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Original Article

# Application of the 4Ts Score in Patients with High-Risk of Vaccine-Induced Immune Thrombotic Thrombocytopenia: A Single Center Experience

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SUMMARY

*Background:* Coronavirus disease 2019 (COVID-19) vaccines may cause rare hematological complications such as vaccine-induced immune thrombotic thrombocytopenia (VITT). In this study, we aim to present our experience and share insights into the diagnostic work-up for patients with a high probability of VITT and assess the utility of the 4Ts score in VITT diagnosis.
*Patients and methods:* The study investigated VITT risks in patients from July to December 2021. Symptomatic individuals post-COVID-19 vaccination were enrolled and underwent anti-PF4 antibody testing, platelet counts, D-dimer tests, and imaging studies. Positive cases underwent heparin-induced platelet aggregation tests for VITT confirmation. The 4Ts scoring system, adapted for VITT, incorporated anti-PF4 antibody titers, aiding in assessing probability based on specific titer ranges.
*Results:* Between July and December 2021, 18 patients with suspected VITT were studied. The median age was 55, with 66.7% men. Symptoms emerged within a median of 13 days post-vaccination. Vaccine brands varied, with 66.7% receiving ChAdOx1. Deep vein thrombosis occurred in 44.4%, and 11.1% had autoimmune diseases. The median anti-PF4 level was 65.3 ng/mL. Treatment modalities included anticoagulants (50%), steroids (66.7%), and intravenous immunoglobulin (16.7%). One confirmed VITT case presented with ischemic bowel disease, a platelet count of 11000/μL, a 4Ts score of 7, and received plasma exchange. Retrospective application of the modified 4Ts scores classified patients into low, intermediate, and high probability groups.
*Conclusion:* The study highlights VITT's clinical features and diagnostic challenges, advocating for 4Ts score use in identifying high-risk cases. Larger studies are warranted to validate these findings.



## 1. Introduction

The coronavirus disease 2019 (COVID-19) pandemic caused severe causalities worldwide. Vaccination against COVID-19 has played a pivotal role in reducing mortality and morbidity associated with the disease. Despite the overwhelming success of vaccines, there have been rare instances of hematological complications, notably vaccine-induced immune thrombotic thrombocytopenia (VITT).[1–3] VITT is an infrequent but critical condition that manifests as severe thrombosis and thrombocytopenia. It has been primarily associated with adenovirus vector COVID-19 vaccines, including the AstraZeneca/COVISHIELD and Janssen/Johnson & Johnson COVID-19 vaccines. The incidence of VITT is estimated to be between 3 to 15 cases per million doses of the implicated vaccines. The condition has been reported globally, with fewer than 50 cases from Asia, Africa, and Latin America combined.[4]

The pathophysiology of VITT is believed to involve the production of anti-platelet factor 4 (PF4) antibodies, which activate platelets and lead to thrombosis. This mechanism is similar to that observed in heparin-induced thrombocytopenia (HIT), although in VITT, the antibodies can bind directly to PF4 without heparin. The diagnosis of VITT usually requires the presentation of thrombocytopenia, clinical evidence of thrombosis, and elevated anti-platelet factor 4 (anti-PF4) antibody.[5–8] Heparin-induced platelet aggregation test was encouraged to use for confirmation of VITT.[3,9,10] Because the confirmation test results were usually unavailable during the early disease course, the best clinical approach to identifying high-risk patients with VITT is not well established. The 4Ts score, originally developed for HIT, is a clinical tool adapted to assess the risk of VITT when confirmatory tests are not immediately available (Table 1).[11,12] However, the reported experience is limited. In this study, we aim to present our experience with VITT, offering insights into the diagnostic process and evaluating the utility of the 4Ts score in diagnosing this condition. Our findings underscore the importance of continued vigilance and research to understand and manage VITT effectively.

