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Improving Pharmacologic Prevention of VTE in Trauma: IMPACT-IT QI Project

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Abstract

Enoxaparin regimens commonly used for prophylaxis fail to achieve optimal anti-factor Xa levels in up to 70 per cent of trauma patients. Accordingly, trauma services at the study institution endeavored to develop a standardized approach to optimize pharmacologic prevention with enoxaparin. An enoxaparin venous thromboembolism (VTE) prophylaxis protocol implemented in October 2015 provided weight-adjusted initial dosing parameters with subsequent dose titration to achieve targeted anti-factor Xa levels. Symptomatic VTE rate was evaluated 12 months pre- and post-implementation. Data were obtained from the trauma registry and charts were reviewed from electronic medical records. The rate of symptomatic VTE significantly declined post-implementation (2.0% *vs* 0.9%, $P = 0.009$). Enoxaparin use was comparable in these two phases validating that the decline in symptomatic VTEs was not due to an increase in enoxaparin use. Symptomatic VTE rate for patients who received enoxaparin in the post-implementation cohort decreased from 3.2 to 1.0 per cent ($P = 0.023$, 95% confidence interval = 0.124–0.856). There was also a significant decrease in the rate of symptomatic deep vein thrombosis (2.8% *vs* 0.9%, $P = 0.040$, 95% confidence interval = 0.117–0.950). This approach to VTE prophylaxis with enoxaparin resulted in a significant reduction in symptomatic VTE rates. Implementation of similar practices may be equally impactful in other institutions that use enoxaparin.

Trauma patients carry an especially high risk for venous thromboembolism (VTE).[1,2] The incidence of deep vein thrombosis (DVT) after severe trauma has been estimated to be as high as 70 per cent in patients not receiving preventative therapies.[1, 2] Sequelae of DVTs, such as pulmonary embolism (PE), have been reported as the third leading cause of death in trauma patients surviving past the initial 24 hours.[3, 4] Numerous factors contribute to the frequency of VTE development after severe trauma, including injury type and severity,

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requirements for massive transfusion, surgical intervention, immobility, extended hospital stays, and acute inflammatory processes.[1–9] Accordingly, present guidelines recommend that all trauma patients without contraindications receive VTE prophylaxis.[1, 4]

Benchmarking data from the American College of Surgeons Trauma Quality Improvement Program (TQIP) suggest that 45 to 60 per cent of all trauma patients admitted to United States hospitals receive pharmacologic VTE prophylaxis.[10] Recommended agents for pharmacologic prophylaxis in this patient population are low-molecular weight heparins (LMWH).[8, 9, 11] These recommendations are reflected in TQIP data suggesting that 70 per cent of the patients who receive pharmacologic VTE prophylaxis are administered LMWHs. Although these agents are the most frequently used modality for pharmacologic prevention, only 35 to 40 per cent of all trauma admissions receive pharmacologic prophylaxis with LMWH. Despite the relatively low rate of pharmacologic prophylaxis with LMWH, TQIP cites an average DVT and PE rate of 1.5 per cent and 0.6 per cent, respectively.[10]

At the study institution, enoxaparin is the most widely used LMWH. The recommended enoxaparin dosing regimen for prophylaxis is 30 mg twice daily administered subcutaneously (SQ).[8, 9, 11] Although enoxaparin does not require laboratory monitoring, the efficacy can be monitored by measuring anti-factor Xa levels. A peak anti-factor Xa level of 0.2 to 0.5 IU/mL has previously been determined to predict adequate VTE prophylaxis.[12–14] However, there is recent evidence that standard enoxaparin dosing may be insufficient to achieve prophylactic anti-factor Xa levels in up to 70 per cent of all trauma patients.[2, 15]

Venous thromboembolism rates at the present study's institution exceeded TQIP benchmarked averages for 2015. Accordingly, the trauma service at the study institution endeavored to improve its performance with respect to VTE prevention. With a growing body of evidence suggesting suboptimal efficacy with routinely used enoxaparin regimens, the approach to the provision of pharmacologic prophylaxis was amended. A protocol consisting of an initial, weight-adjusted enoxaparin regimen with subsequent dosing adjustments guided by anti-factor Xa levels was developed. The aim of this quality improvement analysis was to use this targeted approach to pharmacologic prophylaxis to reduce the rate of symptomatic VTEs.

