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Original Article

Incidence and mortality of heparin-induced thrombocytopenia in critically Ill patients

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Abstract: Objectives: Heparin is widely used as an anticoagulant in hospitals. Ventilated patients on heparin are at a higher risk of developing heparin-induced thrombocytopenia (HIT). This study aims to examine the effect of HIT on mortality in ventilated patients. Methods: We used available ICD-10 codes from 2016 to 2020. Patients with specific ICD-10 codes for HIT and ventilator dependence were included. The incidence of HIT in ventilated patients was calculated, and mortality rates were compared between HIT-positive and HIT-negative ventilated patients. For the year 2020, given the presence of COVID, additional multivariate analysis was performed to adjust for baseline characteristics and confounding factors. Results: We found no significant increase in mortality in patients on ventilation with HIT compared to those without HIT for the years 2016-2019 in the pre-COVID era. However, there was a significant increase in mortality among ventilated patients with HIT in the year 2020 during the first year of the COVID pandemic before vaccination (odds ratio [OR] = 2.03, 95% confidence interval [CI] = 1.26-3.26, P = 0.004). However, after adjusting for age, gender, race, and COVID-19 diagnosis, only COVID-19, but not HIT, showed a significantly higher risk of mortality in ventilated patients with HIT compared to no HIT (OR = 3.80, 95% CI = 3.47-4.15, P < 0.001). Conclusions: These findings suggest HIT is not associated with increased mortality among ventilated patients. However, in the year 2020, COVID-19 infection increased the mortality of ventilated patients by almost 4-fold regardless of HIT. This data suggests that HIT is not an independent risk for mortality in ventilated patients.

Keywords: COVID-19, HIT, heparin induced thrombocytopenia, thrombocytopenia, ventilation, mechanical ventilation, ventilatory support, heparin, anticoagulation

Introduction

Low-molecular weight heparin (LMWH) and unfractionated (UF) heparin are common anticoagulants used in hospitals. However, a feared complication of heparin use is heparin-induced thrombocytopenia (HIT), caused by immune antibodies created against a protein complex of heparin and a platelet protein, platelet factor 4 (PF4) [1]. HIT is a rare complication of anticoagulation, with recent large population studies estimating an incidence of 0.065%, or one in 1500 hospital admissions [2]. HIT is more likely to occur with UF compared to LMW in postoperative patients [3] and in the setting of thromboprophylaxis [4] not limited by language, and from reference lists of key articles were evaluated. Randomized and nonrandomized controlled trials comparing prophylaxis with UFH and LMWH and measuring HIT or thrombocytopenia as outcomes were included. Two reviewers independently extracted data on thromboprophylaxis (type, dose, frequency, and duration. HIT causes a paradoxical hypercoagulable state, leading to increased risk of thrombosis [5] only a subgroup of patients with HIT develops thromboembolic complications. We aimed to identify risk factors for developing HITassociated thrombosis. We analyzed a registry of patients with clinical suspicion of HIT who tested positive using a sensitive functional assay. Patient information was obtained by a standardized questionnaire. By multivariate analysis the association of age, gender, type of patient population, and magnitude of the platelet count decline with the frequency, type (venous or arterial, amputation and death [6].

HIT is associated with longer durations of heparin exposure [7, 8], and duration, rather than

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dose, appears to confer the highest risk of HIT [9], likely due to an increased period allowing for the immune system to develop antibodies against the heparin-platelet factor 4 complex. Usage of heparin has also been increasing over time [10]. Ventilator-dependent patients often have longer exposures to heparin, increasing the theoretical risk of HIT. Additionally, sicker critically ill patients receive a lower proportion of LMW heparin compared to UF [11], also increasing the theoretical risk. Rates of HIT in critical care patients [12] appear higher than in general hospital admissions as above. Though there is no direct mechanistic link between critically ill or mechanically ventilated patients and HIT, the combination of longer heparin durations and higher use of UFH may confer increase risk and thus result in the higher rates seen in critically ill patients. Diagnosing HIT in the ventilated and critically ill patient is challenging due to the fact that most patients have an alternative reason for thrombocytopenia [13]. Several studies have looked at rates and outcomes of HIT in these critically ill patients [12, 14-16], but suffer from small sample sizes due to the inherent rarity of HIT. Other studies have used large population-based data sets to look at incidence of HIT in the general population [2, 17-19], cardiac surgery patients [20, 21], and orthopedic patients [22], but none have looked specifically at ventilated patients. The goal of this study was to examine the effect of HIT on mortality in adult ventilated patients using a large inpatient database.

