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HIT

The keys to improved outcomes in patients with HIT are early recognition and prompt and appropriate management.

By Alane J. Drexler, RN, MS

Mr. Roberts, who had chronic liver disease, was admitted with abdominal pain and found to have thrombosis of the superior mesenteric vein with an ischemic bowel. After surgery to resect the diseased bowel, Mr. Roberts was placed on an unfractionated heparin (UFH) infusion to prevent further ischemic bowel complications. After surgery, his platelet count was 118,000/mm³. Nine days later, his platelet count had fallen to 14,000/mm³, although the patient did not have any active bleeding despite his thrombocytopenia or the ongoing heparin therapy. The hematologist suspected a diagnosis of heparin-induced thrombocytopenia (HIT). A blood test for heparin-associated antibodies returned as positive, indicating he had developed an

antibody, or allergy, to heparin, and confirmed the diagnosis. Although Mr. Roberts had no obvious signs of clotting, upper and lower extremity Doppler studies were

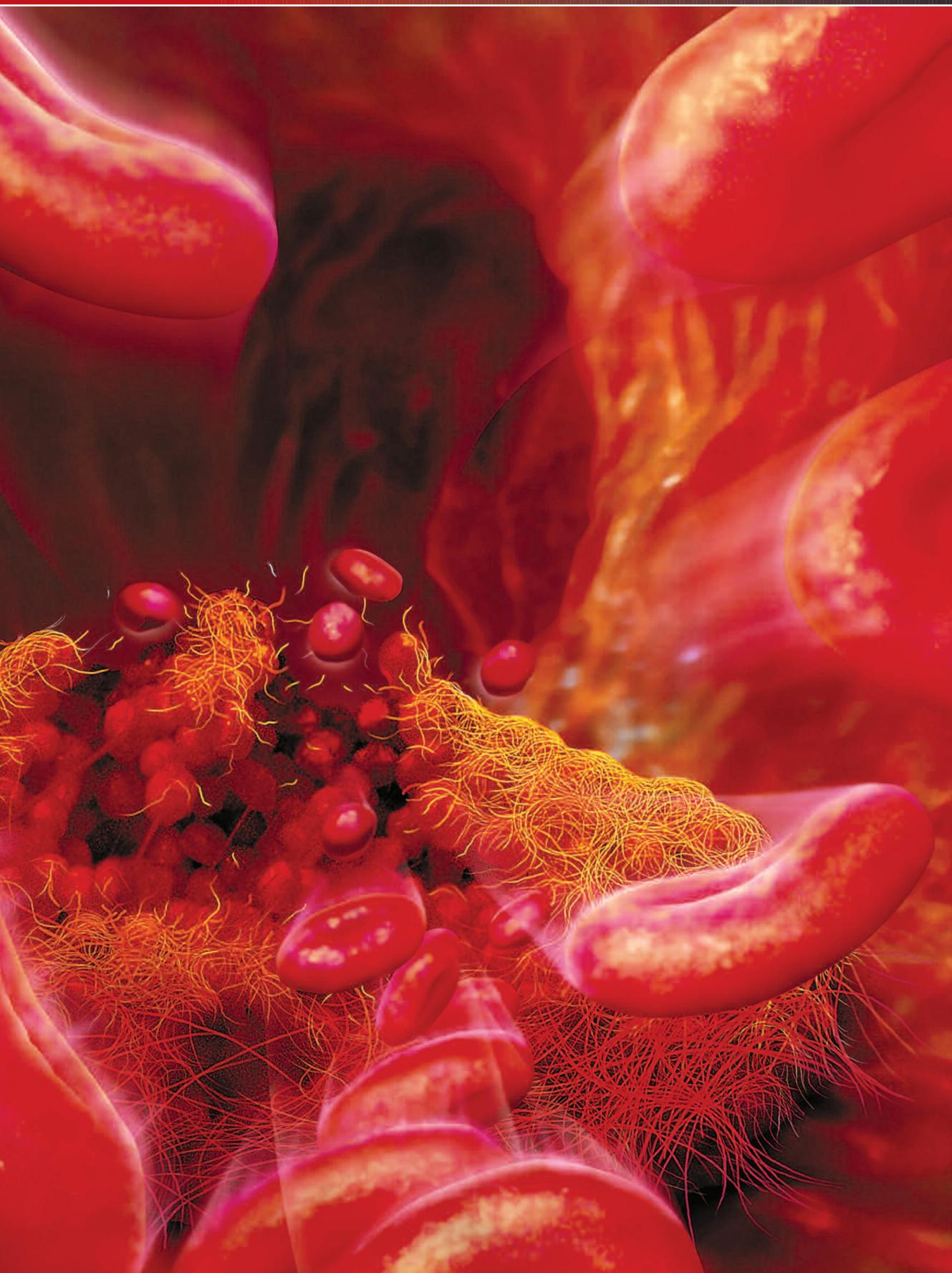
ordered to evaluate for possible occult thrombosis. These studies showed deep vein thrombosis (DVT) in both arms and the right leg. The heparin therapy was discontinued and a direct thrombin inhibitor (DTI) was started.