Materials and Methods

Protocol Development

Historically, pharmacologic VTE prophylaxis in the trauma patient population was not standardized at the study institution. Trauma services VTE Prophylaxis Algorithm prompted the implementation of pharmacologic prophylaxis but did not specify dosing strategies or pharmacologic modalities. The enoxaparin VTE prophylaxis protocol added pharmacologic management components to trauma services' VTE Prophylaxis Algorithm (Fig. 1). The updated algorithm and enoxaparin protocol was approved in October 2015 as a quality improvement endeavor focused on improving trauma patient outcomes through guided pharmacologic prevention of VTEs. Patients without contraindication(s) received an initial dose of enoxaparin based on their weight. Enoxaparin 30 mg administered SQ every 12 hours was ordered if weight was <100 kg and 40 mg SQ every 12 hours when weight was

≥100 kg. Dose administrations were scheduled for noon and midnight. Anti-factor Xa levels were checked with morning labs (approximately 4 AM) after the midnight administration of the 3rd or 4th dose. If a subtherapeutic anti-factor Xa level was reported (<0.2 IU/mL), each scheduled dose of enoxaparin was increased by 20 per cent. If a super-therapeutic level of anti-factor Xa level was reported (>0.5 IU/mL), each scheduled dose of enoxaparin was decreased by 20 per cent. Adjusted doses were rounded to the nearest zero. After each dosing change, anti-factor Xa levels were reordered after the same process and adjusted accordingly. This process was followed until a therapeutic anti-factor Xa level for prophylaxis (0.2–0.5 IU/mL) was achieved. For a patient whose initial 30 mg SQ every 12 hours or 40 mg SQ every 12 hours regimen resulted in a supertherapeutic anti-factor Xa level, the dosing frequency was reduced to once daily and no additional anti-factor Xa levels were evaluated.

Trauma services enoxaparin VTE prophylaxis protocol was contraindicated in patients with renal insufficiency (GFR/CrCL < 30 mL/min), thrombocytopenia <50,000/mcL, or the presence of injuries or clinical conditions outlined in the service's VTE Prophylaxis Algorithm (Fig. 1). Anti-factor Xa levels for monitoring enoxaparin were designated as "Lovenox Heparin Assays" by laboratory services at the study institution and are accordingly referenced as such in the protocol presented in Figure 2.

Patients and Study Design

Patients admitted to trauma services between October 2014 and September 2016 were included in the study. Symptomatic VTE rate was evaluated 12 months before (October 2014 to September 2015) and after (October 2015 to September 2016) implementation of the Trauma Services Enoxaparin VTE Prophylaxis protocol. Data were obtained from the trauma registry and patient charts were reviewed from electronic medical records. General demographics, Injury Severity Score (ISS), pre-existing conditions, presenting injuries, incidence of intensive care unit (ICU) admission, ICU and hospital length of stay (LOS), rate of massive transfusion, surgical procedures, and diagnosis of symptomatic VTE in the post-implementation phase were prospectively collected by the trauma registrars. Additional data extraction, including patients' body mass index (BMI), enoxaparin dosing, anti-factor Xa level(s), date and time of first enoxaparin administration, frequency of enoxaparin use, and enoxaparin failure rates were manually collected. For the purpose of this study, enoxaparin failure rate was operationalized as the development of a VTE after the administration of at least one prophylactic dose of enoxaparin. Venous thromboembolisms documented by the trauma registry were confirmed by investigators to be in compliance with the "ACS NTDB National Trauma Data Standard: Data Dictionary for 2016 Admissions."[15]

Surveillance studies for VTE in patients receiving pharmacologic prophylaxis are not regularly performed at the study institution and accordingly were not routinely obtained in this study. Formal studies evaluating patients for the presence of a VTE were ordered and performed when patients' clinical signs and symptomatology warranted a formal workup. Venous duplex ultrasonography and CT arteriography (CTA) was used for the diagnosis of DVT and PE, respectively.

Descriptive analysis was conducted for each variable. Means and standard deviations were computed for continuous variables and compared using *t*-tests. Data that were not normally distributed were reported as a median with corresponding interquartile range. Categorical variables were presented as proportions and compared using chi-square or Fisher's exact test. Differences in VTE rates for patients who received prophylaxis with enoxaparin were computed with binary logistic regression controlling for variables that significantly differed between the pre- and post-implementation phases. *P* values of ≤0.05 were considered significant. Analyses were performed using SPSS Version 22.0 (IBM Corp, Armonk, NY). Collection and presentation of this data (study #16–201) was approved by the CAMC/WVU-Charleston Division Institutional Review Board for the Protection of Human Subjects at Charleston Area Medical Center Institute.