## 2. Patients and methods

### 2.1. Study population

This study was approved by institution review board (IRB) of

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**Table 1**
The 4Ts scoring system.

| 4Ts category | 0 point | 1 point | 2 points |
|---|---|---|---|
| Thrombocytopenia | Platelet count fall < 30% or platelet nadir < 10 | Platelet count 30%–50% or platelet nadir 10–19 | Platelet count fall > 50% and platelet nadir ≥ 20 |
| Timing of platelet count fall | Platelet count ≤ 4 days without recent exposure | Consistent with days 5–10 fall, but not clear (eg, missing platelet counts); onset after day 10; or fall ≤ 1 day (prior heparin exposure 30–100 days ago) | Clear onset days 5–10 or platelet fall ≤ 1 day (prior heparin exposure within 30 days) |
| Thrombosis or other sequelae | None | Progressive or recurrent thrombosis; non-necrotizing (erythematous) skin lesions; suspected thrombosis (not proven) | New thrombosis (confirmed); skin necrosis; acute systemic reaction postintravenous unfractionated heparin bolus |
| Other causes of thrombocytopenia | Definite | Possible | None apparent |

Footnote: Adapted from reference 12. The 4Ts score comprises the cumulative values across four categories. Scores falling within the ranges of 0–3, 4–5, and 6–8 are indicative of low, intermediate, and high probabilities of heparin-induced thrombocytopenia, respectively.

MacKay Memorial Hospital (IRB number: 23MMHIS115e). We performed chart review and captured clinical information from patients with the risks of VITT from July 2021 to December 2021. The criteria of a patient who was at risk of VITT was defined by ISTH Interim Guidance for the Diagnosis and Treatment on Vaccine-induced Immune Thrombotic thrombocytopenia.[13] The guidance was also endorsed by the Taiwan Centers for Disease Control (CDC). Patients who (1) received COVID-19 vaccination 4–28 days prior to onset of symptoms, and (2) with signs and symptoms suggestive of thromboembolism, including severe, persistent headache with or without vision change, seizure-like activity; severe, persistent abdominal pain; leg swelling or pain; chest pain and/or shortness of breath, were considered at risk for VITT. Only those patients who had been tested for anti-PF4 antibody were enrolled into this study. All patients received further investigation including platelet count, D-dimer test, and imaging study for the evaluation of thrombosis. If anti-PF4 antibody was positive, heparin-induced platelet aggregation test was performed.[14] The modified diagnosis of VITT in this study was defined as below: the presence of venous thrombosis confirmed by imaging, thrombocytopenia, and positive anti-PF4 antibody regardless of the antibody level.[10] This modification ensured that patients with lower anti-PF4 antibody levels, yet meeting the other criteria, were included in our analysis, providing a more comprehensive understanding of VITT presentation and diagnosis.

### *2.2. Detection of anti-PF4 antibodies*

For testing of anti-PF4 antibodies, the serum sample was prepared with an enzyme-linked immunosorbent assay (ELISA) of Anti-Human Heparin Platelet Factor 4 (HPF-4) antibody kit. Absorbance was measured in an ELISA reader at 450 nm (Synergy HTX multimode reader, Biotek, VT, USA). Optical density value > 0.4 was suggestive to be positive.[8]

### *2.3. Anti-PF4 level-adapted 4Ts scoring system in patients with suspected VITT4Ts*

4Ts score system was initially implemented to patients with risk of HIT. The four components included thrombocytopenia, timing of platelet count fall, thrombosis or other sequelae, and other causes for thrombocytopenia. The system has the scores between 0 to 8 points. Scores of 0–3, 4–5, and 6–8 points were classified as low, intermediate, and high probability for HIT, respectively.[12] In addition to the original 4Ts scores, anti-PF4 antibody titer was also incorporated into our anti-PF4 level-adapted 4Ts scoring system. For patients with anti-PF4 antibody titer < 50 ng/mL, 50–100 ng/mL, and > 100 ng/mL, scores of 0, 1, and 2 points were given respectively.