Methods

Case selection

This was a retrospective study using data gathered from the National Inpatient Sample (NIS), using the period from 2016 to 2020. International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM) code Z99.11 was used to identify patients who were at least 18 years old and had dependence on respirator [ventilator] status. We then used ICD-10-CM code D75.82 to identify patients with Heparin-induced thrombocytopenia. Additional codes D75.821 (Non-immune heparin-induced thrombocytopenia), D75.822 (Immune-mediated heparin-induced thrombocytopenia), D75.828 (other heparin-induced thrombocytopenia syndrome) and D75.829 (Heparin-induced thrombocytopenia, unspecified) were also used to identify patients with HIT. Inclusion criteria were any patients > 18 years old, who had respiratory/ventilator status with one of the above codes as a primary or secondary diagnosis code. Patients with missing demographic, hospitalization, or outcome data were excluded. The outcomes of this study analyzed were incidence per year of HIT and mortality for HIT and non-HIT patients for each year.

Observational indicators

The primary observational indicators evaluated in this study were annual incidence of HIT among ventilated patients and in-hospital mortality for HIT and non-HIT ventilated cohorts. Incidence was defined as the proportion of all hospitalized adults with ventilator dependence who also carried any of the HIT-related diagnostic codes. Mortality was defined as death occurring during the index hospitalization, as recorded in the NIS discharge disposition field. Additional observational indicators included demographic characteristics (age, sex, race, primary payer) and hospital-level features (bed size, teaching status, and geographic region). All variables were used exactly as reported in the NIS dataset and were analyzed according to HCUP methodological standards.

Statistical methods

First, incidence of HIT in ventilated patients was calculated for each year. Demographic data for ventilated patients with HIT was also calculated for each year (age, gender, race, primary payer, hospital bed size, hospital teaching/location, hospital region). Mortality of HIT ventilated patients and non-HIT ventilated patients was then calculated for each year. A Student's independent samples t-test ($P < 0.05$ for statistical significance) was used to compare mortality between HIT and non-HIT ventilated patients. For year 2020, due to the emergence of COVID-19, additional multivariate analysis was conducted to adjust the apparent increase in mortality of ventilated patients with HIT for age, gender, race, and COVID-19. Univariate analysis was then conducted on ventilated patients with HIT and COVID-19 and compared to ventilated patients with HIT without COVID-19. Finally, additional multivariate analysis was conducted to adjust for hypertension, obesity, age, gender, and race.