Results

In the 12 months pre- and post-implementation, a total of 1542 and 1802 patients were admitted to trauma services, respectively. The overall rate of symptomatic VTE significantly decreased from pre- to post-implementation (2.0% *vs* 0.9%, $P = 0.009$). To verify that the significant decline in rate was not the result of increased enoxaparin use, an evaluation of the frequency of enoxaparin prophylaxis in the study groups was conducted. Data revealed that enoxaparin use was comparable in both phases with an overall rate of 30.4 per cent pre-implementation and 31.9 per cent post-implementation ($P = 0.602$) (Table 1).

An analysis of patients who received prophylaxis with enoxaparin during the pre- and post-implementation phases was then performed. Patients in both phases were comparable in age, gender, ISS, BMI, and time to first dosing of enoxaparin. Pre-existing conditions were comparable; however, the incidence of pulmonary comorbidities, which were all chronic obstructive pulmonary disease, significantly increased from pre- to post-implementation (2.1% *vs* 4.5%, $P = 0.035$) (Table 2). The incidence of massive transfusions and presenting injuries were also comparable in both time periods (Table 3). An additional comparison of surgical procedures revealed no significant differences in the frequency of interventions between the study cohorts (Table 4).

After adjusting for significant differences in comorbidities, the rate of symptomatic VTEs significantly decreased (3.2% *vs* 1.0%, $p = 0.018$, 95% confidence interval [CI] = 0.121–0.820) when the pre- and post-implementation cohorts were compared. Similar trends were noticed when the symptomatic DVT rate was exclusively compared between the pre- and post-implementation groups (2.8–0.9%, $P = 0.030$, 95% CI = 0.112–0.894). There was also a decrease in the post-implementation PE rate; however, this decrease was not significant (1.1% *vs* 0.2%, $P = 0.246$) (Table 5). The duration of hospitalization before VTE diagnosis was compared between the study cohorts. The median time to VTE diagnosis was10.0 [5.5–22.6] days in the pre-implementation group and 9.72 [3.5–13.7] days in the post-implementation group ($P = 0.470$). In addition, bleeding events were evaluated in the post-implementation cohort. There were four bleeding events that occurred within 48 hours of enoxaparin exposure in the post-implementation phase. All four cases involved erosive or ulcerative gastrointestinal lesions that were subsequently identified via endoscopic or colonoscopic studies.

Additional variables were evaluated to determine their effect on the incidence of symptomatic VTE between the compared cohorts. Time to initiation of enoxaparin prophylaxis was evaluated. There was no difference in the time to enoxaparin initiation with 2.50 days having elapsed in the pre-implementation group and 2.78 days in the post-implementation group ($P = 0.148$). Initiation of enoxaparin therapy was in compliance with the Enoxaparin Prophylaxis Protocol in 73.8 per cent of patients who received enoxaparin in the post-implementation phase. Average levels were lower in patients who developed symptomatic VTEs versus those who did not; however, the difference did not reach statistical significance (0.23 *vs* 0.34, $P = 0.061$). Anti-Xa levels that were not obtained were due to patients' hospitalization being of insufficient duration to allow for assay procurement or protocol noncompliance.

Surveillance studies for VTEs were not routinely performed in the pre- or post-implementation phases. Formal evaluations for VTE via CTA or venous duplex ultrasonography were ordered at the discretion of the trauma service based on clinical suspicion. Accordingly, rates of diagnostic tests for VTEs were evaluated to verify that practices governing the use of these tests did not change between the pre- and post-implementation phases. There were no significant differences in the rate of CTAs performed in the pre- (5.5%) *versus* post-implementation (5.2%) phases ($P = 0.817$). Similar rates of duplex ultrasonography studies were also performed between the study groups (25.7% *vs* 21.5%, $P = 0.109$) (Table 6).