HIT's history

Heparin-induced thrombocytopenia occurs when the body develops an antibody, or allergy, to heparin. When this happens, instead of heparin acting as an anticoagulant, thrombocytopenia, and paradoxically, thrombosis, may develop. Heparin, frequently used in the critical care unit, is the medication most likely to cause thrombocytopenia.¹ In critical care patients, HIT may be an underlying, unrecognized condition



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with possible life and limb threatening consequences. It's important for the critical care nurse to become knowledgeable about this potentially catastrophic phenomenon as the ability to assess for and recognize HIT are critical keys to exemplary patient outcomes.

Heparin has been important in the treatment of thrombosis since the 1950s. By 1958, reports began to emerge of patients experiencing multiple emboli after starting heparin therapy. After this had been observed in several patients, it was concluded there was a relationship between the use of heparin and the thrombotic complications. Often, such patients either required multiple surgeries to remove emboli, suffered amputations, or died. This condition became known as the "white clot syndrome," since the thrombi were frequently noted to be pale, salmon-colored clots. In 1973, thrombocytopenia was recognized as an integral part of this syndrome and it became known as "Heparin-induced thrombocytopenia."²

Pathophysiology of HIT

When there is vascular injury in response to trauma, surgery, cardiopulmonary bypass, or inflammation, platelets become activated and release various components as a part of the hemostasis process.^{3,4} One of these components is platelet factor 4 (PF4). If the patient is receiving heparin (H), heparin molecules may bind to PF4 resulting in PF4-H complexes. Some patients develop antibodies to PF4-H. The antibody may attach to the PF4-H creating a new complex.³ This PF4-H-antibody complex may

bind to the surface of platelets and cause further, intense platelet activation and the release of procoagulant-rich platelet microparticles.⁵ This, in turn, may activate the blood coagulation system, generating thrombin and causing clot formation.^{4,6} The combination of platelet activation and removal of circulating antibody-coated platelets typically results in the thrombocytopenia characteristic of HIT. It is, however, possible for thrombosis to be present without thrombocytopenia. (See [Pathophysiology of HIT](#).)³

Clinical features of HIT

The clinical features of HIT are thrombocytopenia, a decreased platelet count, and possible thrombosis occurring after heparin therapy. Any type, route, or amount of heparin can trigger HIT, however, UFH triggers HIT more often than low-molecular-weight heparin (LMWH).⁷

Thrombocytopenia: In the context of HIT, thrombocytopenia is defined as a fall in the platelet count of more than 50% from the patient's preheparin baseline. It's important to recognize that if the initial platelet count is high; such a change may not result in true thrombocytopenia since the value may remain in the "normal" range. This underscores the importance of regularly monitoring the platelet counts during and after heparin therapy. The fall in platelet count typically starts 5 to 14 days after the initial heparin exposure.⁸ Any fall in platelets in conjunction with heparin therapy should raise suspicions of HIT. Although the platelet count may be very low, HIT patients generally

do not bleed unless other complications, such as sepsis, are also present.⁹

Thrombosis: Thrombotic events may be venous, (pulmonary embolus [PE], DVT) or arterial (occlusion of lower limb arteries, stroke, and myocardial infarction). Some patients may experience a "prothrombotic storm" with multiple thromboses such as DVT, PE, stroke, etc.⁸ Alternatively, thrombosis in the patient with HIT may be asymptomatic and only discovered when assessment tests such as a Doppler ultrasound are performed. In general, HIT should be suspected when any new, progressive, or recurring thrombosis develops during or soon after heparin therapy, regardless of the platelet count.⁷

