



Thrombotic Risk Associated with Inferior Vena Cava Filter Placement in Patients with Heparin-Induced Thrombocytopenia

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ABSTRACT

Purpose: This study sought to define thromboembolic risk and mortality in patients with heparin-induced thrombocytopenia (HIT) undergoing inferior vena cava filter (IVCF) placement, in light of the American Society of Hematology's 2018 guidelines against routine use of IVCFs in this population.

Methods: A total of 26 patients with HIT who received IVCFs were retrospectively reviewed, and the outcomes of this group were compared with those of 4,707 controls with either HIT or IVCFs alone and with reported outcomes in prior studies.

Results: The patient group demonstrated 6- and 12-month mortality rates of 26.9% and 30.8%, respectively, which did not differ significantly from those of the control groups and were in line with published mortality rates in the literature. The measured thromboembolic risk of 19.2% in the patient group was also within the range of published rates for patients with HIT or IVCF alone.

Conclusions: IVCF placement did not significantly increase the risk of thromboembolism or death in patients with HIT and may be a viable option in the subset of these patients who are not candidates for anticoagulation.

ABBREVIATIONS

ASH = American Society of Hematology, DVT = deep venous thrombosis, HIT = heparin-induced thrombocytopenia, IVCF = inferior vena cava filter, KPNC = Kaiser Permanente Northern California, PE = pulmonary embolism

Heparin-induced thrombocytopenia (HIT) is an adverse drug reaction to heparin, resulting in a reduced platelet count. There are 2 forms of the disorder, HIT Type I and Type II, with only the latter considered clinically significant. HIT Type I involves a mild thrombocytopenia that typically develops within 2 days of heparin exposure, which is mediated

by platelet aggregation and typically resolves spontaneously with continued heparin administration (1). By contrast, HIT Type II (hereafter referred to simply as HIT) is characterized by a more severe drop in platelet count 5–10 days after heparin exposure and constitutes a contraindication to future heparin administration. The mechanism for this form of the disorder is the formation of antibodies against a complex of heparin and platelet factor 4, which induces a prothrombotic state by causing platelet activation and thrombin generation (2).

Up to half of the patients affected by HIT experience thromboembolic events, most commonly deep venous thrombosis (DVT) and pulmonary embolism (PE), although peripheral arterial thrombosis and strokes can also occur (3–6). Rare but serious complications include phlegmasia alba dolens and phlegmasia cerulea dolens, a spectrum of severe DVTs of the extremities that can result in life-threatening limb ischemia and gangrene. In the case of phlegmasia cerulea dolens, amputation rates of 25%–40% and mortality rates of 20%–50% have been reported in the literature (7).

The incidence of HIT has been measured at 0.1%–0.2% in patients receiving a prophylactic-dose subcutaneous

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None of the authors have identified a conflict of interest.

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J Vasc Interv Radiol 2021; 32:1629–1634

<https://doi.org/10.1016/j.jvir.2021.08.025>

RESEARCH HIGHLIGHTS

- Current guidelines recommend against routine placement of inferior vena cava filters (IVCFs) in patients with heparin-induced thrombocytopenia (HIT) on the basis of limited data, suggesting a higher risk of thromboembolic complications in this patient population.
- Using a larger sample size of 26 patients with HIT with IVCFs as well as 4,707 controls with either HIT or IVCFs alone, this study demonstrated that the rate of thromboembolic events and overall mortality was not increased in patients with HIT receiving IVCFs.
- These findings suggest that IVCFs should be considered as a therapeutic option for patients with HIT for whom anticoagulation is contraindicated.

unfractionated heparin and low molecular weight heparin and 0.8%–2.6% in patients receiving a therapeutic-dose intravenous unfractionated heparin (8,9). Clinical suspicion for HIT can be estimated using the 4Ts score, which considers the extent of thrombocytopenia, the timing of onset relative to heparin exposure, the presence of thrombosis, and the possibility of other explanations for thrombocytopenia. Confirmatory testing includes immunoassays that detect antibodies against the heparin-PF4 complex and functional assays that measure platelet activation induced by a patient's serum (eg, serotonin release assay) (3).

