Heparin is one of the most commonly administered parenteral therapeutic agents in hospitals. Each year, approximately 12 million patients—or one third of all hospitalized patients—are exposed to heparin.1,2 A serious complication called heparin-induced thrombocytopenia (HIT), which is associated with significant morbidity and mortality, may develop in some patients who receive heparin. Among patients exposed to heparin, HIT develops in approximately 1% to 5% (up to 600,000 patients). Moreover, many clinicians think that HIT is underrecognized and underdiagnosed.3,5

Consequences of HIT include venous or arterial thrombosis and, rarely, bleeding.1,6 In patients with acute thrombosis, HIT can be fatal. Treatment of HIT includes discontinuation of heparin therapy and the use of parenteral nonheparin anticoagulants such as direct thrombin inhibitors. In order to minimize the impact of HIT, however, preventive measures are essential. Nurses play a critical role in the early detection and prevention of the complications of HIT. This article provides a discussion of the risk factors, pathogenesis, diagnosis, manifestation, and management of HIT from the perspective of critical care nurses.

Risk Factors for HIT

The clinical importance of HIT is influenced by 4 factors: the widespread and increasing use of heparin, the potentially devastating consequences of the disorder, the unpredictability with which HIT occurs, and the uncertainty regarding diagnosis and treatment. All types of heparin, from bovine or porcine sources, can cause HIT.1,6,7 Although both low-molecular-weight heparin (LMWH) and unfractionated heparin can cause HIT, the abnormality is more common in patients treated with unfractionated heparin than in patients treated with LMWH.8,9 However, the incidence of thrombosis in patients with HIT who have received unfractionated heparin and those who received LMWH is thought to be similar.
Use of heparin is ubiquitous in hospitals, and the administration of heparin may not be documented. Use of heparin should be avoided or minimized wherever possible, for example, by flushing peripheral and central catheters with isotonic sodium chloride solution or a nonheparinized solution rather than with heparin. All efforts should be made to limit the duration of heparin administration. The use of alternative anticoagulant drugs or LMWH rather than unfractionated heparin should be considered.

The primary groups of patients affected by HIT are orthopedic, cardiovascular, and trauma patients. In addition, the relationship between the frequency of formation of an immunoglobulin G (IgG) antibody associated with HIT and the risk for HIT appears to vary depending on the population of patients. In a study of 744 patients receiving unfractionated heparin during or after cardiac surgery, unfractionated heparin after orthopedic surgery, or LMWH after orthopedic surgery, IgG antibodies associated with HIT were more likely to form in patients undergoing cardiac surgery than in orthopedic patients. Among the patients in whom antibodies did form, HIT was more likely to develop in the orthopedic patients than in the cardiac surgery patients.

Immune Versus Nonimmune HIT

The term HIT refers to a decrease in the platelet count shortly after the start of treatment with heparin and is now preferably reserved for describing immune-mediated HIT. A decrease in platelet count that occurs before day 5 of treatment usually is not immune-mediated HIT, but it may be a form of nonimmune heparin-associated thrombocytopenia (formerly called type I HIT), which generally resolves despite continued exposure to heparin. This transient, benign, and self-limiting form of thrombocytopenia appears to be caused by a direct agglutinating effect of heparin on platelets. In contrast, immune-mediated HIT (formerly called type II HIT and now more simply HIT) is due to an immunoglobulin-mediated reaction to the complex composed of heparin and platelet factor 4 (PF4) that forms during heparin treatment.

Immune-mediated HIT is characterized by the development of thrombocytopenia 5 to 14 days after initiation of heparin therapy (although time of onset can be sooner in patients who have been exposed to heparin in the preceding 3 months). In addition, thrombocytopenia associated with immune-mediated HIT should be considered when the platelet count decreases to less than 150 x 10^9/L or by 30% to 50% during treatment. The characteristics of the immune and nonimmune forms of HIT are compared in Table 1. From here on, the term HIT refers to immune-mediated HIT.