Table 1. Demographic information of ventilated patients, 2016-2020

	2016	2017	2018	2019	2020	All Years
Ventilatory Dependent	85725	92485	94725	96580	104425	473940
HIT	345	395	380	220	375	1715
Age Mean (SD)	62.39 (17.84)	60.80 (17.83)	61.47 (14.48)	62.20 (16.10)	60.05 (16.33)	61.29 (16.52)
Gender						
Male	55.1%	63.3%	55.3%	54.5%	62.7%	58.6%
Female	44.9%	36.7%	44.7%	45.5%	37.3%	41.4%
Race						
White	60.6%	69.2%	60.3%	65.9%	48.6%	60.7%
Black	18.2%	20.5%	20.5%	18.2%	15.3%	18.6%
Hispanic	15.2%	6.4%	5.5%	11.4%	25.0%	12.6%
Asian/Pac Isl	4.5%	1.3%	6.8%	4.5%	4.2%	4.2%
Native American	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	1.5%	2.6%	6.9%	0.0%	6.9%	3.9%
Primary Payer						
Medicare	65.2%	52.5%	48.7%	56.8%	41.9%	52.5%
Medicaid	13.0%	17.9%	19.7%	9.1%	20.3%	16.7%
Private including HMO	17.4%	24.4%	21.1%	29.5%	29.7%	24.0%
Self-Pay	1.5%	2.6%	5.3%	2.3%	4.1%	3.2%
No Charge	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	2.9%	3.5%	5.2%	2.3%	4.0%	3.6%
Hospital Bed size						
Small	14.5%	13.9%	9.3%	22.7%	17.3%	14.9%
Medium	29.0%	26.6%	28.9%	15.9%	34.7%	28.0%
Large	56.5%	59.5%	61.8%	61.4%	48.0%	57.1%
Hospital Teaching/Location						
Rural	0.0%	1.3%	1.3%	0.0%	1.3%	0.9%
Urban non-teaching	27.5%	22.8%	13.2%	15.9%	16.0%	19.2%
Urban Teaching	72.5%	75.9%	85.5%	84.1%	82.7%	79.9%
Hospital Region						
Northeast	15.9%	24.1%	17.1%	15.9%	12.0%	17.2%
Midwest	31.9%	25.3%	23.7%	22.7%	10.7%	22.7%
South	37.7%	31.6%	42.1%	50.0%	48.0%	41.1%
West	14.5%	19.0%	17.1%	11.4%	29.3%	19.0%

Gathered from national inpatient sample using ICD-10-CM codes.

Results

Incidence across years

From 2016 to 2020, 473,940 patients were identified as being ventilatory dependent. Of these, 1,715 were identified with HIT (0.36% overall incidence). Data by year and additional demographic data are provided in **Table 1**. The incidence of HIT in ventilated patients was infrequent, but similar across years (**Figure 1**).

Mortality differences between years

In terms of mortality rates, year 2016 HIT mortality of ventilated patients was 20.29% compared to 19.77% for others (OR 1.03, 95% CI 0.58-1.84, P = 0.91), year 2017 was 21.52% compared to 19.48% (OR 1.13, 95% CI 0.63-2.02, P = 0.67), 2018 was 25.00% compared to 18.99% (OR 1.42, 95% CI 0.84-2.42, P = 0.19), 2019 was 22.73% compared to 18.71% (OR 1.28, 95% CI 0.63-2.61). However, year 2020 showed a significant mortality difference between groups, 41.33% compared to 25.78% (OR 2.03, 95% CI 1.26-3.26, P = 0.004) (**Figure 2**).

2020 COVID multivariate analysis

For year 2020, additional multivariate analysis of ventilated patients with mortality showed

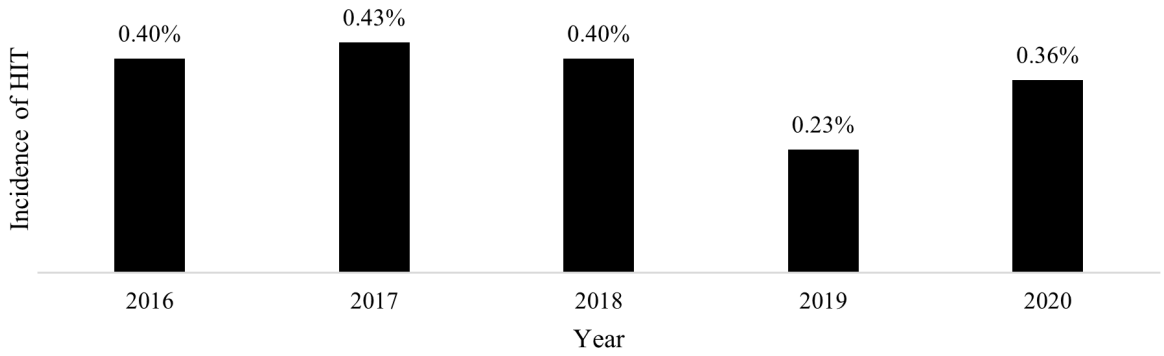

Figure 1. Incidence of HIT in ventilated patients, 2016-2020.

