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A Rare Case of Thrombocytopenia Suspecting ITP due to Helicobacter Pylori Infection

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Abstract: ***Helicobacter pylori H. pylori is a common bacterium that affects a large portion of the global population, particularly in developing countries. This case report discusses a 28yearold male with immune thrombocytopenic purpura ITP following an H. pylori infection. Initially presenting with fever and general weakness, the patients symptoms persisted despite standard treatment. Further evaluation revealed erosive gastritis with H. pylori infection, which was confirmed through a rapid urease test and histopathology. Following a course of H. pylori eradication therapy, the patients platelet counts gradually improved. This case underscores the importance of investigating H. pylori infection in patients with persistent thrombocytopenia and suggests that H. pylori eradication can lead to an increase in platelet count.***

Keywords: Helicobacter pylori, immune thrombocytopenic purpura, platelet count, eradication therapy, gastritis

1. Introduction

Helicobacter pylori (H. pylori) is a gram - negative, spiral - shaped, flagellated, microaerophilic bacillus that inhabits the stomach and spreads via feco - oral and oral - oral routes. Its prevalence is particularly high in developing countries, affecting over 50% of the global population, either symptomatically or asymptomatically. H. pylori is known to contribute to the development of gastritis, peptic ulcer disease, gastric atrophy, and increases the risk of gastric adenocarcinoma and mucosal - associated lymphoid tissue (MALT) lymphoma. Additionally, H. pylori infection is linked to non - gastrointestinal conditions, including coronary artery disease, pernicious anaemia, immune thrombocytopenic purpura (ITP), and other autoimmune disorders. ITP is typically a diagnosis of exclusion, with its underlying causes remaining uncertain according to various studies. The connection between H. pylori and ITP was first highlighted by Gasbarrini et al., who demonstrated that H. pylori eradication therapy could effectively improve platelet counts in patients with chronic ITP. The prevalence of *H. pylori* infection is high (49.94% - 83.30%) in India, but the incidence of gastric cancer is comparatively low indicating mixed results for the association between *H. pylori* and gastric cancer

Immune thrombocytopenic purpura (ITP) is an autoimmune disorder characterized by the production of autoantibodies against platelet membrane antigens, resulting in the destruction of platelets by the reticuloendothelial system. In healthy adults, platelet counts typically range from 150 to 400 × 10^9/L, with a lifespan of 8 to 10 days. The presence of autoantibodies and immune complexes in the bloodstream can lead to a platelet count dropping to 100 × 10^9/L or lower.

Figure 1: Positive Rapid urease test

2. Case Report

A 28yr old male presented to the emergency with complaints of fever since 5days and generalised weakness in the last 4 days. The patient is a non - smoker, non - diabetic and non - hypertensive was in his usual state of health about 5days ago, when he developed fever. It was sudden in onset, high grade, continuous associated with chills and rigors. It was documented at 102 to 103F and relieved by antipyretics. Fever was associated with generalised weakness, lethargy, and inability to carry out routine activities of daily living. Pt. had no bleeding gums, hematemesis, melena, or haemoptysis. In view of thrombocytopenia pt. was evaluated for thrombocytopenia. No significant family, personal and drug history. General physical examination was normal and on routine evaluation and complete hemogram revealed Hb 10g/dl, TLC 5, 200, Platelet count 45, 000, Peripheral smear showed erythropenia showing normocytic normochromic, platelets showing thrombocytopenia with normal morphology and size of platelets with mild leukopenia. Erythrocyte sedimentation rate 44mm/hr, C - reactive protein 25. Liver function test, renal function test and serum electrolytes were within normal limits. Dengue serology was negative, Peripheral smear and Quantitative buffy coat for malarial parasite was negative. HIV, HBV, HCV viral serology, blood cultures, were negative. So, the case was diagnosed as viral

pyrexia with thrombocytopenia and pt. was initiated on supportive treatment was given. Pt. improved clinically, but platelet count remained low. In view of persistent thrombocytopenia, pt. was further evaluated which showed LDH, PT, aPTT, INR, Reticulocyte counts to be within normal limits. Fever profile and dengue serology was negative. So, suspecting Immune thrombocytopenia purpura, pt. was initially started on steroids, but no improvement in platelet counts were observed. After 10 days, pt. gave history of epigastric pain, bloating and belching's. In view of dyspepsia symptoms, upper GI endoscopy was done which showed erosive gastritis with antral ulcer. A biopsy was taken from the antrum, and a rapid urease test was done which turned out positive. Histopathological examination of biopsy showed loss of superficial epithelium of gastric mucosa with h. pylori bacilli. Pt. was started on h. pylori kit containing Tinidazole 500mg, Amoxicillin 750mg, Omeprazole 20 mg for 14 days. Subsequently, pt. clinically improved. His platelet counts were documented to be in increasing trend in follow up.