Pathophysiology of HIT

The underlying pathophysiology of HIT (Figure 1) is an immune reaction to heparin. First, heparin binds to PF4, forming a highly immunogenic complex on the surface of platelets. Then, in susceptible patients, an IgG antibody to the antigenic heparin-PF4 complex develops. The resulting antibody binds with the heparin-PF4 complex, which in turn activates platelets via Fc receptors present on the surface of the platelets. Thrombocytopenia develops as the reticuloendothelial system destroys activated platelets. In addition, platelet activation leads to the generation of procoagulant-rich microparticles, which contribute to a thrombotic state.

Consequences of HIT

HIT thrombotic syndrome (HITTS) is the most serious complication associated with HIT. Without the use of an alternative treatment, the overall risk of thrombosis in patients with HIT is 38% to 76%, according to data from 3 studies involving 425 patients. If thrombotic syndrome develops, limb amputation is required in about 20% of patients, and 30% to 50% of

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Immune</th>
<th>Nonimmune</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of onset after start of heparin therapy, days</td>
<td>≥5 days, unless recent previous exposure to heparin</td>
<td>&lt;5</td>
</tr>
<tr>
<td>Frequency, % of patients</td>
<td>&lt;5</td>
<td>10-30</td>
</tr>
<tr>
<td>Decrease in platelets</td>
<td>Moderate to severe</td>
<td>Slight</td>
</tr>
<tr>
<td>Antibodies present</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Thrombosis, % of patients</td>
<td>30-75</td>
<td>0</td>
</tr>
<tr>
<td>Management</td>
<td>Stop heparin and start treatment with alternative anticoagulants</td>
<td>Observe patient</td>
</tr>
</tbody>
</table>
Table 2 Clinical criteria for diagnosing heparin-induced thrombocytopenia in most patients

<table>
<thead>
<tr>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombocytopenia &lt;150 x 10^9/L or a 30%-50% decrease in platelet count</td>
</tr>
<tr>
<td>from baseline</td>
</tr>
<tr>
<td>Onset 5-14 days after starting any dose, any type, or any route of</td>
</tr>
<tr>
<td>heparin exposure</td>
</tr>
<tr>
<td>Exclusion of other causes of thrombocytopenia</td>
</tr>
</tbody>
</table>

Timing of Onset

In most patients, the classical signs and symptoms of HIT develop between 5 and 14 days after heparin

And third, in hospitalized patients, thrombocytopenia may arise from many other causes, including disseminated intravascular coagulation, septicemia/sepsis hemodilution, hypercoagulable states, hemodialysis, multisystem organ failure, and primary bone marrow disorder. Thus, other causes of thrombocytopenia should be ruled out before HIT is diagnosed.

Recognition of HIT is important to proper management of the condition, but the diagnosis of HIT can be challenging. Several clinical indicators can point astute nurses toward a diagnosis of HIT in patients receiving heparin therapy. HIT should be suspected if a patient has recently been exposed to heparin and if evidence of at least one of the following clinical events is apparent: an unexplained decrease in platelet count and/or the presence of thrombosis.

The clinical criteria for diagnosing HIT in most patients are listed in Table 2.

Diagnosis of HIT

Three factors may help explain why HIT is underdiagnosed. First, the name of the disease creates some confusion, because clinically significant thrombocytopenia may not be present in all patients with HIT. That is, the thrombocytopenia may be relative (a 30%-50% decrease) rather than absolute (less than the upper limit of the reference range). Second, thrombocytopenia in HIT is paradoxically associated with thrombosis, not with bleeding.

About 50% of patients with HIT develop deep venous thrombosis, and 25% of patients develop pulmonary embolism and acute systemic reaction, respectively. Other reported complications related to HIT include skin lesions at the injection site, acute limb ischemia, warfarin-associated venous limb gangrene, and acute thrombotic stroke or myocardial infarction.

Critical care nurses can assess patients for signs of thrombosis and also use measures to prevent deep venous thrombosis. In addition to initiating anticoagulant therapy, other preventive measures include the following:

- using sequential compression devices and pneumatic compression stockings,
- increasing the patient’s activity,
- encouraging early ambulation,
- ensuring adequate hydration, and
- using exercises for preventing deep venous thrombosis.