Once a diagnosis of HIT has been established, it is imperative that heparin be discontinued and replaced by an alternative anticoagulant, typically a direct thrombin inhibitor or direct oral anticoagulant. Warfarin should also be avoided in these patients until they have been appropriately anticoagulated and the platelet count has recovered. The resolution of thrombocytopenia can be expected within a week of discontinuing heparin, although the thrombotic risk may remain elevated for months.

Inferior vena cava filters (IVCFs) are intravascular devices that are placed to prevent PEs from occurring in patients with thromboembolic disease who have failed anticoagulation or have an absolute contraindication to anticoagulation. Contraindications that would warrant IVCF placement include active bleeding, a severe bleeding diathesis, recent or planned procedures with high bleeding risk, major trauma, and a history of intracranial hemorrhage. IVCFs have been placed in patients with HIT for whom anticoagulation was contraindicated or unsuccessful. However, there are vanishingly few case reports and studies reporting outcomes in such patients.

The largest study to date to investigate IVCF placement in the setting of HIT was published as an abstract by Jung et al (10) in 2011. The authors retrospectively reviewed patient charts using the International Classification of Diseases, Ninth Revision (ICD-9) codes for HIT, thrombocytopenia, and IVCF placement and ultimately identified 10 patients with true HIT, as determined by hematologic evaluation and/or positive immunoassays, who had IVCFs placed. Of these

STUDY DETAILS

Study type: Retrospective, observational, cohort study

Level of evidence: 3 (SIR-C)

10 patients meeting the inclusion criteria, 9 (90%) had experienced thromboembolic events, leading the authors to conclude that IVCFs carry a high risk of thrombosis in patients with HIT. Moreover, alluding to anecdotal evidence published elsewhere in the literature linking IVCF placement in HIT to phlegmasia cerulea dolens (11,12), the authors warn against the risk of “devastating thromboses in this extreme hypercoagulable milieu,” stating that “placement of IVC filters in the setting of HIT or even prior insertion of IVC filter is associated with higher risk of thrombosis, and could lead to devastating anticoagulation-resistant limb threatening thrombosis” (10).

Based in large part on the findings of Jung et al (10), the American Society of Hematology (ASH), in their 2018 guidelines for the management of venous thromboembolism, “recommends against routine insertion of an [IVCF]... in patients with [HIT]” (2). The guidelines also cited a retrospective study by Hong et al (13), which showed a 9.7% risk of upper extremity DVTs in patients with HIT receiving central venous catheters. Finally, a number of randomized controlled trials of IVCFs in patients without HIT were referenced to demonstrate a higher rate of recurrent DVTs and only “trivial reductions in [PEs]...and major bleeding” following the placement of IVCFs (14,15). In light of these findings, many of which were only indirectly related to the question of IVCF placement in patients with HIT, the guidelines concluded that the risks associated with IVCF placement in this setting outweigh the benefits.

Given the scarcity of data on the safety of IVCFs in patients with HIT and the limited options and dire need of methods for minimizing the thrombotic risk in patients with HIT who have failed anticoagulation or have a contraindication to it, this retrospective study set out to investigate outcomes in patients with HIT receiving IVCFs.

