H. pylori eradication: a therapy for
immune thrombocytopenic purpura?
Angela Chen
Pharmacy Department, St Vincent’s Hospital Melbourne

Objective
To present a case where Helicobacter pylori eradication assisted platelet recovery in refractory immune thrombocytopenic purpura (ITP).

Introduction
ITP is an acquired haematological disorder characterised by thrombocytopenia that is primarily due to an autoantibody-mediated destruction of platelets. The current treatment of ITP includes corticosteroids, intravenous immunoglobulin as well as subsequent therapies such as rituximab, TPO-receptor agonist and splenectomy.

H. pylori is a bacterial infection associated with several diseases such as peptic ulcer disease, chronic gastritis, adenocarcinoma and gastric mucosa associated lymphoid tissue lymphoma. There is also a proposed association between H. pylori and ITP.

Our patient is a 42 year old male with no past medical history. He first presented in December 2017 with fluctuating thrombocytopenia initially attributed to his Truvada® pre-exposure prophylaxis and antibiotics for Mycoplasma genitalium (doxycycline and moxifloxacin).

In May 2018, one month after ceasing Truvada® and the suspect antibiotics, he was treated with high-dose dexamethasone. Despite this his platelets dropped to 3x10^9/L in June, confirming the diagnosis of ITP. After two more hospital admissions and further treatment with steroids and intravenous immunoglobulin, his platelets remained low.

Our patient was screened for serum anti-H. pylori IgG antibodies during two hospital admissions and returned a positive result during his second admission in mid-June. During this admission he was commenced on rituximab 100mg weekly and H. pylori triple eradication therapy with twice-daily esomeprazole 20mg, amoxicillin 1g and clarithromycin 500mg. After one week, his platelets had risen to 89x10^9/L from 25x10^9/L.

Despite this initial increase in platelet count, he was readmitted to hospital in July as his platelets had dropped to 10x10^9/L. At the time he had completed two weeks of triple eradication therapy and was due the third of four doses of weekly rituximab. High dose prednisolone was re-commenced at 1mg/kg/day. Follow up in August revealed that our patient had not responded to rituximab and was still reliant on long-term steroids. In addition, a H. pylori breath test conducted just prior to this follow up was negative, two months after completing eradication therapy.

Investigations, Case Progress and Outcomes

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Studies increasingly report improved platelet counts after completing triple eradication therapy. A review of 13 studies revealed an overall response rate of 52%. The time of assessment for platelet response varied from 1 to 6 months after eradication therapy. Some studies allowed for concurrent ITP therapy while others required a washout period. Due to the differences in population, variability of study design and definition of platelet response, comparison of results between studies are complicated. Larger randomised controlled trials are still required in this area.

Our patient experienced an initial improvement in platelet count after 1 week of triple therapy, which some case reports have also described. However commencing concurrent rituximab is a potential confounder. His platelet counts have remained stable since August and appears to be gradually improving since August. The plan is to wean prednisolone at a slower rate.

References

Contact
Angela Chen
Clinical Pharmacist; St Vincent’s Hospital Melbourne
angela.chen@svha.org.au

Discussion and Conclusion

Studies increasingly report improved platelet counts after completing triple eradication therapy. A review of 13 studies revealed an overall response rate of 52%. The time of assessment for platelet response varied from 1 to 6 months after eradication therapy. Some studies allowed for concurrent ITP therapy while others required a washout period. Due to the differences in population, variability of study design and definition of platelet response, comparison of results between studies are complicated. Larger randomised controlled trials are still required in this area.

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Currently no established mechanism exists on H. pylori in the pathogenesis of ITP. However, several theories attempt to explain this platelet response, including molecular mimicry, platelet aggregation induced by H. pylori and potential immunomodulatory effects of eradication therapy.

Clinicians should be vigilant regarding the assessment of H. pylori infection and consider its eradication in ITP as it may be an effective strategy in some patients. Furthermore, H. pylori eradication in ITP patients is a treatment option that should be considered given its low cost and minimal risk of toxicity.