Figure 1 Pathophysiology of heparin-induced thrombocytopenia.

treatment is started. However, HIT can develop quickly in patients reexposed to heparin. Therefore, knowing a patient’s history of exposure to heparin is crucial. In addition, delayed-onset HIT has occurred 9 to 45 days after treatment with heparin was discontinued.

Rapid-onset HIT is due to platelet activation caused by residual circulating antibodies associated with HIT. It is characterized by a decrease in the platelet count that begins soon after treatment with heparin is started and can occur within minutes, hours, or days of heparin administration. The patient will have recently been exposed to heparin, typically within the past 100 days, although rapid-onset HIT after a 165-day heparin-free interval has been described. In contrast, delayed-onset HIT occurs days or weeks after heparin has been stopped, perhaps after the patient has been discharged from the hospital. The patient may then again have signs or symptoms of thrombosis and be readmitted to the hospital. In patients with a delayed onset of HIT, development of new thromboses is typical, and further exposure to heparin can quickly worsen a patient’s clinical condition.

Degree of Thrombocytopenia

The actual platelet counts in patients with HIT may not be as important as the occurrence of a sudden decrease in platelet count of 30% to 50% from the patient’s baseline count. Decreases exceeding 50% are particularly indicative of HIT.

Laboratory Testing

To date, no reference standard diagnostic test for HIT is available. Current tests for HIT include activation assays, which detect the presence of functional antibodies, and antigen assays, which detect the presence of heparin-PF4 antibodies (Figure 2). Laboratory confirmation of the presence of antibodies to heparin can verify a diagnosis, but the assays used for HIT have variable sensitivity and specificity. In addition, assays often require a long turnaround time, and because of the hypercoagulable state of HIT, confirmatory results may not be available before treatment has been started. Thus, diagnosis of HIT is primarily based on clinical signs and symptoms; laboratory confirmation should be sought, but treatment must not be delayed.

Guidelines for Managing Patients With HIT

The first step in managing a patient with confirmed or suspected HIT is to stop administration of all forms of heparin. These include, but are not limited to, use of heparin infusions, LMWH, heparin flushes, heparin in dialysate, continuous hemofiltration catheters, heparin-coated pulmonary catheters, guidewires, and devices containing heparin. In addition, when HIT is suspected, or if a history of HIT is determined, critical care nurses must document this finding in the patient’s chart. In September 2004, the American College of Chest Physicians published clinical guidelines on the management of HIT that were developed by consensus of a nationwide panel of 87 physicians (Table 3).

Alternative Anticoagulants

In addition to discontinuation of heparin, the guidelines for management of HIT recommend the use of alternative anticoagulants in patients with strongly suspected or confirmed HIT, whether or not HIT is complicated by thrombosis. Discontinuation of heparin therapy alone is not sufficient, because it leaves patients in a hypercoagulable state. Currently, only 2 nonheparin anticoagulants, argatroban and lepirudin, are approved for use in the medical management of HIT. Argatroban is indicated in the United States for the prophylaxis or treatment of thrombosis associated with HIT and in patients undergoing percutaneous coronary intervention who have HIT or are at risk for HIT. Lepirudin is indicated for anticoagulation in patients with HIT and associated thromboembolic disease in order to prevent further thromboembolic complications (Table 4). These
agents are known as direct thrombin inhibitors (Table 5).

Unlike heparin, direct thrombin inhibitors directly block the activation of thrombin and do not trigger the antibody-mediated reactions that lead to HIT. A third direct thrombin inhibitor, bivalirudin, is approved for use in patients with or at risk for HIT during percutaneous coronary intervention but is not approved for medical use in HIT.