## 3. Results

From July 2021 to December 2021, a total of 30 patients were suspected to have VITT after COVID-19 vaccination, based on their clinical presentation. Eighteen adult patients (60%) with available anti-PF4 antibody titer were enrolled onto this study (Table 2). Me-

**Table 2**
The characteristics of 18 patients with suspected vaccine-induced immune thrombotic thrombocytopenia (VITT).

| Characteristics | |
|---|---|
| Median age, years (range) | 55 (26–84) |
| Gender, n (%) | |
| Male | 12 (66.7) |
| Female | 6 (33.3) |
| Days from vaccination to symptoms, median (range), n (%) | 13 (2–51) |
| < 4 days | 5 (27.8) |
| 4–30 days | 5 (27.5) |
| > 30 days | 8 (44.4) |
| Vaccine brands, n (%) | |
| ChAdOx1 | 12 (66.7) |
| mRNA-1273 | 4 (22.2) |
| BNT162b2 | 2 (11.1) |
| Vaccine injection times, n (%) | |
| First | 9 (50) |
| Second | 9 (50) |
| Symptoms appeared after which dose, n (%) | |
| First | 9 (50) |
| Second | 9 (50) |
| Deep vein thrombosis, n (%) | |
| Yes | 8 (44.4) |
| No | 10 (55.6) |
| Intracranial hemorrhage, n (%) | |
| Yes | 2 (11.1) |
| No | 16 (88.9) |
| Autoimmune disease, n (%) | |
| Yes | 2 (11.1)* |
| No | 16 (88.9) |
| Neoplasms, n (%) | |
| Yes | 4 (22.2)[#] |
| No | 14 (77.8) |
| Anti-PF4 antibody titer (ng/mL), median (range), n (%) | 65.3 (10.5–400.9) |
| < 50 | 6 (33.3) |
| 50–100 | 7 (38.9) |
| > 100 | 5 (27.8) |
| Lowest fibrinogen level (mg/dL), median (range) | 231.5 (30–700) |
| D-dimer level (ng/mL), median (range) | 3108 (440–> 10000) |

Footnotes: PF4, platelet factor 4.
* One systemic lupus erythematosus and 1 ankylosing spondylitis. [#] Each of one breast cancer, cholangiocarcinoma, lung cancer, and uterine myoma. All of these diseases were preexisting comorbidities.

dian age was 55 years of age (26–84 year). Twelve patients (66.7%) were men and 6 patients (33.3%) were women. The median time from vaccination to symptom was 13 days (range, 2–51 days). The vaccination brands were ChAdOx1, mRNA-1273 and BNT162b2 in12 patients (66.7%), 4 patients (22.2%) and 2 patients (11.1%), respectively. Nine patients (50%) received only one dose and 9 patients (50%) received two doses. Nine patients (50%) developed the symptoms after the first dose and 9 patients (50%) after the second one. Deep vein thrombosis and intracranial hemorrhage developed in 8 (44.4%) and 2 patients (11.1%), respectively. Two (11.1%) and 4 (22.2%) patients had history of autoimmune diseases and malignancy, respectively. Median anti-PF4 level was 65.3 ng/mL (10.53–400.93). The range of the titer of anti-PF4 antibody were < 50 ng/mL, 50–100 ng/mL and > 100 ng/mL in 6 patients (33.3%), 7 patients (38.9%) and 5 patients (27.8%), respectively. The 4Ts score and their corresponding probabilities of VITT in 6 patients with low anti-PF4 Ab titer (< 50 ng/mL) are shown in Table 3. The 4Ts score is used to help with the clinical diagnosis of VITT. Two of the 6 patients had intermediate or high probabilities of VITT. Although the remaining 4 patients had low probabilities of VITT, the diagnosis of VITT was based on our clinical judgment. It is noteworthy that one cannot completely exclude the diagnosis of VITT solely based on a low 4Ts score.