METHODS

Patient Selection and Subclassification

Following approval by the institutional review board of Kaiser Permanente Northern California (KPNC), the process of patient selection began with an automated search of the KPNC electronic medical record database for patient charts labeled with diagnostic and procedural codes corresponding to the placement of an IVCF and either a diagnosis of HIT or its presence on the problem list. This search was limited to records between the years 2006 and 2015, comprising more than 4 million patients within the KPNC network. The charts of patients labeled with diagnostic codes for both IVCF placement and HIT were manually reviewed to ensure that they had received an IVCF and that they had a confirmed HIT diagnosis using immunoassays and

Table 1. Demographics of the Patient and Control Groups

Demographic variable	HIT only (n = 814)	IVCF only (n = 3,893)	HIT and IVCF (n = 26)	P value	Total (N = 4,733)
Age, mean ± SD	67.46 ± 13.9	67.30 ± 15.9	65.3 ± 11.8	.78	67.32 ± 15.5
Sex				.09	
Female, n (%)	378 (46.4)	1,946 (50.0)	16 (61.5)		2,340 (49.4)
Male, n (%)	436 (53.6)	1,947 (50.0)	10 (38.5)		2,393 (50.6)

HIT = heparin-induced thrombocytopenia; IVCF = inferior vena cava filter.

Table 2. Average Treatment Effects on 6- and 12-Month Mortality in Patient and Control Groups

Outcome variable	Patient vs control group		ATE	P value
6-mo mortality	HIT+IVCF (n = 26)		−0.02	.86
12-mo mortality	HIT only (N = 814)		−0.06	.54
6-mo mortality	IVCF only (N = 3,893)		−0.02	.88
12-mo mortality	IVCF only (N = 3,893)		−0.06	.61
6-mo mortality	HIT or IVCF (N = 4,707)		−0.02	.86
12-mo mortality	HIT or IVCF (N = 4,707)		−0.06	.59

ATE = average treatment effects; HIT = heparin-induced thrombocytopenia; IVCF = inferior vena cava filter.

functional assays. Patients whose charts had been miscoded, meaning that they had not had an IVCF placed or had negative or incomplete HIT testing, were excluded from the study. Moreover, patients who were found to have IVCFs placed more than 14 days after being diagnosed with HIT were also excluded to ensure that patients in the study were at their peak HIT-associated thromboembolic risk.

The aforementioned search of the KPNC database yielded 2 control groups for comparison with the patient (HIT+IVCF) group. The first comprised all patients whose records had been coded with a diagnosis of HIT or its inclusion in the problem list between the years 2006 and 2015. The second included all patients with procedural codes for IVCF placement during this period. The far larger sample sizes of these control groups compared with the HIT+IVCF group precluded a manual chart review to exclude the miscoded patients.

Patients included in the study were subsequently subclassified on the basis of the likelihood of HIT and the timing of IVCF placement. A high likelihood of HIT was attributed to patients with a positive serotonin release assay or HIT antibody level of more than 1 and an intermediate likelihood to those with a HIT antibody level of less than 1. Patients were also subdivided based on whether their IVCF was placed more than 14 days before their HIT diagnosis or within 14 days before or after the diagnosis.

Measured Outcomes and Statistical Analysis

The HIT+IVCF group and its constituent subgroups were analyzed and compared with the control groups to determine thromboembolic risk after IVCF placement. More specifically, the outcomes measured included new or extended DVTs, new or extended PEs, inferior vena cava thrombosis,

lower extremity phlegmasia cerulea dolens, critical limb ischemia, and overall mortality. For the assessment of new or extended DVT or PE and inferior vena cava thrombosis, radiologic images were reviewed by a board-certified radiologist having an additional subspecialty certification in vascular and interventional radiology (S.L.W). Differences in outcomes, namely, thromboembolic events and mortality, in the various subgroups were analyzed using chi-squared and Fisher exact tests.

The demographics of the patient and control groups were also the subject of statistical analysis. To control for potential confounders of the treatment effect on 6- and 12-month mortality, propensity score matching was performed using the demographic variables of age and sex. Patients were paired using the nearest neighbor matching of logit-transformed propensity scores. The demographics of the patient and control groups were also compared using descriptive statistics and analyzed using a 2-sample *t* test and analysis of variance test.

All statistical analysis was performed using the Stata 15.1 IC software. *P* values of less than .05 were considered statistically significant.

