Developmental strategy of SARS-CoV-2 vaccine and the mechanism of Vaccine-induced immune thrombotic thrombocytopenia (VITT)

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Vaccine-induced thrombotic thrombocytopenia (VITT), a condition characterized by simultaneous acute thrombosis and thrombocytopenia, is a sever side effect in several COVID-19 vaccines. VITT is caused by antibodies that recognize platelet factor 4 (PF4, also called CXCL4) bound to platelets, resulted in platelet activation and systemic intravascular coagulopathy. However, the mechanisms for induction of anti-PF4 Ab-dependent and -independent pathways are still unclear.

More and more evidences demonstrated the activated platelets and platelet-derived extracellular vesicles (EVs) contribute to aberrant induction of neutrophil extracellular traps (NETs), which further interact with activate platelets to form immunothrombosis and thrombocytopenia. Recent study suggests that spike protein can activate platelets via C-type lectins. As dengue virus has been shown to activate platelet C-type lectin 2 (CLEC2) to release EVs, thereby activate neutrophils to induce NET formation. Furthermore, C-type lectins have been shown to contribute significantly in COVID-19 and virus-induced NETosis. Thus, it would be very important to understand whether the spike protein in COVID-19 vaccines can directly activate platelets to induce NET formation, and design novel COVID-19 vaccine to prevent activation of platelets in the future.