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Immunologic Thrombocytopenic Purpura Associated with Helicobacter Pylori Infection: A Case Report from Senegal

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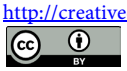

Abstract

We present a 27-year-old female patient received for epistaxis associated with gingivorrhagia. In her medical history, she had undergone an oesogastroduodenal fibroscopy one year earlier, which revealed a bulbar ulcer with gastritis. On admission, she presented with a bleeding syndrome, an anemic syndrome with poor hemodynamic tolerance, and epigastric sensitivity. The blood count performed in the emergency on the citrate tube showed a bicytopenia (regenerative anemia + thrombocytopenia). The HELIKIT test was positive. Thus, the hypothesis of an acute immunological thrombocytopenic purpura associated with a Helicobacter pylori infection seemed to us the most probable with a Khellaf hemorrhagic score of 8. She had benefited from a bolus of methyl prednisone: 15 mg/kg/day for 3 days then relay per os with prednisone 1 mg/kg/day (and adjuvant treatment); the eradicating treatment of Helicobacter pylori had been simultaneously started.

Keywords

Immunological Thrombocytopenic Purpura, Helicobacter Pylori, Corticosteroids

1. Introduction

Immunologic thrombocytopenic purpura (ITP) or autoimmune thrombocytopenic purpura, formerly known as idiopathic thrombocytopenic purpura, is defined as isolated acquired thrombocytopenia (platelets < 100 G/L) of autoim-

mune origin with or without the presence of purpura [1]. ITP is the most common autoimmune cytopenia in adults [2], the main mechanism of which is increased destruction of platelets by the reticuloendothelial system, particularly the splenic system, and inadequate platelet production by the bone marrow [3].

It can be primary or secondary to dysimmune diseases (systemic lupus erythematosus, anti-phospholipid syndrome, variable common immunodeficiency or lymphoproliferative syndrome with autoimmunity), viral (HIV, hepatitis C virus), bacterial (Helicobacter pylori), hematological (chronic lymphocytic leukemia, Hodgkin's disease, etc.), and can also occur following medication or transfusion [4].

Moreover, Helicobacter Pylori infection affects more than 50% of humans in developed countries; its prevalence reaches 80% or more among adults in Africa [5].

In Senegal, most epidemiological studies on Helicobacter pylori concern the endoscopic population where the prevalence reaches 80% [6]. No data on the extra-digestive manifestations of this bacterium are available. It is in this sense that we report this extra-digestive manifestation of a Helicobacter pylori infection which seems unknown in our context.

2. Observation

We present a 27-year-old female patient living in Matam (Senegal) who was admitted for epistaxis associated with gingivorrhagia, the onset of which was one month before her admission.

In addition, she reported an episode of blackish stools without any notion of iron intake, all evolving in a non-febrile context without alteration of the general state. In her medical history, she had undergone an oesogastroduodenal fibroscopy one year earlier, which revealed a bulbar ulcer associated with gastritis. On admission, she presented with a hemorrhagic syndrome consisting of unilateral epistaxis and gingivorrhagia associated with non-infiltrated ecchymotic purpura on the extension side of the hands; an anemic syndrome poorly tolerated hemodynamically; and epigastric tenderness without a palpable mass.

The blood count performed in an emergency on the citrate tube showed deep thrombocytopenia at 2 G/L associated with normocytic normochromic anemia at 8.5 g/dl strongly regenerative (reticulocytes rate at 463 G/L); the blood smear was normal. The direct antiglobulin test was negative. The labelled urease breath test (Helikit) was positive. The oesogastroduodenal fibroscopy was not feasible because of the anaemia and the profound thrombocytopenia.

The haemostasis test, creatinine levels, liver function tests, serologies (HIV and HCV), serum protein electrophoresis, TSH levels and anti-nuclear antibody tests were unremarkable.