Discussion

The heightened risk of VTE in trauma patients has been consistently documented throughout the literature.[1–9, 11, 15] As previously mentioned, the hyper-coagulable state experienced by patients after severe trauma is thought to be multifactorial. Several risk factors are commonplace in the hospitalized patient and are not unique to those who experience trauma. A few risk factors, however, such as massive transfusion, orthopedic injury, and the need for frequent surgical intervention are fairly unique. Of note, it is estimated that approximately 25 per cent of all patients with severe trauma present with a coagulopathy.[16–22] Those presenting with severe coagulopathy will often require massive transfusion. Coagulopathic patients surviving the initial 24 hours may develop a subsequent, prolonged hypercoagulable state that is thought to be influenced by the early depletion of endogenous protein C.[16–22] This physiologic process likely has an additive effect in predisposing these patients to hypercoagulability. In addition, the hypercoagulable state that ensues after severe polytrauma may persist for up to three weeks after injury(s).[7] There also seems to be an escalation in hypercoagulability for up to 10 days in a subset of patients who receive surgical intervention for orthopedic injuries.[5] As a result, VTE continues to be a significant cause of morbidity and mortality in the trauma patient population. Implementation of routine strategies targeted at minimizing the occurrence of VTEs has proven to be effective. This is indicated by the low incidence of VTE in the present study population at baseline (2.0%). Indeed, the relatively low frequency of VTE found in this study has been supported by previous data where mechanical and/or pharmacologic prevention is practiced.[7]

Despite the relatively low incidence of VTE with the implementation of preventative therapies, the impact on morbidity and mortality can be profound. A study of 7937 trauma patients conducted by Paffrath et al. found that the development of VTE after trauma occurred in approximately 1.8 per cent (n = 146) of patients. The incidence of sepsis, multiple organ failure, and hospital LOS, however, were all significantly higher for patients who developed a post-trauma VTE. In addition, the adjusted odds ratio for in-hospital mortality was doubled for patients who developed a post-trauma VTE. In the 146 patients of Paffrath's study who developed a VTE, 118 (80.8%) were receiving either mechanical or pharmacologic prophylaxis at the time of VTE diagnosis.[7] Despite the low incidence of post-trauma VTE when prophylaxis is used, the sequelae associated with its failure can be significant.

The potential efficacy of prophylaxis with LMWH has been shown to be enhanced when therapy achieves optimal anti-factor Xa levels.[2, 15] Monitoring of anti-factor Xa levels, however, has traditionally been reserved for patients who are considered to have altered LMWH pharmacokinetics such as obese, geriatric, and renal dysfunction patients.[12, 14] Recent literature suggests that this paradigm may need to be revisited as derangements in the pharmacokinetics of LMWH may not be limited to these previously highlighted populations.[2, 15, 23, 24]

The presence of altered LMWH pharmacokinetics and resulting suboptimal anti-factor Xa levels has been linked to increased VTE rates in the trauma patient population when traditional enoxaparin regimens of 30 mg SQ every 12 hours have been studied. Malinoski et al. reported the results of a study evaluating the efficacy of 30 mg of enoxaparin administered SQ twice daily in 54 critically ill trauma and surgery patients. In this study, peak anti-Xa levels were drawn after the third dose of enoxaparin. Subtherapeutic levels (≤0.1 IU/mL) were recorded in 50 per cent (n = 27) of the patients. The DVT rate in this subtherapeutic group was significantly higher than that of the patients who achieved therapeutic anti-factor Xa levels (37% *vs* 11%, $P = 0.026$).[2] An additional prospective study by Costantini et al.[15] measured anti-factor Xa levels in 61 trauma patients also receiving 30 mg of enoxaparin SQ twice daily. In this study, the regimen resulted in subtherapeutic anti-factor Xa levels (≤0.2 IU/mL) in 70 per cent of patients.

These studies suggest that higher doses of enoxaparin may improve the achievement of therapeutic anti-factor Xa levels, simultaneously improving clinical efficacy in the prevention of VTEs. This hypothesis was supported by a study conducted by Nunez et al., which compared the frequency of achieving therapeutic anti-factor Xa levels between historic (30 mg of enoxaparin SQ every 12 hours) and a prospective cohort that received 0.6mg/kg SQ every 12 hours of enoxaparin. Therapeutic anti-factor Xa levels were achieved in 8 per cent of the historic cohort and 61 per cent of the weight-based dosing cohort ($P < 0.0001$). The authors of this study concluded that a weight-based enoxaparin dosing strategy of 0.6 mg/kg administered SQ every 12 hours may provide a superior strategy for VTE prophylaxis in critically ill trauma patients.[3]

The present quality improvement study evaluated the efficacy of a weight-adjusted therapy for the initiation of pharmacologic prevention of symptomatic VTE with further dosing

adjustments guided by the prospective evaluation of anti-factor Xa assays. With the implementation of this pharmacotherapeutic approach, the trauma service's overall rate of symptomatic VTE significantly decreased. The enoxaparin protocol also facilitated a significant improvement in pharmacologic prevention, as evidenced by the decrease in symptomatic VTEs during the post-implementation phase for patients who received prophylaxis with enoxaparin.

