopathic thrombocytopenic purpura, Crohn's disease

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More on the appropriate fluorochrome-conjugated CD34 antibody choice for the flow cytometric detection of circulating progenitor cells

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We have collected data showing that the phycoerythrin (PE)-conjugated 8G12 (HPCA-2) CD34 MoAb allows an increased flow cytometric resolution of small number of circulating CD34⁺ hematopoietic cells.