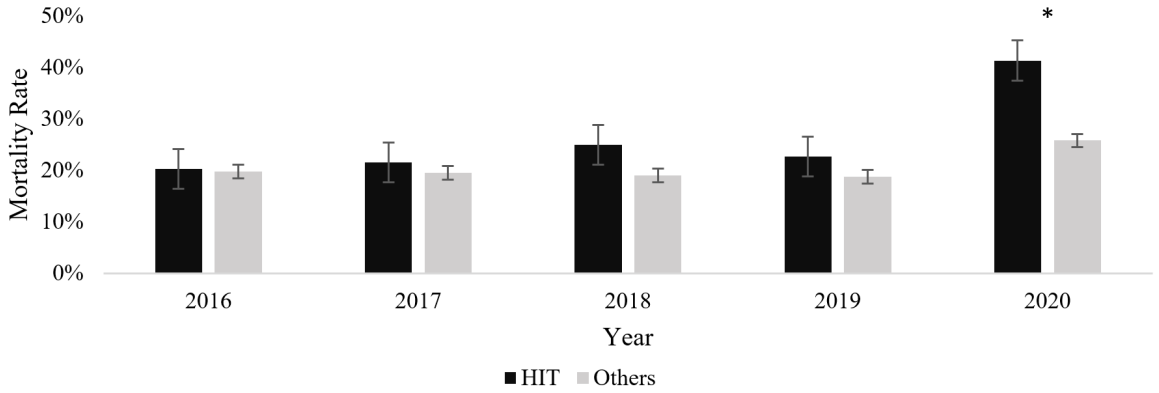

Figure 2. Mortality rate of ventilated patients with HIT compared to ventilated patients without HIT, 2016-2020.

age, sex (female), and COVID-19 diagnosis to be additional statistically significant risk factors (**Table 2**). Univariate analysis for year 2020 of ventilated patients with HIT, comparing COVID-19 mortality to non-COVID-19 mortality showed a mortality rate of 54.84% versus 31.82% (OR 2.60, 95% CI 0.98-6.90, P = 0.05). Finally multivariate analysis of ventilated patients with HIT yielded results in **Table 3**.

Discussion

Overview of main findings

In this large retrospective analysis of ventilated patients from 2016 to 2020, we found that heparin-induced thrombocytopenia (HIT) was an uncommon complication, occurring in 0.36% of patients overall. Incidence remained relatively stable over the study period. Mortality among ventilated patients with HIT did not differ significantly from those without HIT in the pre-COVID years (2016-2019). However, in 2020, coinciding with the COVID-19 pandemic, mortality was higher in ventilated patients with HIT (41.3%) compared to those without HIT (25.8%), which was statistically significant on unadjusted analysis. After adjusting for age, sex, race, and COVID-19 diagnosis, HIT itself was not independently associated with increased mortality. Instead, COVID-19 infection emerged as a strong predictor of death (OR = 3.80, 95% CI = 3.47-4.15, P < 0.001), along with older age and female gender. Overall, these findings suggest that while HIT remains a rare complication in ventilated patients, it does not independently increase mortality, and outcomes are strongly influenced by concurrent COVID-19 infection.

Comparison of incidence of HIT with prior literature

The overall incidence of HIT in ventilated patients in our study (0.36%) aligns with prior literature reporting low rates of this complication across different patient populations. Earlier studies have demonstrated variable incidence depending on patient population and type of heparin exposure. A 2007 study of HIT in a

Table 2. Multivariate analysis for year 2020 in ventilated patients who have died

	p-value	OR (95% C.I)
HIT	0.42	1.24 (0.74-2.07)
Age	<0.001	1.03 (1.02-1.03)
Gender (Female)	<0.001	0.85 (0.79-0.91)
Race		
White	REF	
Black	0.98	1 (0.90-1.10)
Hispanic	0.05	1.12 (1-1.26)
Asian/Pac Isl	0.42	1.07 (0.90-1.28)
Native American	0.48	0.87 (0.58-1.29)
Other	0.38	1.09 (0.90-1.32)
COVID-19	<0.001	3.80 (3.47-4.15)