RESULTS

During the 10-year period between 2006 and 2015, the records of 3,934 patients in the KPNC network were labeled with procedural codes for IVCF placement, 814 patients with codes for HIT, and 39 patients with codes for both IVCF placement and HIT. Upon reviewing the charts of these 39 patients, 2 were found not to have actually received IVCFs, 6 had not been diagnosed with HIT, and 1 had incomplete laboratory test results for HIT. An additional 4 patients carrying a diagnosis of HIT had received their IVCFs well more than 14 days after their diagnosis (range,

Table 3. Demographics of HIT+IVCF Subgroups

Variable	High likelihood of HIT* (n = 17)	Intermediate likelihood of HIT† (n = 9)	P value	IVCF placement ± 14 days of HIT diagnosis (n = 17)	IVCF placement >14 d before HIT diagnosis (n = 9)	P value	Total (N = 26)
Age, mean ± SD	67.4 ± 9.0	61.4 ± 15.8	.23	66.5 ± 9.3	63.1 ± 16.0	.49	65.3 ± 11.8
Sex, n (%)			.70			.19	
Female	10 (58.8)	6 (66.7)		12 (70.6)	4 (44.4)		16 (61.5)
Male	7 (41.2)	3 (33.3)		5 (29.4)	5 (55.6)		10 (38.5)
IVCF type, n (%)			.18			.78	
Permanent	2 (11.8)	3 (33.3)		3 (17.7)	2 (22.2)		5 (19.2)
Retrievable	15 (88.2)	6 (66.7)		14 (82.3)	7 (77.8)		21 (80.8)
Post-HIT anticoagulation, n (%)			.50			.02	
Argatroban	2 (17.6)	0 (0.0)		2 (11.8)	0 (0.0)		2 (7.69)
Coumadin	11 (64.7)	5 (55.6)		13 (76.5)	3 (33.3)		16 (61.5)
Fondaparinux	0 (0.0)	1 (11.1)		0 (0.0)	1 (11.1)		1 (3.9)
None	3 (17.65)	3 (33.3)		2 (11.76)	4 (44.4)		6 (23.1)
Other	0 (0.0)	1 (11.1)		0 (0.0)	1 (11.1)		1 (3.9)

HIT = heparin-induced thrombocytopenia; IVCF = inferior vena cava filter.

*SRA-positive or HIT antibody level >1.

†HIT antibody level <1.

111–1397 days) and were therefore excluded, leaving the HIT+IVCF group with a sample size of 26. The demographics of this patient group and the 2 control groups consisting of all patients with HIT and IVCFs, respectively, are summarized in **Table 1**. The propensity score matching performed on these groups did not demonstrate a significant difference in the average treatment effect on 6- or 12-month mortality (**Table 2**).

Of the 26 individuals in the HIT+IVCF group, 17 met the criteria for a high likelihood of HIT, and the remaining 9 were found to have an intermediate likelihood. The number of patients who had their IVCFs placed within 14 days of HIT diagnosis and more than 14 days before HIT diagnosis was also 17 and 9, respectively. The demographics of these subgroups and the types of IVCFs placed and alternative anticoagulation initiated after HIT diagnosis are described in **Table 3**. The specific IVCF models that were used include the Günther Tulip (Cook Medical, Bloomington, Indiana) (n = 10), OPTEASE (Cordis, Fremont, California) (n = 7), Argon Option (Argon, Frisco, Texas) (n = 2), Celect (Cook Medical) (n = 2), TRAPEASE (Cordis) (n = 2), VenaTech LP (n = 2) (B Braun, Bethlehem, Pennsylvania), and Bird's Nest (Cook Medical) (n = 1). The indications for which IVCFs were placed in the HIT+IVCF group are outlined in **Table 4**. The most common indication was a proximal DVT or PE with a contraindication to anticoagulation, accounting for 69.2% of cases. This figure is in line with a previously published study reporting the indications for IVCF placement in all patients within the KPNC network, which found that 76% of IVCFs were placed for a similar indication (16).