Other clinical symptoms:

Skin lesions at subcutaneous injection sites and systemic reactions when an intravenous (I.V.) heparin infusion is started may also occur.¹⁰

Patient population and type of heparin

The patient population and type of heparin plays a role in the frequency of HIT. Approximately 50% of cardiac surgery patients who receive UFH develop the PF4-H antibody, although probably less than 1% develop clinical HIT.¹¹ In contrast, only 15% of orthopedic surgical patients who receive postoperative prophylaxis with UFH develop the antibody, but 5% develop thrombosis.¹⁰ The move to LMWH for orthopedic surgery has reduced the incidence of HIT to less than 1%.⁷ There's a very low risk of HIT in obstetrical and medical patients who receive LMWH.¹⁰

Transient nature of antibodies

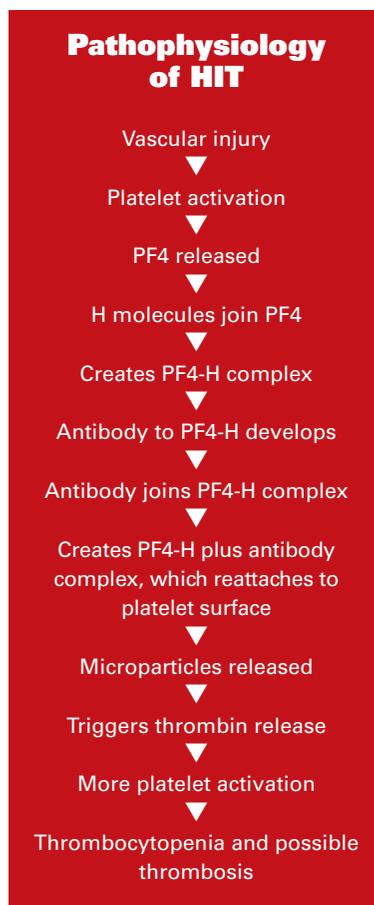
Heparin antibodies tend to be transient and, in most cases, will eventually become undetectable. Typically, 60% to 90% of patients will test negative for the heparin antibody after 100 days, although it may be detectable in some individuals for up to 6 months or longer.^{9,12,13}

Presentations of HIT

There are three known distinct presentations of HIT. In **classic HIT**, the patient develops thrombocytopenia 5 to 14 days after beginning heparin therapy. If there's a history of previous heparin exposure in these patients, it would most likely be a "remote" history defined as more than 100 days prior.⁹

Rapid onset HIT is characterized by a fall in the platelet count shortly (within 1 to 3 days) after heparin is started. These patients generally have received heparin in the recent past, and have a preexisting circulating PF4-H antibody. In such cases, reexposure of heparin rapidly causes thrombocytopenia and possibly thrombosis, without the typical delay needed to generate a new antibody.^{9,12}

Delayed onset HIT begins days or weeks after the discontinuation of heparin therapy. It's possible that some cases represent a delayed clinical recognition of HIT, although most patients appear to have a truly delayed onset of the condition.⁹ The recognition and prompt treatment of HIT is essential in both instances. Typically, a patient receives heparin and is discharged after an uneventful hospital course. After a period rang-



ing from several days to a few weeks, the patient develops symptoms of thrombosis and returns to the hospital, frequently presenting to the emergency department (ED).¹⁴ In these cases, if the possibility of delayed onset HIT isn't considered, and the patient is reexposed to heparin, his or her condition will be significantly exacerbated by the anticoagulant treatment.

Diagnostic tests

Heparin-induced thrombocytopenia is a clinicopathologic syndrome which implies that both the clinical findings and laboratory test results are crucial components in the diagnosis.¹⁵ Nevertheless, treatment of the condi-

tion should not await the return of laboratory test results.