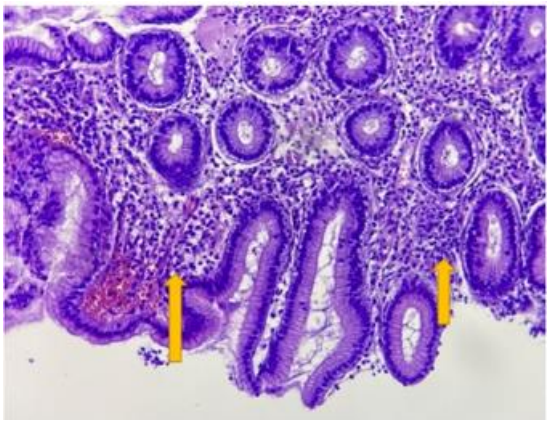

Figure 2: Gastric antral mucosa with massive chronic inflammatory cells: lymphocytes and plasma cells (arrow).

Discussion: Numerous mechanisms have been proposed for the development of immune thrombocytopenic purpura (ITP) linked to H. pylori infection. These include molecular mimicry, platelet aggregation, disruptions in phagocytosis, an enhanced response from plasmacytoid dendritic cells (pDCs), and the host's immune reaction to H. pylori's virulence factors. The pathophysiology of ITP is characterized by the production of IgG antibodies that target platelet surface proteins like GPIIb - IIIa and GIb - IX. However, many aspects of ITP's pathogenesis remain unclear and warrant further research. Additionally, antibodies formed against H. pylori antigens, such as the cytotoxin - associated gene A (CagA), can exhibit cross - reactivity with various glycoprotein antigens (GP IIb/IIIa, GP Ib/IX, and GP Ia/IIa) found on platelet membranes.

Conclusion: Recent literature has highlighted a strong connection between H. pylori infection and the development of immune thrombocytopenic purpura (ITP). Various studies have proposed different mechanisms for H. pylori - induced ITP, with the most cited being molecular mimicry. This involves the production of autoantibodies against the H. pylori virulence factor CagA, which can cross - react with several platelet surface antigens such as GP IIb/IIIa, GP Ib/IX, and GP Ia/IIa. These dendritic cells activate the host immune response, leading to the release of various interleukins. The presence of anti - H. pylori IgG and von Willebrand factor (vWf) on the membranes of different H. pylori strains can result in platelet activation and aggregation. Ultimately, the host immune system responds by producing antibodies against CagA and through the binding of VacA to multimerin - 1 on platelet surfaces, contributing to thrombocytopenic purpura. Notably, H. pylori eradication therapy (triple therapy) has also been shown to significantly increase platelet counts in ITP patients compared to pre - treatment levels. A clear association between ITP and Helicobacter pylori could thus be established.

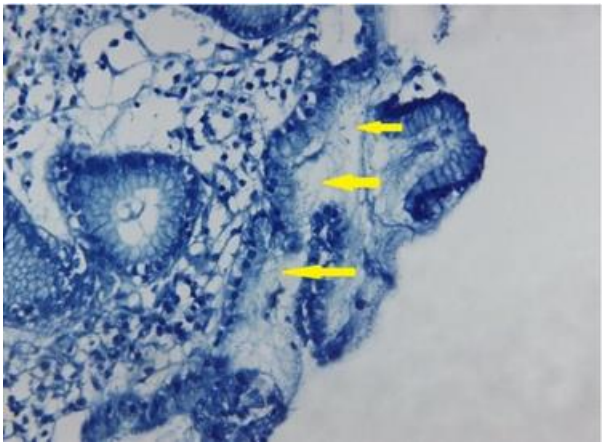

Figure 3: *H. pylori* infection proved by Giemsa stain. Yellow arrow highlights the presence of bacteria in gastric mucosa.

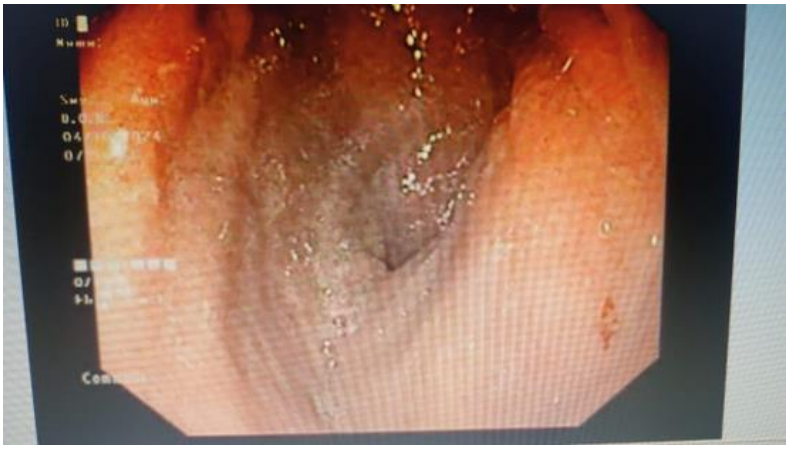

Figure 4: Upper GI endoscopy showing antral ulcer

3. Summary

Though h. pylori infection causing ITP is a rare phenomena, it's possibility should not be excluded. Provided a thorough and proper lab examination and systemic examination has been performed along with a upper GI endoscopy to rule out other possible contributing factors is necessary.

4. Conclusion

This case highlights the potential link between Helicobacter pylori infection and immune thrombocytopenic purpura ITP. Despite standard treatment failing to resolve the patients thrombocytopenia, the subsequent identification and eradication of H. pylori resulted in clinical improvement and an increase in platelet count. These findings suggest that H. pylori eradication therapy may be a valuable treatment option in ITP patients, emphasizing the need for further research into this association.

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