Despite this study's large sample size and the considerable improvement elicited in the frequency of symptomatic VTE and enoxaparin failures, the study is not without limitations. The data for the pre-implementation and a portion of the post-implementation cohorts were collected retrospectively. Accordingly, this data could have been affected by inaccuracies in the medical record and data extraction errors. In addition, noncompliance with the protocol may have been influenced by the frequent turnover of resident staff and the need for continued education concerning the new practice. The incidence of pharmacologic prophylaxis with enoxaparin was just above 30 per cent in both the pre- and post-implementation study groups. Although this seems to be a low rate of pharmacologic prophylaxis, it is consistent with national benchmarking data provided in American College of Surgeons TQIP reports for 2015 and 2016. In these reports, LMWH was used for the provision of pharmacologic VTE prophylaxis for approximately 35 to 36 per cent of all patients.[10] Finally, given the inherent limitations of a quality improvement study; it was not within the scope of this study to evaluate this approach to enoxaparin prophylaxis on the development of asymptomatic VTEs.

Conclusions

Given the significant impact of VTEs on morbidity and mortality in trauma patients who experience these post-injury complications, significant efforts should be taken to ensure that preventative therapy is consistently optimized. This study provides clinical evidence supporting the efficacy of an original protocol for the management of enoxaparin in the prevention of symptomatic VTEs in the trauma patient population. This weight-adjusted approach to initial dosing followed by additional titration(s) based on anti-factor Xa levels resulted in a dramatic decrease in symptomatic VTE rates. This approach offers an efficacious management strategy that could be implemented by other institutions that use enoxaparin for prophylaxis and have the ability to evaluate enoxaparin therapy with anti-factor Xa assays.

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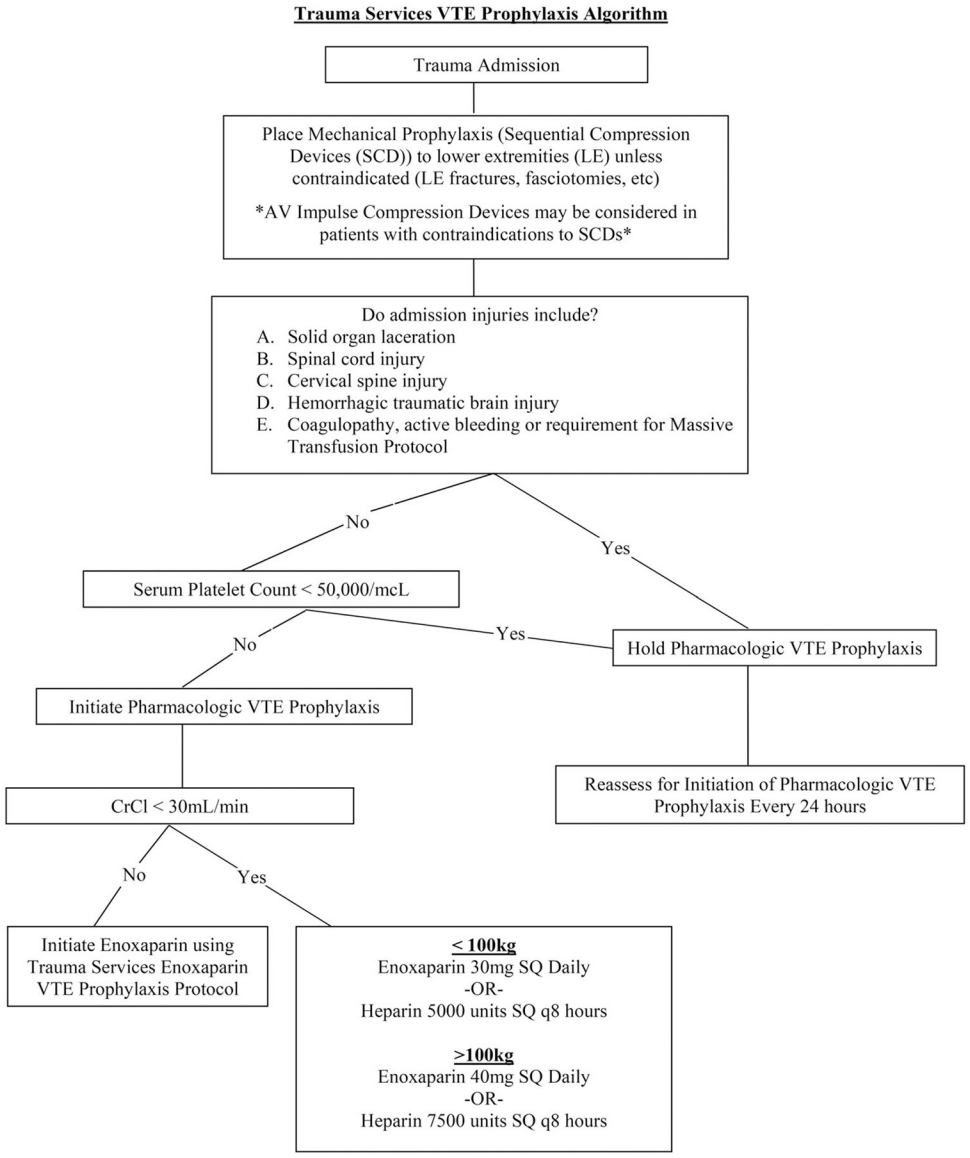

