Acute immune thrombocytopenic purpura post first dose of COVID-19 vaccination

The rapid pace of COVID-19 vaccine development and the uncertainty of potential adverse effects have led to concerns about safety and some hesitancy in vaccine uptake. In the UK, four COVID-19 vaccines—ChAdOx1-S (Oxford–AstraZeneca, hereafter ChAdOx1), mRNA BNT162b2 (Pfizer–BioNTech), mRNA-1273 (Moderna) and Janssen—have been authorised for use in the national vaccination programme. Reports of extremely rare adverse events of concurrent thrombosis and thrombocytopenia following vaccination with ChAdOx1 have been well documented and have led to several concerns. Vaccines are thought to be most likely due to virally induced molecular mimicry: the binding of pathogenic antibodies to platelet and megakaryocytes can cause thrombocytopenia by various methods, including opsonisation, direct activation of complement and apoptotic pathways. A case of ITP following a dose of BNT162b2 mRNA in the USA was published. The New York Times recounted 36 reports of ITP following doses of BNT162b2 mRNA and ChAdOx1, which were submitted to the US government’s Vaccine Adverse Event Reporting System. More recently, a Scottish prospective cohort study found an association between ChAdOx1 and ITP, but not with BNT162b2 mRNA. Simpson et al estimated an incidence of 1.13 ITP cases per 100,000 doses. Of note, there were no reported cases of ITP in the 11,636 participants analysed in the earlier AstraZeneca trial. Viral infections, including COVID-19 infection, can also cause acute ITP or worsen stable chronic ITP. Thus, it is essential to rule out COVID-19 infection as a trigger even in those who are asymptomatic.

ITP is an immune-mediated condition in which antibody-coated or immune complex-coated platelets are destroyed prematurely by the reticuloendothelial system, leading to peripheral thrombocytopenia. Diagnosing ITP can be challenging, with no single ‘gold standard’ test to reliably prove the diagnosis and an extensive list of differential diagnoses. The presumptive diagnosis can be made when other causes of thrombocytopenia have been excluded. Serial platelet monitoring after commencing therapy serves dual purposes of assessing response to treatment and providing additional evidence to support the diagnosis. Most cases are mild to severe but are highly responsive to IVIG.

Treatment recommendations have also changed in the setting of the COVID-19 pandemic. Previously, first-line treatment was with steroids and IVIG. The current guidelines from NHS England promote the use of thrombopoietin receptor agonists (TPO-RAs) as first-line therapy for new or relapsed acute ITP in adults and children aged over 1 year. These should be commenced under the guidance of haematologists. However, patients hospitalised with COVID-19 infection requiring steroids for immunosuppression may not be suitable candidates for TPO-RAs. Another therapeutic agent is rituximab, an anti-CD20 monoclonal antibody that binds to the CD20 antigen on the B-cell surface, activating complement-dependent B-cell cytotoxicity. The B-cell depleting effect of rituximab results in a diminished humoral immune response to vaccinations. Thus, rituximab should be avoided or delayed for those patients who have not yet been vaccinated. This is a challenge for those patients already on rituximab immunosuppression for treatment of rheumatic disease, non-Hodgkin’s lymphoma, chronic lymphocytic leukaemia and vasculitis.

Although existing COVID-19 vaccines are associated with a small risk of developing ITP, recent evidence suggest that the overall benefits of vaccination greatly outweigh ITP-associated risks. We suggest that patients with pre-existing ITP have a baseline platelet count recorded prior to vaccination and successive counts depending on their clinical history. It is important that clinicians are made aware of the potential to develop ITP secondary to the COVID-19 vaccines and to seek early input from clinical haematologists if patients become thrombocytopenic or exhibit clinical features suggestive of this.

Jessica Sue Yi Wong, James Hong-En Kang, Kyaw Zin Maw

1Haematology, James Paget University Hospitals NHS Foundation Trust, Great Yarmouth, UK
2Gastroenterology, James Paget University Hospitals NHS Foundation Trust, Great Yarmouth, UK

Letter

Postgrad Med J March 2022 Vol 98 No e2

e129
Letter

Correspondence to Dr Jessica Sue Yi Wong, James Paget University Hospitals NHS Foundation Trust, Great Yarmouth NR31 6LA, UK; Wongsyjessica@gmail.com

Contributors JSYW wrote the original draft of the manuscript. JH-EK revised the manuscript. All authors reviewed and approved the final version of the manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; internally peer reviewed.

This article is made freely available for personal use in accordance with BMJ’s website terms and conditions for the duration of the covid-19 pandemic or until otherwise determined by BMJ. You may download and print the article for any lawful, non-commercial purpose (including text and data mining) provided that all copyright notices and trademarks are retained.

© Author(s) (or their employer(s)) 2022. No commercial re-use. See rights and permissions. Published by BMJ.


Accepted 15 August 2021
Published Online First 26 August 2021

doi:10.1136/postgradmedj-2021-140947

ORCID iDs Jessica Sue Yi Wong http://orcid.org/0000-0002-3217-6495
James Hong-En Kang http://orcid.org/0000-0001-9635-7343

REFERENCES