Table 4. Indications for IVCF Placement in the HIT+IVCF Group

Indication	n	%
Proximal DVT/PE with contraindication to anticoagulation	18	69.2
Recurrent PE despite adequate anticoagulation	2	7.7
Massive PE* with residual DVT	3	11.5
Proximal DVT/PE and inability for therapeutic anticoagulation	1	3.9
Free-floating iliofemoral thrombus	1	3.9
Prophylactic for surgery at high risk for VTE	1	3.9

DVT = deep venous thrombosis; HIT = heparin-induced thrombocytopenia; IVCF = inferior vena cava filter; PE = pulmonary embolism; VTE = venous thromboembolism.

*Massive PE is defined as one resulting in sustained hypotension and requiring inotropic support.

A total of 5 thromboembolic complications occurred in 3 of the 26 (11.5%) individuals in the HIT+IVCF group. All 3 patients demonstrated radiologic or clinical evidence of DVT progression, with 1 (3.9%) patient also demonstrating complete inferior vena cava thrombosis and 1 (3.9%) patient experiencing phlegmasia cerulea dolens (**Table 5**). In the latter case, the association between IVCF placement and phlegmasia was not well established. The patient was found to have extensive DVTs in the bilateral lower extremities and discoloration and coolness in the upper extremities 10 days after a neurosurgical procedure despite prophylactic heparin administration, prompting heparin dosing at therapeutic levels and IVCF placement the following day. In the ensuing days, clinical suspicion for

Table 5. Outcomes in the HIT+IVCF Group and Subgroups

Outcome variable	High likelihood of HIT* (n = 17)	Intermediate likelihood of HIT† (n = 9)	P value	IVCF placement ± 14 days of HIT diagnosis (n = 17)	IVCF filter placement >14 d before HIT diagnosis (n = 9)	P value	Total (N = 26)
6-mo mortality	4 (23.5)	3 (33.3)	.59	5 (29.4)	2 (22.2)	.69	7 (26.9)
12-mo mortality	4 (23.5)	4 (44.4)	.27	5 (29.4)	3 (33.3)	.84	8 (30.8)
DVT progression	3 (17.7)	0 (0.0)	.26	2 (11.8)	1 (11.1)	.73	3 (11.5)
Complete IVC thrombosis	1 (5.9)	0 (0.0)	.65	1 (5.9)	0 (0.0)	.65	1 (3.9)
Phlegmasia cerulea dolens	1 (5.9)	0 (0.0)	.65	0 (0.0)	1 (11.1)	.35	1 (3.9)
Total thrombotic complications‡	5 (29.4)	0 (0.0)		3 (17.7)	2 (22.2)		5 (19.2)
Patients with thrombotic complications	3 (17.7)	0 (0.0)	.26	2 (11.8)	1 (11.1)	.73	3 (11.5)

DVT = deep venous thrombosis; HIT = heparin-induced thrombocytopenia; IVC = inferior vena cava; IVCF = inferior vena cava filter.

*SRA-positive or HIT antibody level >1.

†HIT antibody level <1.

‡Cumulative incidence of DVT progression, partial or complete IVC thrombosis, and phlegmasia.

Table 6. Mortality in the HIT+IVCF and Control Groups

Outcome variable	HIT only (n = 814)	IVCF only (n = 3,893)	HIT and IVCF (n = 26)	P value	Total (N = 4,733)
6-mo mortality	240 (29.5)	1,288 (33.1)	7 (26.9)	.11	1,535 (32.4)
12-mo mortality	284 (34.9)	1,486 (38.2)	8 (30.8)	.17	1,778 (37.6)

HIT = heparin-induced thrombocytopenia; IVCF = inferior vena cava filter.