Fig. 1.
Trauma Services VTE Prophylaxis Algorithm.

Trauma Services Enoxaparin VTE Prophylaxis Protocol

1. Enoxaparin dosing scheduled at 12:00 and 00:00
   - <100 kg → 30mg SQ BID or >100 kg → 40mg SQ BID
2. Check an anti-factor Xa level (Lovenox Heparin Assay) after 3rd or 4th dose (with a.m. labs)
   - If sub-therapeutic (<0.2 IU/mL) → Increase each dose by 20% (each dose rounded to the nearest zero)
   - If super-therapeutic (>0.5 IU/mL) → Decrease each dose by 20% (each dose rounded to the nearest zero)
     - ➢ Regimens of 30mg SQ q12h should be changed to once daily regimens
       - <100 kg → 30mg SQ qday or >100kg → 40mg SQ qday
3. After each dosing change (BID regimens only), repeat anti-factor Xa level (Lovenox Heparin Assay) after the 3rd or 4th dose and adjust accordingly as outlined above
4. Protocol management is contraindicated for any of the following reasons:
   - Renal insufficiency (GFR/CrCl <30 mL/min), thrombocytopenia <50, 000/mcL, injuries/clinical scenarios as outlined in "Trauma Service's VTE Algorithm"

Fig. 2.
Trauma Services Enoxaparin VTE Prophylaxis Protocol.

Table 1.

Overall VTE Rate and Enoxaparin Use

All Patients	Pre-implementation (n = 1542)	Post-implementation (n = 1802)	P Value
Overall VTE rate	31 (2.0%)	17 (0.9%)	**0.009**
Use of enoxaparin prophylaxis	470 (30.4%)	576 (31.9%)	0.602

Bold face indicates statistical significance (P ≤ 0.05).

Table 2.

Demographics of Patients Treated with Enoxaparin

	Pre-implementation (n = 470)	Post-implementation (n = 576)	P Value
Age	48.85 ± 18.34	51.14 ± 19.55	0.053
Gender (male)	286 (60.9%)	360 (62.5%)	0.585
ISS	12.80 ± 7.70	12.81 ± 8.33	0.990
BMI	28.43 ± 8.40	29.12 ± 10.62	0.280
Time to enoxaparin therapy (days)	2.50 ± 2.99	2.78 ± 3.27	0.155
ICU Admission *	332 (96.8%)	267 (95.7%)	0.472
ICU LOS *	5.26 ± 9.17	5.74 ± 7.82	0.499
Hospital LOS †	9.28 ± 14.64	9.47 ± 11.15	0.814
Comorbidities			
Smoking	216 (46.0%)	256 (44.4%)	0.625
Malignancy	2 (0.4%)	4 (0.7%)	0.696
Neurologic	51 (10.9%)	57 (9.9%)	0.614
Pulmonary	10 (2.1%)	26 (4.5%)	**0.035**
Gastrointestinal	5 (1.1%)	10 (1.7%)	0.440
Cardiovascular	208 (44.3%)	245 (42.5%)	0.576
Renal failure	0 (0.0%)	2 (0.3%)	0.201
Autoimmune	9 (1.9%)	19 (3.3%)	0.183
Hematologic	31 (6.6%)	48 (8.3%)	0.290
Diabetes mellitus	73 (15.5%)	79 (13.7%)	0.407