Many patients with HIT require long-term anticoagulation for an underlying condition and therefore may be candidates for warfarin therapy. HIT is a consumptive process and may cause depletion of the natural anticoagulant protein C. Early introduction of warfarin may exacerbate protein C depletion when the highly prothrombotic HIT state exists. The resulting imbalance between the natural anticoagulant and procoagulant proteins could lead to greater thrombotic risk and cause warfarin-induced thrombosis and venous limb gangrene.27,28 Warfarin is contraindicated in any patient with acute HIT until the patient has received treatment with an alternative anticoagulant for several days and the platelet count has recovered (or is >100 x 10^9/L). Guidelines for the use of oral anticoagulants and conversion of direct thrombin inhibitors to warfarin in patients with HIT are described in Table 6 and Figure 3.28

**Platelet Count Monitoring**

Monitoring platelet counts is essential for the timely detection of HIT. The guidelines for management of HIT recommend monitoring platelet counts during specific periods, depending on the patient’s history with heparin and the risk of HIT developing (Table 7). Platelet counts should be tracked until recovery. Because patients with HIT rarely bleed spontaneously, thrombocytopenia does not usually need to be treated with platelet transfusions. Platelet transfusions may increase the risk for platelet-mediated thrombosis.

Because of the availability of acceptable alternative anticoagulants, such as the direct thrombin inhibitors, it is advisable to avoid both unfractionated heparin and LMWH in patients with a history of HIT. In a

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**Table 3** American College of Chest Physicians 2004 guidelines for treatment of confirmed or strongly suspected cases of heparin-induced thrombocytopenia

<table>
<thead>
<tr>
<th>Step</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stop heparin therapy</td>
<td>Initiate therapy with alternative, nonheparin anticoagulant (eg, direct thrombin inhibitors)</td>
</tr>
<tr>
<td>Avoid vitamin K antagonist therapy</td>
<td>until platelet count has recovered</td>
</tr>
<tr>
<td>Administer phytonadione (vitamin K) if warfarin has already been started</td>
<td></td>
</tr>
<tr>
<td>Avoid low-molecular-weight heparin and prophylactic platelet transfusions</td>
<td></td>
</tr>
<tr>
<td>Assess lower limbs for deep venous thrombosis</td>
<td></td>
</tr>
</tbody>
</table>

---

**Table 4** Comparison of argatroban and lepirudin

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Argatroban</th>
<th>Lepirudin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Half-life</td>
<td>40-50 min</td>
<td>1.3 h</td>
</tr>
<tr>
<td>Elimination</td>
<td>Hepatic</td>
<td>Renal</td>
</tr>
<tr>
<td>Dosing</td>
<td>2 µg/kg per minute</td>
<td>0.4 mg/kg slow intravenous bolus followed by 0.15 mg/kg per hour as a continuous intravenous infusion for 2-10 days if clinically needed</td>
</tr>
<tr>
<td>Activated partial thromboplastin time</td>
<td>6</td>
<td>13.9</td>
</tr>
<tr>
<td>Antidote</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Pregnancy category</td>
<td>B</td>
<td>B</td>
</tr>
</tbody>
</table>

*Body weight up to 110 kg.
Reduce dose with liver failure.
Reduce dose with renal failure.

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**Table 5** Indications for use of a direct thrombin inhibitor

| Patients treated with a direct thrombin inhibitor for heparin-induced thrombocytopenia should meet one of the following criteria: |
| History of heparin-induced thrombocytopenia |
| Unexplained thrombocytopenia <150 x 10^9/L or 30%-50% less than the baseline |
| Unexplained thrombosis with or without thrombocytopenia |
| Laboratory test positive for heparin antibodies |
| Skin necrosis due to heparin |

| Patients with confirmed or suspected heparin-induced thrombocytopenia should be treated with a direct thrombin inhibitor rather than heparin for continued anticoagulation unless use of a direct thrombin inhibitor is contraindicated |
| Every molecule of heparin must be discontinued (ie, unfractionated heparin, low-molecular-weight heparin, flushes, and all coated catheters—pulmonary artery and dialysis catheters) |
prospective study, argatroban provided adequate anticoagulation for venous or arterial thrombosis in patients with a history of HIT, without major bleeding or thrombotic complications. In special circumstances, such as in the event of cardiac or vascular surgery, it may be necessary to expose a patient to heparin again. Repeat administration of heparin may be considered if HIT occurred a long time before (>100 days), the patient is negative for anti-bodies to the heparin-PF4 complex, and the exposure time will be limited to the surgical procedure itself.