We analyzed the treatment modalities for patients with suspected VITT. Nine patients (50%) received anticoagulants (low-molecular-weight heparin or oral anticoagulant). Twelve patients (66.7%) received steroid. Intravenous immunoglobulin (IVIg) was administrated in 3 patients (16.7%). Plasma exchange was performed in three patients (16.7%). One female patient was confirmed to have VITT. The patient presented with ischemic bowel disease. The platelet count was 11000 cells/μL. The D-dimer was > 10000 ng/mL. The anti-PF4 antibody was 136.23 ng/mL. The optic density was 1.044. The heparin-induced platelet aggregation test was positive. Her 4Ts score was 7, indicating a "high probability" of VITT.

We have retrospectively applied the modified 4Ts score to these patients (Table 4). Nine patients (50%) were classified as "low probability" (0–3 points). Six patients (33%) were classified as "intermediate probability" (4–5 points). Three patients (17%) were classified as "high probability" (6–8 points). The score of the patient who was confirmed to be VITT was 7.

**Table 3**
The 4Ts score and their corresponding probabilities of VITT in 6 patients with low anti-PF4 antibody titer (< 50 ng/mL).

| 4Ts score | 4Ts score group | 4Ts score plus PF4 score | 4Ts plus PF4 score group | Anti-PF4 antibody titer (ng/mL) |
|---|---|---|---|---|
| 0 | Low | 0 | Low | 30.65 |
| 2 | Low | 2 | Low | 36.31 |
| 2 | Low | 2 | Low | 49.05 |
| 3 | Low | 3 | Low | 46.31 |
| 4 | Intermediate | 4 | Intermediate | 10.53 |
| 6 | High | 6 | High | 35.09 |

Footnotes: High, high probability; Intermediate, Intermediate probability; Low, low probability; PF4, anti-platelet factor 4.

## 4. Discussion

In this retrospective, single-armed observational study, we aimed to investigate the diagnostic criteria and timelines associated with VITT within our cohort. Our findings provide valuable insights into the challenges of diagnosing VITT and the implications for patient management. One notable observation from our study is the infrequent occurrence of VITT, even among high-risk populations. The sole confirmed VITT case in our cohort belonged to the "high probability" category based on the 4Ts score, underscoring the precision of this diagnostic tool. However, it is essential to acknowledge that our findings may not be generalizable to all populations, and variations in VITT prevalence across different demographic groups and regions warrant further investigation.

A critical aspect of our study is the identification of diagnostic delays in VITT recognition, with a median time to diagnosis of approximately 30 days. This delay may have significant implications for patient outcomes, highlighting the importance of expediting the diagnostic process. The prolonged turnaround time for anti-PF4 antibody testing, particularly when conducted at external facilities, emerged as a key contributor to these delays. Moreover, the complexity of the diagnostic pathway, including multiple steps such as clinical suspicion, testing, and result interpretation, further compounds the challenges of timely VITT diagnosis.

In comparison to other studies, our findings regarding the diagnostic utility of the 4Ts score and anti-PF4 antibody testing may vary. Differences in patient populations, healthcare settings, and testing methodologies could contribute to discrepancies in diagnostic outcomes. For example, studies conducted in regions with higher VITT prevalence or different vaccination protocols may report higher rates of VITT diagnosis or different patterns of diagnostic testing utilization. These variations underscore the importance of contextualizing study findings within the broader literature and considering the influence of local factors on diagnostic practices.

It is crucial to acknowledge the biases inherent in our retrospective, single-armed observational study design. Firstly, the retrospective nature introduces the potential for selection bias, as patient data were collected based on predefined criteria from medical records. This may lead to an overrepresentation of certain patient demographics or clinical presentations, affecting the generalizability of our findings. Additionally, the lack of a control group limits our ability to compare outcomes or assess causality. Moreover, reliance on medical records for data extraction may introduce information bias, as data completeness and accuracy are subject to documentation practices and clinician interpretation.