* ICU LOS and incidence was calculated with patients who stayed ≥1 day in the ICU.

† Hospital LOS was computed with patients having a hospital LOS of ≥1 day.

Bold face indicates statistical significance (P ≤ 0.05).

Table 3.

Incidence of Massive Transfusion and Injuries for Patients Treated with Enoxaparin

	Pre-implementation (n = 470)	Post-implementation (n = 576)	P Value
Massive transfusion *	33 (7.02%)	41 (7.12%)	0.950
Presenting injuries			
Cardiovascular	21 (4.5%)	31 (5.4%)	0.499
Thoracic	195 (41.5%)	226 (39.2%)	0.446
Urogenital	11 (2.3%)	16 (2.7%)	0.789
Adrenal	8 (1.7%)	6 (1.0%)	0.422
Gastrointestinal	38 (8.1%)	58 (10.1%)	0.269
Nervous system	253 (53.8%)	306 (53.1%)	0.976
Spinal cord	17 (3.6%)	22 (3.8%)	0.864
Traumatic brain injury	187 (39.8%)	219 (38.0%)	0.545
Peripheral nervous system	23 (4.9%)	24 (4.2%)	0.572
Orthopedic	306 (65.1%)	376 (65.2%)	0.984
Lower extremity fractures	180 (38.3%)	212 (36.8%)	0.605
Upper extremity fractures	131 (27.9%)	148 (25.7%)	0.419
Sprain/strain	36 (7.7%)	33 (5.7%)	0.211
Pelvic fractures	79 (16.8%)	92 (15.9%)	0.707
Dislocation	47 (10.0%)	44 (7.6%)	0.178
Other orthopedic	9 (1.9%)	10 (1.7%)	0.821
Oromaxofacial	77 (16.4%)	77 (13.4%)	0.167

Broad categories of injuries commonly associated with increased VTE risk (nervous system and orthopedics) are further divided into subgroups.

* Defined as > 10 units of red blood cells in 24 h.

Table 4.

Surgical Procedures for Patients Treated with Enoxaparin

	Pre-implementation (n = 470)	Post-implementation (n = 576)	P Value
Procedures per study cohort			
Cardiothoracic	8 (1.7%)	8 (1.4%)	0.694
Vascular	23 (4.9%)	25 (4.3%)	0.644
Urogenital	4 (0.9%)	3 (0.5%)	0.827
Gastrointestinal	20 (4.3%)	29 (5.0%)	0.594
Neurosurgical	36 (7.7%)	46 (8.0%)	0.857
Skin and mucosa	34 (7.2%)	39 (6.8%)	0.800
Orthopedic	210 (44.7%)	248 (43.1%)	0.604
Oromaxofacial	35 (7.4%)	31 (5.4%)	0.162

Table 5.

Time to Enoxaparin Initiation and VTE Rates

	Pre-implementation (n = 470)	Post-implementation (n = 576)	P Value	95% CI
Time to initiation				
Days	2.50 ± 2.99	2.78 ± 3.26	0.148	—
VTE rates				
VTE	15 (3.2%)	6 (1.0%)	**0.018**	0.121 to 0.820
DVT	13 (2.8%)	5 (0.9%)	**0.030**	0.112 to 0.894
PE	5 (1.1%)	1 (0.2%)	0.246	0.064 to 2.024

Bold face indicates statistical significance (P ≤ 0.05)

Table 6.

Incidence of Diagnostic Tests for VTE

	Pre-implementation (n = 470)	Post-implementation (n = 576)	P Value
CTA rate	26 (5.5%)	30 (5.2%)	0.817
Duplex rate (lower and upper extremities)	121 (25.7%)	124 (21.5%)	0.109