Conclusion

Although techniques for preventing HIT have yet to be developed, the risk of HIT can be reduced by limiting the duration and extent of patients’ exposure to heparin. The risk of HIT may be somewhat reduced by the use of LMWH rather than unfractionated heparin because, although the risk of HIT still exists, the risk is lower with LMWH. Increasing use of non-heparin anticoagulants such as fondaparinux, a factor Xa inhibitor, or the direct thrombin inhibitors may result in a reduced incidence of HIT.

HIT can develop rapidly and can cause serious or even fatal consequences if it is not detected and treated early. Clearly, the most effective way to manage HIT is to try to prevent it from occurring in the first place. However, increased awareness of the signs and symptoms of the disorder is essential to prevent potentially dire, if not fatal, consequences. Critical care nurses can facilitate recognition by educating physicians, pharmacists, other nurses, and patients about HIT. Critical care nurses can ensure that heparin protocols address parameters for monitoring platelet count. Nurses can monitor patients for early signs of any HIT complications and clearly document and communicate each patient’s history of HIT. Critical care nurses can participate in quality control and surveillance programs.

Table 6 Guidelines for use of oral anticoagulants in patients with heparin-induced thrombocytopenia

| Do not use oral anticoagulants alone |
| Wait for the platelet count to increase to near normal while heparin-induced thrombocytopenia is “cooled” (100 x 10^9/L to 150 x 10^9/L) |
| Initiate modest doses, avoid overshooting the international normalized ratio, do not give bolus doses |
| Overlap minimum of 4-5 days |
| Be sure international normalized ratio is >2.0 for 2 consecutive days |
| Ensure adequate levels of alternative anticoagulant during transition |

Figure 3 Conversion of direct thrombin inhibitors to warfarin. Abbreviation: INR, international normalized ratio.

Lepirudin

Check INR daily

Argatroban

Overlap minimum 4-5 days
When INR >2.3, stop lepirudin infusion for 4-6 hours and measure INR again
If <2.0, resume infusion; if >2.0, continue warfarin alone

Overlap minimum 4-5 days
When INR >4.0, stop argatroban infusion for 4 hours and measure INR again
If <2.0, resume infusion; if >2.0, continue warfarin alone

Table 7 Guidelines for when and in whom to do platelet counts

<table>
<thead>
<tr>
<th>When to do platelet counts</th>
<th>In which patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>All patients</td>
</tr>
<tr>
<td>After heparin administration</td>
<td>Patients with known or suspected heparin exposure within previous 100 days</td>
</tr>
<tr>
<td>Repeat within 24 hours</td>
<td>Patients receiving therapeutic dose of unfractionated heparin</td>
</tr>
<tr>
<td>Every other day until day 14 or until heparin is discontinued (whichever comes first)</td>
<td>Postoperative patients receiving unfractionated heparin for prophylaxis</td>
</tr>
<tr>
<td>Starting day 4, then every 2 to 3 days until day 14 or until heparin is discontinued (whichever comes first)</td>
<td>Medical patients receiving prophylactic unfractionated heparin, postoperative patients receiving low-molecular-weight heparin, and patients receiving heparin flushes</td>
</tr>
<tr>
<td>No routine monitoring recommended</td>
<td>Medical/obstetric patients receiving only low-molecular-weight heparin or heparin flushes</td>
</tr>
</tbody>
</table>
and read literature to find and communicate HIT-related research. In short, the actions of critical care nurses can play a central role in treating, preventing, and building awareness of HIT.

