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Case Report

Acute Immune Thrombocytopenia Secondary to Helicobacter Pylori Infection in a Child: A Case Report

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Abstract

Helicobacter pylori (H. pylori) infection is increasingly recognized as a trigger for secondary immune thrombocytopenia (ITP). We report the case of a 4-year-old boy with acute ITP refractory to first-line immunosuppressive therapy who showed significant clinical improvement following the detection and eradication of an *H. pylori* infection.

This case underscores the importance of considering *H. pylori* as an underlying etiology in pediatric patients presenting with ITP, particularly those with gastrointestinal symptoms or a suboptimal response to standard treatment.

Keywords: H. pylori, Gastric cancer, Hematologic disorders, Bacterial eradication, Autoimmune disease, Bleeding.

Introduction

Helicobacter pylori (H. pylori) is a gram-negative spiral bacterium responsible for chronic gastritis, peptic ulcer disease, and gastric cancer. Beyond its gastrointestinal manifestations, *H. pylori* has been associated with various extra-digestive conditions, including autoimmune and hematologic disorders. Among these, the link with immune thrombocytopenia (ITP) is supported by the most robust evidence, with numerous studies demonstrating platelet count recovery following successful bacterial eradication [1].

Primary ITP is an immune-mediated disorder characterized by isolated thrombocytopenia (peripheral blood platelet count <100x10^9/L) and an increased risk of bleeding. When a predisposing condition, such as an infection or autoimmune disease, is identified, the diagnosis is secondary ITP. Infections linked to secondary ITP include hepatitis C, HIV, and *H. pylori* [2]. The association was first documented in 1998 when an Italian group reported that platelet counts increased in 8 of 11 ITP patients following *H. pylori* eradication therapy [2].

The pathogenic mechanisms remain incompletely understood but are thought to involve molecular mimicry, wherein antibodies directed against *H. pylori* antigens (e.g., Urease B) cross-react with platelet surface glycoproteins such as IIIa, leading to their destruction [2]. We present a pediatric case of acute ITP that was refractory to intravenous immunoglobulin (IVIG) and corticosteroids but responded successfully to *H. pylori* eradication therapy.

Case Presentation

A 4-year-old boy, with a known sickle cell trait but no other significant medical history, was admitted to the hospital with a one-day history of a sudden petechial rash on his upper and lower limbs, accompanied by two days of epistaxis. His family reported intermittent epigastric pain and a history of a respiratory infection two weeks prior. He was developmentally normal and fully vaccinated.

On examination, the patient was vitally stable. A petechial

rash and purpura were observed on his face, abdomen, and limbs. The remainder of the physical examination was unremarkable.

Investigations

Initial laboratory studies revealed severe thrombocytopenia with an undetectable platelet count. Hemoglobin was normal at 12 g/dL. Coagulation studies showed a normal activated partial thromboplastin time (aPTT) of 33.9 s, a slightly prolonged prothrombin time (PT) of 15.1 s, and a normal international normalized ratio (INR) of 1.13. Blood smear confirmed leukocytosis with neutrophilia and a left shift, few reactive lymphocytes, and significantly decreased platelets. Bone marrow aspiration revealed normal cellularity with no blasts, ruling out marrow failure or malignancy. Further workup for secondary causes of ITP, including viral studies (EBV, CMV, HIV, Hepatitis B and C) and immunological profiles (complement, immunoglobulins), were negative or within normal limits (Table 1).

Test	Result	Reference Range
Cytomegalovirus (CMV) IgG	Negative	-
Direct Antiglobulin Test (DAT)	Negative	-
Epstein-Barr Virus (EBV) Serology	Negative	-
Human Immunodeficiency Virus (HIV)	Negative	-
Hepatitis C Virus (HCV) Antibody	Negative	-
Hepatitis B Surface Antigen (HBsAg)	Negative	-
C3 Complement	1.08 g/L	0.83 – 1.52
C4 Complement	0.12 g/L	0.13 – 0.37
Immunoglobulin A (IgA)	1.1 g/L	0.3 – 1.5
Immunoglobulin G (IgG)	27.63 g/L	5.4 – 13.6
Immunoglobulin M (IgM)	0.98 g/L	0.4 – 1.5
Immunoglobulin E (IgE)	< 25 kIU/L	-
Von Willebrand Antigen	101%	70 – 130
C-reactive Protein (CRP)	< 1 mg/L	-

Table 1: Results of Laboratory Investigations for Differential Diagnosis of Secondary ITP.