Table 3. Multivariate analysis of ventilated patients with HIT, year 2020

	p-value	OR (95% C.I)
COVID-19	0.232	2.13 (0.61-7.47)
Hypertension	0.874	0.91 (0.29-2.89)
Obesity	0.431	1.96 (0.36-10.71)
Age	0.011	1.05 (1.01-1.09)
Gender (Female)	0.285	0.51 (0.15-1.77)
Race		
White	REF	
Black	0.447	2.07 (0.31-13.75)
Hispanic	0.133	3.08 (0.70-13.50)
Asian	0.838	0.77 (0.06-9.85)
Other	0.458	2.69 (0.19-37.84)

teaching institution reported a 0.2% incidence of HIT, with an increased incidence of 0.76% among patients receiving therapeutic-dose IV heparin; notably, approximately half of all new HIT cases occurred in cardiovascular surgery patients [23]. Similarly, a 2003 study of 598 medical ward patients receiving subcutaneous unfractioned heparin found an incidence of 0.8% [24].

In surgical and specialty populations, incidence rates varied more widely. A recent 2023 analysis of distinct patient subgroups found an incidence of 0.16 per 1000 patient-years in general surgical patients, 0.52 in cardiac, vascular, and interventional cardiology patients, 0.75 in orthopedic surgery patients, 0.15 in medical patients, and 1.0 in nephrology/hemodialysis patients, highlighting higher risk in patients undergoing procedures with significant heparin exposure [25]. A 2005 prospective study of general medical patients only receiving low-molecular-weight heparin and found an incidence of 0.8%, comparable to that seen in studies of unfractionated heparin [26].

Large administrative database analyses demonstrate even lower incidence in broader populations. An analysis of the 2020 NIS found an overall incidence of 0.00035% among all hospitalized patients, reflecting the rarity of HIT outside high-risk populations [27]. Focused NIS studies in surgical cohorts similarly reported low but variable rates: a 2017 study of NIS data from 2010-2012 found a 0.31% incidence among vascular surgery patients [28], while a 2018 study of NIS data from 2009-2013 reported 0.63% in cardiopulmonary bypass patients, and notably lower rates in orthopedic procedures (0.04% in hip arthroplasty, 0.02% in knee arthroplasty) [2].

Overall, these findings contextualize our observed incidence of 0.36% in ventilated patients, suggesting that HIT remains a rare but clinically relevant complication, with risk influenced by the type of heparin, the intensity of exposure, and the underlying patient population.

Comparison of mortality of HIT with prior literature

The mortality associated with HIT varies widely across different patient populations, reflecting both underlying comorbidities and the clinical context in which HIT develops. In our cohort of ventilated patients, the observed in-hospital mortality ranged from 20.29% to 25.00% (excluding year 2020), which is lower than the rates reported in certain high-risk populations but comparable to broader administrative data.

In postcardiac surgery patients, HIT has been associated with particularly high mortality. A 2024 retrospective study of 41 patients found a mortality rate of 53.3% among postcardiac surgery patients who developed HIT, underscoring the severity of HIT in this population [29]. Similarly, an analysis of NIS data from 2009-2010 and reported an 11.1% mortality rate among HIT patients undergoing cardiac surgery, while noting a low overall incidence of HIT at 0.3% [21].

Outside the cardiac surgery setting, mortality appears lower but remains clinically relevant. An analysis of the NIS from 2016-2019 in patients with end-stage renal disease (ESRD) who developed HIT and found an 11% mortality rate [30]. These findings suggest that while the absolute mortality associated with HIT is variable, patients with critical illness or complex surgical and comorbid profiles are at higher risk.

COVID interaction with HIT

In our study, ventilated patients with HIT in 2020, the year of the COVID-19 pandemic, demonstrated higher unadjusted mortality compared to non-HIT ventilated patients. However, after adjusting for COVID-19 diagnosis in regression analysis, this difference was no longer statistically significant, suggesting that COVID-19 may have been a key driver of the observed increased mortality.