Thus, the hypothesis of an acute immunological thrombocytopenic purpura associated with Helicobacter pylori infection seemed to us the most probable with a Khellaf hemorrhagic score of 8 in favor of a severe hemorrhage. She was

given a transfusion of platelets and red blood cells and a bolus of methyl prednisone: 15 mg/kg/day for 3 days, followed by prednisone 1 mg/kg/day (and adjuvant treatment); treatment to eradicate Helicobacter pylori was started at the same time. The evolution was marked by an improvement of the hemorrhagic syndrome; the control blood count showed a platelet count of 364 G/L, a hemoglobin level of 11 g/dl with a predominantly neutrophilic hyperleukocytosis of 14 G/L. However, the C-reactive protein was negative.

3. Discussion

Helicobacter pylori infection is a model of slow bacterial infection, developing over several decades, testifying to a remarkable adaptation to the gastric niche by various mechanisms (surface infection, urease activity, modulation of the inflammatory response, etc.). If a majority of infected persons have only minimal gastric inflammatory lesions without significant pathological consequences, this equilibrium is disrupted in a minority of patients who will develop associated diseases of varying severity. Thus, since the discovery of this bacterium, many studies have been published concerning its hypothetical role in these various extra digestive diseases [7]. Its association with ITP is therefore known in the Western literature but rarely described in Africa.

The pathophysiological mechanism remains unclear, with several interrelated phenomena (molecular mimicry, circulating immune complexes). The bacterium could play a role in the pathogenesis of ITP, through a direct connection to the platelet, resulting in its destruction. The case of our 27-year-old patient, whose clinical picture combines digestive symptoms and a hemorrhagic syndrome (epistaxis, petechial purpura), is the first reported in Senegal. The Helikit test was used to identify Helicobacter pylori infection. This test is a single administration respiratory test containing 13C-labeled urea. It allows the detection of active infection by demonstrating the urease activity of H. pylori. It is recommended in these situations to confirm the diagnosis with at least one reliable test for the detection of the bacteria [8]. We therefore opted for this test because it is inexpensive, available in our context, rapid and of high specificity. Moreover, upper GI endoscopy was not feasible due to the unfavorable benefit-risk balance for our patient. The search for this infection in our patient was systematic, given its high prevalence in developing countries, as well as her history of gastritis and the reported symptomatology [9].

The patient was put under eradicating treatment as well as corticotherapy in the form of bolus of methylprednisolone and a relay per os at a dose of 1 mg/kg/day for a short duration (21 days). The evolution was favorable, marked by an improvement of the haemorrhages, a disappearance of the gastric symptomatology, and a normalization of all the elements of the blood count and this in a stable way during 1 year, without recurrence.

This has been observed by several teams, which found that eradication of Helicobacter pylori was accompanied by a significant increase in platelets in pa-

tients with autoimmune thrombocytopenic purpura. It should be noted that the first data, mainly from Italy and Japan, were based on relatively short follow-up periods, making it impossible to know whether the efficacy of this possible treatment was long-lasting [10]. However, an Italian team led by Giovanni Emilia and colleagues from the University of Modena reported long-term follow-up results in 75 patients with autoimmune thrombocytopenic purpura. Almost half of these patients (38 cases) had the bacteria (a similar proportion to the population). Eradication was successful in 34 patients, or 89% of those treated. After a median follow-up of 60 months, a durable response to eradication in terms of platelet count recovery was observed in 68% of eradicated patients. Of the remaining patients, one relapsed after showing a response and the others did not respond at all.

The researchers also looked at factors that may influence the risk of developing autoimmune purpura in people with Helicobacter pylori. Compared to controls, they found a higher frequency of strains carrying the CagA virulence gene (70% of patients vs. 26% of controls), particularly associated with vacAs1 and iceA [11].

4. Conclusion

The association of immunological thrombocytopenic purpura and Helicobacter pylori infection is not to be ignored in our context of tropical practice, where the prevalence in the endoscopic population is very high. Thus, the diagnosis of purpura should lead to a systematic search for Helicobacter pylori infection in patients with a history or symptoms of digestive or extra digestive manifestations attributable to the bacteria. This is generally a good prognostic association because eradication of Helicobacter pylori significantly and permanently improves the patient's platelet count in up to 90% of cases.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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