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Letter to Editor Open Access

Inflammation and Platelet Activation after Corona Virus Disease 2019 Vaccines - Possible Mechanisms behind Vaccine-Induced Immune Thrombocytopenia and Thrombosis (VITT)

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Letter to Editor

The introduction of COVID-19 vaccines has provided the best opportunity of containing the global COVID-19 pandemic. Vaccine-induced Immune Thrombocytopenia and Thrombosis (VITT) has been related to the adenovirus-vector-based Oxford/AstraZeneca [ChAdOx1] (AZ) and Johnson & Johnson [Ad26.CoV2.S] COVID-19 vaccines. The pathophysiology of COVID-19 VITT is still a mystery, particularly the processes that promote platelet activation, platelet factor (PF)4 release, complex formation, and PF4 antibody generation. This is a prospective study that looks at how different COVID-19 vaccines affect inflammation (CRP, TNF-α, IL-1β, IL-6, IL-8, IL-10), vascular endothelial activation (syndecan-1, thrombomodulin, E-selectin, ICAM-1, ICAM-3, VCAM-1), platelet activation (P-selectin, TGF-β, sCD40L) and aggregation (Multiplate® impedance aggregometry), whole blood coagulation (ROTEM®), thrombin generation and PF4 antibodies to reveal potential differences between AZ and mRNA vaccines in individuals without VITT [1]. The participants in the study were 80 vaccinated people (55 AZ and 55 mRNA) and 55 non-vaccinated healthy controls who were age and gender matched. The main findings were that both vaccines increased inflammation and platelet activation, though AZ vaccination caused a more pronounced increase in several inflammatory and platelet activation markers than mRNA vaccination, and that post-vaccination thrombin generation was higher after AZ vaccination than after mRNA vaccination. There was no difference between the vaccine groups in terms of PF4 antibody levels or the proportion of people with positive PF4 antibodies. This is the first study to show that AZ immunisation results in increased inflammation, platelet activation, and thrombin production when compared to mRNA vaccination in a head-to-head comparison. Specific components of the AZ adenovirus vector may act as initial triggers of (hyper) inflammation, platelet activation, and thrombin generation, potentially lowering the threshold for a cascade of events that both trigger complications related to excessive inflammation, platelet activation, and coagulation activation as seen in epidemiological studies and promote VITT development when combined with high-titer functionally active PF4 antibodies [2].

Three vaccines against severe acute respiratory syndrome coronavirus 2 and coronavirus disease 2019 have been approved or given emergency use authorisation by the US Food and Drug Administration to date. The Pfizer-Bio NTech BNT162b2 messenger RNA coronavirus disease 2019 vaccine and the Moderna mRNA-1273 messenger RNA coronavirus disease 2019 vaccine have shown great efficacy and few side effects in clinical trials and real-world observational studies, respectively [3].

A 20-year-old male college student in good health developed tinnitus and hematuria shortly after vaccination, quickly progressing to a syndrome that included systemic inflammation, acute kidney injury requiring hemodialysis, acute, bilateral, complete sensorineural hearing loss, radiographic evidence of acute multifocal ischemic strokes, pericardial effusion complicated by tamponade physiology requiring pericardial evacuation, pleural effusions requiring evacuation, A comprehensive clinical and research study that included cytokine analysis, whole blood cytometry by time of flight, and whole exome sequencing came up empty [4].

While the overall wellbeing profile of the BNT162b2 Covid-19 vaccine stays incredible for everyone, uncommon genuine occasions have been accounted for. In this report, we depict an instance of multisystem aggravation and organ brokenness of obscure component starting not long after organization of the first shot of BNT162b2 Covid-19 vaccine in a formerly healthy beneficiary [5].

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