These findings align with prior literature showing that critically ill COVID-19 patients are at elevated risk for thrombocytopenia and HIT-like syndromes. Pooled analyses indicate that HIT incidence is higher in critically ill COVID-19 patients, particularly those receiving therapeutic-dose heparin, and mortality in this population is substantial [31, 32] there is ongoing concern regarding a heightened incidence of heparin-induced thrombocytopenia (HIT. Severe thrombocytopenia and the presence of anti-PF4 antibodies have been observed even in heparin-naïve patients, suggesting spontaneous HIT can occur in the setting of COVID-19 [33, 34].

Our results support these observations, indicating that the increased mortality seen in HIT ventilated patients during 2020 may be largely attributable to the impact of COVID-19 rather than HIT itself. This emphasizes the importance of accounting for COVID-19 infection when evaluating outcomes in critically ill patients with HIT and highlights the complex interplay between critical illness, viral infection, and HIT risk.

Clinical implications

Our findings suggest that HIT remains a rare but clinically important complication in ventilated patients. While incidence in our cohort aligns with prior studies showing low overall rates of HIT, mortality remains high among affected patients, particularly in those with comorbid conditions such as COVID-19.

The elevated mortality observed in ventilated HIT patients during 2020 highlights the need for heightened vigilance in critically ill patients with COVID-19, as the virus may exacerbate platelet activation and increase HIT risk. Clinicians should consider early recognition and diagnosis of HIT, particularly in patients receiving therapeutic heparin or showing unexplained thrombocytopenia, and should weigh the potential benefits of alternative anticoagulants when appropriate.

Additionally, our results underscore the importance of integrating COVID-19 status into risk stratification for HIT-related mortality. Adjusting for COVID-19 in regression analyses neutralized the mortality difference between HIT and non-HIT ventilated patients, suggesting that management strategies must consider both viral infection and HIT in critically ill populations.

Overall, these findings reinforce the need for careful monitoring of platelet counts, timely laboratory testing for HIT, and proactive consideration of non-heparin anticoagulation in high-risk ventilated patients, particularly during periods of viral outbreaks or in patients with severe inflammatory states.

Strengths of study

This study leverages a large, nationally representative database of ventilated patients, allowing for robust estimates of HIT incidence and outcomes in this high-risk population. Our use of ICD coding to identify HIT diagnoses is supported by prior validation work, which demonstrated a sensitivity of 90.9% and specificity of 94.4% for these codes, suggesting that the results of our study are likely reliable [2]. Additionally, the study spans multiple years, including the COVID-19 pandemic, which allows for assessment of potential interactions between viral illness and HIT. Finally, the inclusion of regression analyses adjusting for confounders, such as COVID-19 status, strengthens the causal inference regarding HIT and mortality.

Limitations

Despite its strengths, this study has several limitations. First, it is retrospective and relies

on administrative coding, which may be subject to misclassification or underreporting of HIT cases. While prior validation supports the accuracy of the codes used, laboratory confirmation of HIT was not available. Second, detailed clinical data such as platelet counts, heparin exposure, timing of HIT onset, or anticoagulation management are not captured in the database, limiting our ability to assess mechanisms or individual treatment effects. Third, residual confounding from unmeasured variables may persist, particularly in critically ill populations with multiple comorbidities. Finally, while we were able to adjust for COVID-19 status, the database does not capture the severity of infection or treatments received, which may influence both HIT incidence and outcomes.

Conclusions

In summary, HIT in ventilated patients is rare but associated with substantial mortality. Our findings suggest that the elevated mortality observed during the COVID-19 pandemic may be largely attributable to the infection itself rather than HIT alone. Clinicians should maintain vigilance for HIT in critically ill patients, particularly during periods of heightened inflammatory or infectious risk, and consider alternative anticoagulation strategies when appropriate. Large, prospective studies with laboratory-confirmed HIT and detailed clinical data are needed to further delineate the relationship between HIT, COVID-19, and outcomes in ventilated patients.

Disclosure of conflict of interest

None.

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