In our cohort, suspicion of VITT persisted in some patients despite normal platelet counts. In most of these patients, the final results of their work-up did not support the diagnosis of VITT. This

**Table 4**
Application of VITT-adapted 4Ts scoring system in patients with suspected VITT.

| Points | 4Ts scoring system, n (%) | Anti-PF4 antibody titer for 4Ts scoring system (ng/mL), median (range) | Death in 4Ts group, n (%) | 4Ts plus anti-PF4 antibody scoring system, n (%) | Anti-PF4 antibody titer for 4Ts plus anti-PF4 antibody scoring system (ng/mL), median (range) | Death in 4Ts plus anti-PF4 antibody group, n (%) |
|---|---|---|---|---|---|---|
| 0–3 | 9 (50) | 52.6 (30.65–400.93) | 2 (22.2)* | 8 (44.4) | 50.8 (30.65–400.93) | 2 (25)* |
| 4–5 | 6 (33) | 77.8 (10.53–148.54) | 1 (16.7)[#] | 2 (11.1) | 98.8 (10.53–187.1) | 0 (0) |
| 6–8 | 3 (17) | 54.5 (35.09–136.23) | 1 (33.3)[@] | 8 (44.4) | 77.8 (35.09–148.54) | 2 (25)[#@] |

0–3 points: low probability. 4–5 points: Intermediate probability. 6–8 points: high probability.
Footnotes: n, number; PF4, platelet factor 4. * One sepsis and 1 pneumonia; [#] One spontaneous subarachnoid hemorrhage and intraventricular hemorrhage; [@] One ischemic bowel disease, liver infarction, and multiorgan failure.

suggests a lack of familiarity among primary care physicians with VITT diagnosis, potentially leading to overuse of anti-PF4 antibody testing in thromboembolic presentations during the pandemic. In our patients, treatment approaches varied, reflecting uncertainty in optimal management for suspected VITT. Anticoagulants and steroids were commonly administered, with IVIg reserved for high-risk patients, aligning with recent recommendations.[15–17]

Consistent with prior research findings, the 4Ts score emerged as a valuable tool for early VITT diagnosis and management. After the COVID-19 vaccination programs started worldwide, several reports regarding to VITT have been published.[1–3] One report incorporated symptoms, D-dimer level and anti-PF4 antibody level to define the types of VITT.[3] While global reports on VITT increased post-COVID-19 vaccination, accessibility to anti-PF4 antibody testing remains inconsistent worldwide. A study in India utilized 4Ts score to evaluate the probability of VITT.[11] They claimed that the score could help clinicians to initiate proper managements. Our findings underscore the utility of the 4Ts score in identifying high-risk patients, emphasizing that anti-PF4 antibody testing, followed by heparin-induced platelet aggregation, aids in confirming VITT diagnoses.

Our study has certain limitations inherent in its retrospective design. Firstly, the limited number of cases precludes extensive statistical analyses. Secondly, not all patients underwent the heparin-induced platelet aggregation test, introducing variability in diagnostic confirmation. Thirdly, the diverse array of treatments employed hinders direct outcome comparisons. Despite these constraints, our study provides valuable insights into the real-world diagnostic challenges of VITT within an Asian medical center. Introducing a novel disease entity, such as VITT, poses significant challenges for physicians. The varied diagnostic approaches employed in patients suspected of having VITT underscore the complexity of navigating through unfamiliar medical territory. The incorporation of a scoring system, such as the 4Ts score, proves beneficial in refining both the diagnosis and treatment of VITT. Despite these challenges and limitations, our study contributes to the growing understanding of VITT and lays the groundwork for further research in this evolving medical landscape.

## 5. Conclusion

In conclusion, we have demonstrated the clinical characteristics and diagnosis of VITT in an Asian medical center. VITT is still rare and the diagnosis remains to be a medical challenge. The use of the 4Ts score for the diagnosis of VITT may help identify high-risk patients, and prompt management of these patients is advised. Larger studies are warranted to validate these findings and enhance our understanding of VITT in diverse populations.

## Conflicts of interest

We declare that there are no conflicts of interest relevant to this study.

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