Hospital Course and Treatment

The patient was diagnosed with acute ITP. First-line therapy was initiated with IVIG (administered on 22/07/2020 and 24/07/2020) and oral Prednisolone (starting 24/07/2020). Due to a persistently undetectable platelet count, a pulse dose of IVIG and intravenous Methylprednisolone was administered on 28/07/2020, followed by a tapering course of oral steroids. Esomeprazole was also started. Despite this aggressive immunosuppressive regimen, the platelet count showed a minimal response, rising to only 3x10⁹/L.

Given the patient's history of epigastric pain and the poor response to therapy, an *H. pylori* stool antigen test was performed, which returned positive. Consequently, eradication therapy with clarithromycin and amoxicillin was initiated on 04/08/2020 for a 15-day course. Following this treatment, the patient's platelet count began to rise steadily. He was discharged on 23/08/2020 with a platelet count of 40x10⁹/L on a tapering dose of oral Prednisolone and Pantoprazole.

Follow-up

The patient's platelet count normalized to 159x10⁹/L by 15/11/2020 and has remained
within normal limits since, confirming a sustained response.

Date (2020)	Platelet Count (x10⁹/L)	Key Clinical Event
21-Jul	0	Admission (undetectable)
22-Jul	0	First IVIG dose
24-Jul	0	Second IVIG dose; Start Prednisolone
27-Jul	0	Start Methylprednisolone & Esomeprazole
28-Jul	0	IVIG Pulse Dose

30-Jul	3	Post-IVIG/Steroids - Minimal Response
4-Aug	3	Start H. pylori Eradication Therapy
23-Aug	40	Discharge
15-Nov	159	Follow up, Normalized Platelet Count

Table 2: Trend of Platelet Count During Hospitalization and Follow-up.

Discussion

This case illustrates a classic presentation of acute ITP in a child that was unresponsive to standard first-line therapies. The pivotal step was the investigation for secondary causes, which led to the identification of an *H. pylori* infection. The temporal relationship between the initiation of eradication therapy and the subsequent platelet recovery strongly suggests a causative role for *H. pylori*.

The association between H. *pylori* and ITP is well-documented, particularly in adults, with systematic reviews indicating that over 50% of patients may achieve platelet count recovery after eradication therapy [7]. Evidence in pediatric populations, while less extensive, is growing. A randomized controlled study from Iran found that 64.2% of children treated for both ITP and *H. pylori* achieved a complete response, compared to a cohort receiving ITP treatment alone [8]. Our patient's course mirrors these findings; his platelet count rose to only 3x10⁹/L after immunosuppression but increased markedly to 40x10⁹/L following antibiotic therapy, eventually achieving full normalization.

While childhood ITP often follows a benign, self-limiting course, an estimated 15-20% of cases progress to chronic disease [5]. Therefore, identifying and treating underlying triggers like *H. pylori* is critical not only for managing the acute presentation but also for potentially preventing chronicity. The non-invasive nature of *H. pylori* testing (stool antigen or urea breath test) and the favorable safety profile of eradication therapy make it a reasonable investigation in the initial workup of ITP patients, especially those with gastrointestinal symptoms or an atypical response to treatment [3, 4].

Conclusion and Recommendation

This case adds to the body of evidence supporting the role of *H. pylori* as a treatable cause of secondary ITP. We recommend that screening for *H. pylori* infection be considered in the diagnostic workup of children presenting with acute ITP, particularly in cases refractory to conventional therapy or when accompanied by suggestive symptoms like abdominal pain. Eradication therapy is a low-risk intervention that can lead to significant clinical improvement and may prevent the evolution to chronic ITP.

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