Platelet counts: The key to the recognition and diagnosis of HIT is the platelet count. Knowing the baseline preheparin count is critical, as this is the value future platelet counts will be compared to for assessing the percentage of platelet fall. Patients who are at the highest risk for developing HIT, such as those receiving UFH, should have platelet counts at least every other day. Patients receiving LMWH, which has less risk of HIT, can be monitored 2 to 3 times a week.⁷

Specific tests for the PF4-H antibody: Laboratory confirmation of the diagnosis of HIT rests with the demonstration of a circulating antibody to the PF4-H complex. Commonly used tests include platelet aggregation (PA), the serotonin release assay (SRA), and the enzyme-linked immunosorbent assay (ELISA). The PA and SRA tests are both activation (or functional) tests, which rely on the ability of the PF4-H antibody to activate platelets.^{15,16} Both methods require the laboratory to obtain normal donor platelets for the test, and are quite technically demanding. The SRA also uses radioisotopes and isn't offered in most laboratories. The ELISA tests for the presence of PF4-H antibodies in the blood directly.^{15,16} This test is more sensitive for HIT antibodies than the SRA, but is less specific for clinical HIT.⁷ The test is offered by more laboratories as it is easier to perform than the SRA. It's important to be aware that a patient may test positive for the presence of the heparin antibody without having clinical signs of HIT. This

is particularly true of patients recovering from cardiovascular surgery.

The “iceberg” theory

The fact that patients may have a circulating HIT antibody, without clinical HIT has led to the elaboration of the iceberg theory.^{7,9} In this analogy, patients with clinical HIT are usually obvious and may be compared to that part of the iceberg that is visible above the waterline. For cardiac surgery patients, these are the minority. A much larger proportion of patients may develop HIT with thrombocytopenia as their only symptom. Such individuals may or may not be recognized depending on the vigilance of their healthcare providers, and are represented by that portion of the iceberg just below the waterline. Far below the waterline however and generally unrecognized, are those patients that develop antibodies without any clinical symptoms attributable to HIT.

Managing HIT

Consider the various ways clinicians can manage HIT.

Stopping heparin: The ultimate goal of the management of HIT is to reduce the risk of thrombosis by reducing platelet activation and thrombin generation. The first line of treatment, when HIT is suspected, is to immediately stop all sources of heparin including “hidden” heparin and treat promptly with a DTI.¹ Sources of heparin clearly include therapeutic heparin, such as UFH, and LMWH, but should also include heparin flushes and heparin-coated invasive lines. As alternatives, DTIs may be used in place of UFH or LMWH, normal saline

(NS) flushes instead of heparin flushes, and heparin-coated invasive lines replaced with nonheparin coated lines.

It must be emphasized that in thrombocytopenic patients with HIT but no thrombosis, stopping heparin alone isn’t an adequate treatment; after cessation of heparin, there’s a 50% chance of a new thrombosis developing over the next month.⁷ Effective anticoagulation, usually in the form of a DTI, is required.

Alternative anticoagulation: Since HIT is accompanied by the formation of thrombin, the class of I.V. anticoagulants, known as DTIs, is a central component of the management of HIT. As their name implies, these agents bind directly with and inactivate thrombin.⁵ A DTI should be administered without delay when there is clinical suspicion of HIT and should not await the results of laboratory tests for HIT. The three DTIs currently available are lepirudin, argatroban, and bivalirudin. It’s important to note that there are no reversal agents for DTIs, analogous to the use of protamine sulfate for heparin. They do, however, have relatively short half-lives, and knowledge of these is important in their use and management.

Lepirudin (Refludan): Lepirudin is approved for treating HIT patients with thrombosis to prevent the development of further thromboembolism. It’s cleared through the kidneys and has a half-life of about 60 minutes. In patients with decreased renal function, significant dosage adjustment is required. Patients may develop antibodies to lepirudin, which may impair the clearance of the drug and rarely

cause a serious anaphylactic reaction.^{5,17}

Argatroban: Argatroban is approved for the prophylaxis and treatment of thrombosis in patients with HIT. It’s cleared through the liver and isn’t recommended for patients with liver impairment. Argatroban has a half-life of approximately 40 minutes, and because it’s derived from L-arginine, a natural amino acid, it does not appear to trigger antibody formation.¹⁸ Activated partial thromboplastin (aPTT) values return to baseline after 2 to 4 hours.¹⁹

Bivalirudin (Angiomax): Bivalirudin is approved for use in percutaneous coronary intervention (PCI) and also for HIT in PCI. Although not approved for prophylaxis or treatment of HIT, successful “off-label” use in these settings has been reported.^{20,21} It’s metabolized mainly in the plasma (80%) with the remainder (20%) through the kidneys. Some dosage adjustment is needed in patients with renal dysfunction, but much less than lepirudin. Bivalirudin has a very short half-life of 25 minutes. This makes it useful for patients who might need anticoagulation stopped for invasive procedures or who have a high risk of bleeding. Bivalirudin is a synthetic derivative of hirudin. Even though it does not seem to trigger the development of antibodies, if a patient has had lepirudin in the past, it may cross-react with antibodies that may have been formed from the previous lepirudin exposure.²¹