HIT rose, and the patient's vascular complications persisted, culminating in the patient's death despite a modified anticoagulation regimen. As the patient began demonstrating signs of phlegmasia the day prior to IVCF placement, it remains unclear whether this intervention exacerbated the patient's vascular complications or contributed to their death.

The overall mortality rate in the HIT+IVCF group at 6 and 12 months was 26.9% and 30.8%, respectively. No significant difference was found in mortality or in the incidence of thromboembolic events between the patient subgroups based on the likelihood of HIT or the timing of IVCF placement. The 6- and 12-month mortality rates of controls with either HIT or IVCFs alone were not significantly different from those of patients with both HIT and IVCFs (Table 6). Mortality rates among these 3 groups ranged from 26.9% to 33.1% at 6 months and from 30.8% to 38.2% at 12 months. At both the time points, the IVCF control group demonstrated the highest mortality and the HIT+IVCF group demonstrated the lowest.

DISCUSSION

Prior publications have reported high rates of thromboembolic events, including limb-threatening phlegmasia cerulea dolens, in patients with HIT following the placement of IVCFs and central venous catheters (10,13). The most recent ASH guidelines recommended against the routine insertion of IVCFs in patients with HIT largely based on these data (2). However, with a sample size more than twice the only directly comparable study, this study has demonstrated a substantially lower incidence of thromboembolic complications (19.2%) in patients with HIT with IVCFs. In fact, a rate of 19.2% is no higher than the estimated thrombotic risk in all patients with HIT; large retrospective studies comprising hundreds of patients with HIT have reported an incidence of 29%–53% for all thromboembolic events and 25%–40% for PEs in particular (4–6). Moreover, the rate of inferior vena cava thrombosis in this study (3.9%) fell within the reported range of 2%–30% for all patients with IVCFs (17). Similarly, the observed 12-month mortality rate in the HIT+IVCF group (30.8%) was not only in line with prior studies (4,18,19) of patients with HIT that had reported mortality rates between 15% and 30% but also was actually lower than those of the 2 control groups in this study comprising individuals who had either been diagnosed with HIT (34.9%) or had an IVCF placed (38.2%). These findings suggest that the presence of an IVCF did not substantially increase the risk of thrombosis or death.

Although this study is the largest of its kind to date, the rarity of IVCF placement in patients with HIT renders the sample size as one of the key limitations. By contrast, the far larger sample sizes of the control groups presented a different limitation, namely that the records of patients in

these groups could not be reviewed to verify that a diagnosis of HIT had been made or that an IVCF had been placed. These control groups provided data on mortality but not thromboembolic events, necessitating a reliance on the previously reported rates of thrombosis for comparison of the HIT+IVCF group. In addition, the retrospective design of the study constitutes another methodologic limitation.

In conclusion, this study demonstrated that the thrombotic risk in patients with HIT receiving IVCFs was no higher than what would be expected in patients with either a HIT diagnosis or an IVCF alone and that there was no significant difference in mortality rates between these 3 groups. This runs counter to the limited data available in the literature, where thrombotic rates in patients with HIT have been reported to be as high as 90%, as well as the most recent ASH guidelines, which recommended against IVCF placement in patients with HIT based primarily on those limited data (2,10). The findings of this study therefore warrant further validation with additional studies, as they may change the standard of care. If it is determined that IVCFs are relatively safe in the setting of HIT, patients, particularly those who are not candidates for anticoagulation, would be provided with a means of mitigating a high risk of morbidity and mortality associated with thromboembolic events.

ACKNOWLEDGMENTS

The authors thank Regina Loo and Timothy Weber of the data analytics division at Kaiser Permanente Santa Clara for their assistance in identifying the patient and control groups using diagnostic and procedural codes.