References

1. Heparin-induced thrombocytopenia (HIT) occurs in approximately what fraction of hospitalized patients?
   a. One half  
   b. One third  
   c. One fourth  
   d. One fifth

2. Which one of the following is the most common consequence of HIT?
   a. Acute thrombosis  
   b. Acute bleeding/hemorrhage  
   c. Disseminated intravascular coagulation  
   d. Arterial emboli

3. Which of the following statements concerning HIT is true?
   a. Increased patient age  
   b. Acute thrombosis  
   c. Male sex of the patient  
   d. Orthopedic patients

4. The lowest occurrence of HIT occurs with which type of heparin?
   a. Bovine-sourced heparin  
   b. Low-molecular-weight heparin (LMWH)  
   c. Porcine-sourced heparin  
   d. Unfractionated heparin

5. Which patient population is most likely to develop HIT after forming immunoglobulin G (IgG) antibodies associated with this disorder?
   a. Cardiac surgery patients  
   b. Trauma patients  
   c. Neurovascular surgery patients  
   d. Orthopedic patients

6. Which of the following factors can lead to earlier development of thrombocytopenia after initiation of heparin therapy?
   a. Increased patient age  
   b. Coexisting cardiovascular disease  
   c. Male sex of the patient  
   d. Exposure to heparin in the preceding 3 months

7. Which of the following must be true before the diagnosis of HIT can be made?
   a. Disseminated intravascular coagulation and septicemia have been ruled out.  
   b. Clinically significant thrombocytopenia is present.  
   c. Heparin administration will occur only during the surgical procedure.  
   d. The patient’s platelet count is greater than 100

8. Which platelet count is most indicative of HIT?
   a. Decrease below 150 × 10^9/L  
   b. Decrease of 30% to 50% from the patient’s baseline  
   c. Decrease of 30% to 50% from the patient’s baseline and presence of acute bleeding  
   d. Decrease greater than 50% from the patient’s baseline

9. When HIT is suspected or confirmed, the critical care nurse should immediately take what action?
   a. Remove the patient’s pulmonary catheter  
   b. Check to see what dialysate is being used  
   c. Change from unfractionated heparin to LMWH  
   d. Obtain a laboratory measurement for fibrin split products

10. Which alternative anticoagulant is indicated for use in the management of HIT?
    a. Bivalirudin  
    b. Aspirin  
    c. Argatroban  
    d. Warfarin

11. Warfarin therapy in patients with HIT should not be instituted until which of the following laboratory results is obtained?
    a. Platelet count is > 100 × 10^9/L  
    b. Protein C level is within normal limits  
    c. Activated partial prothrombin time of ≤1.0  
    d. International normalized ratio >2 for 2 consecutive days

12. Which of the following statements regarding platelet count monitoring is true?
    a. Medical/obstetric patients receiving heparin flushes in addition to LMWH should be monitored every other day unless day 14 or heparin is discontinued (whichever comes first).  
    b. Patients receiving unfractionated heparin for postoperative prophylaxis should be monitored starting day 4, then every 2 to 3 days until heparin is discontinued or day 14 (whichever comes first).  
    c. Postoperative patients receiving LMWH should be monitored starting day 4, then every 2 to 3 days until heparin is discontinued or day 14 (whichever comes first).  
    d. The patient’s platelet count is greater than 100 × 10^9/L

13. A patient with a history of HIT may be reexposed to heparin in the event of vascular surgery under what conditions?
    a. The patient is negative for antibodies to the heparin-IgG immunoglobin complex.  
    b. The patient’s initial heparin treatment occurred more than 100 days earlier.  
    c. Heparin administration will occur only during the surgical procedure.  
    d. The patient’s platelet count is greater than 100 × 10^9/L

14. Critical care nurses can best improve early recognition of HIT by ensuring which of the following?
    a. Existence of heparin protocols that address parameters for monitoring platelet counts  
    b. Documentation of each patient’s history of HIT  
    c. Administration of LMWH rather than unfractionated heparin  
    d. Communication of HIT-related research findings to physicians, pharmacists, and patients

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

1. a  2. a  3. a  4. a  5. a  6. a  7. a  8. a  9. a  10. a  11. a  12. a  13. a  14. a

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