REFERENCES

1. Arepally GM. Heparin-induced thrombocytopenia. *Blood* 2017; 129:2864–2872.
2. Cuker A, Arepally GM, Chong BH, et al. American Society of Hematology 2018 guidelines for management of venous thromboembolism: heparin-induced thrombocytopenia. *Blood Adv* 2018; 2:3360–3392.
3. Greinacher A. CLINICAL PRACTICE. Heparin-induced thrombocytopenia. *N Engl J Med* 2015; 373:252–261.
4. Warkentin TE, Kelton JG. A 14-year study of heparin-induced thrombocytopenia. *Am J Med* 1996; 101:502–507.
5. Nand S, Wong W, Yuen B, Yetter A, Schmulbach E, Gross Fisher S. Heparin-induced thrombocytopenia with thrombosis: Incidence, analysis of risk factors, and clinical outcomes in 108 consecutive patients treated at a single institution. *Am J Hematol* 1997; 56:12–16.
6. Greinacher A, Farnier B, Kroll H, Kohlmann T, Warkentin TE, Eichler P. Clinical features of heparin-induced thrombocytopenia including risk factors for thrombosis. A retrospective analysis of 408 patients. *Thromb Haemostasis* 2005; 94:132–135.
7. Chinsakchai K, Ten Duis K, Moll FL, de Borst GJ. Trends in management of phlegmasia cerulea dolens. *Vasc Endovascular Surg* 2011; 45:5–14.
8. Smythe MA, Koerber JM, Mattson JC. The incidence of recognized heparin-induced thrombocytopenia in a large, tertiary care teaching hospital. *Chest* 2007; 131:1644–1649.
9. Martel N, Lee J, Wells PS. Risk for heparin-induced thrombocytopenia with unfractionated and low-molecular-weight heparin thromboprophylaxis: a meta-analysis. *Blood* 2005; 106:2710–2715.
10. Hong AP, McCarthy JJ, Baker KR, Rice L. Safety of IVC filters with heparin-induced thrombocytopenia: a retrospective study. *Blood* 2011; 118:2225.
11. Rice L. Heparin-induced thrombocytopenia: myths and misconceptions (that will cause trouble for you and your patient). *Arch Intern Med* 2004; 164:1961–1964.
12. Baker K. Points to remember: Tips on Heparin-Induced Thrombocytopenia (HIT). *Methodist Debaque Cardiovasc J* 2014; 10:57.
13. Hong AP, Cook DJ, Sigouin CS, Warkentin TE. Central venous catheters and upper-extremity deep-vein thrombosis complicating immune heparin-induced thrombocytopenia. *Blood* 2003; 101:3049–3051.
14. Mismetti P, Laporte S, Pellerin O, et al. Effect of a retrievable inferior vena cava filter plus anticoagulation vs anticoagulation alone on risk of recurrent pulmonary embolism: a randomized clinical trial. *JAMA* 2015; 313:1627–1635.
15. Decousus H, Leizorovicz A, Parent F, et al. A clinical trial of vena caval filters in the prevention of pulmonary embolism in patients with proximal deep-vein thrombosis. *Prévention du Risque d'Embolie Pulmonaire par Interruption Cave Study Group. N Engl J Med* 1998; 338:409–415.
16. Wang SL, Cha HH, Lin JR, et al. Impact of physician education and a dedicated inferior vena cava filter tracking system on inferior vena cava filter use and retrieval rates across a large US health care region. *J Vasc Interv Radiol* 2016; 27:740–748.
17. Caplin DM, Nikolic B, Kalva SP, et al. Quality improvement guidelines for the performance of inferior vena cava filter placement for the prevention of pulmonary embolism. *J Vasc Interv Radiol* 2011; 22:1499–1506.
18. King DJ, Kelton JG. Heparin-associated thrombocytopenia. *Ann Intern Med* 1984; 100:535–540.
19. Lewis BE, Wallis DE, Berkowitz SD, et al. Argatroban anticoagulant therapy in patients with heparin-induced thrombocytopenia. *Circulation* 2001; 103:1838–1843.