It’s important to be aware that the DTIs are monitored by the aPTT in a similar manner to heparin. However, they may also

affect the prothrombin time and the international normalized ratio (INR), which can complicate the transition to warfarin therapy.^{19,21}

Warfarin (Coumadin): The use of warfarin alone as a treatment for HIT is contraindicated. Due to the extreme hypercoagulable state in HIT, initial unopposed warfarin therapy can trigger worsening of venous thrombosis, venous limb gangrene and/or skin necrosis. Skin necrosis is caused by tiny thrombotic events in the central fatty areas of the body.²³ Therefore, warfarin needs to be teamed with DTI therapy. Warfarin may be slowly initiated after the patient's platelet count has recovered to at least 100,000/mm³ while on DTI therapy.¹⁹ The starting dose should be the same as a typical daily maintenance dose, and certainly

less than 5 mg. Loading doses (greater than 5 mg) should not be used. The DTI and warfarin should overlap by at least 5 days, until the INR has been therapeutic for 2 days.¹⁹ DTIs, especially argatroban, have a varying impact on the INR. Therefore, it's important to follow the manufacturer's therapeutic INR recommendation for the overlap of warfarin. Warfarin therapy should be continued for at least 2 to 4 weeks after initiation, due to the continued risk of HIT-associated thrombosis.⁵ In addition, long-term anticoagulation with warfarin may be needed for an ongoing condition. (See **Summary of HIT treatment.**)

Additional HIT management points

Inferior vena cava filters (IVCs) should be avoided, as they may

increase the risk of thrombosis in the inferior vena cava, DVT, and PE.^{12,24}

If HIT is ruled out

If HIT can be excluded after a thorough evaluation of clinical symptoms and laboratory testing, including repeat testing as indicated, heparin therapy may be reinitiated.¹

Case studies

The following three case studies, classic presentation, rapid onset, and percutaneous inserted central catheter (PICC) line with heparin flushes, illustrate common cases of HIT.

Case 1: Classic presentation

The case at the beginning of this article illustrates the classic characteristics of HIT. Mr. Roberts received UFH, which triggered thrombocytopenia 9 days after

Summary of HIT treatment

Step	Action	Comment
1	HIT suspected	Notify physician.
2	Stop heparin	Based on the prescriber's orders, stop all heparin, including flushes and drips, and assist in changing heparin coated invasive lines as needed.
3	Send laboratory samples	Treat patient as if they have HIT while waiting for results. Retesting may be needed.
4	DTI therapy—start and monitor	On physician orders—DTI should be started before lab results are back. Dosage adjustments are made based on the aPTT.
5	Watch for platelet count recovery	When platelet count is at least 100,000/mm ³ , dual anticoagulation therapy with warfarin can be instituted.
6	Initiate warfarin therapy	Initiate warfarin slowly at less than 5 mg, typical of the daily maintenance dose range. <i>Do not give a loading dose of greater than 5 mg.</i>
7	DTI/warfarin overlap	Overlap at least 5 days and until INR is therapeutic for 2 days.
8	INR therapeutic value	It varies with the DTI follow manufacture's recommended INR value.
9	Discontinue DTI	When the INR value has been therapeutic for 2 days.
10	Recheck INR	After DTI is discontinued, retest INR.
11	Continue warfarin	Continue up to 4 weeks or ongoing as clinical condition warrants (for example, atrial fibrillation or mechanical heart valve).

the start of heparin therapy. This timing put the thrombocytopenia in the typical 5 to 14 day window for HIT. Even though the platelet count fell to 14,000/mm³, there was no bleeding. Mr. Roberts had no obvious signs of clotting, however, the ultrasound testing was ordered due to the risk of thrombosis in HIT. This case illustrates the classic presentation of HIT.

However, the critical care nurse needs to be aware that HIT may occur in different presentations.

Case 2: Rapid onset HIT

Mr. Green had an uncomplicated postoperative course following coronary artery surgery and was discharged home. He was doing well for about 6 days when he noticed some right thigh and calf pain. His physician sent him to the ED, where an ultrasound exam indicated he had extensive right calf and thigh DVT. He was also found to have bilateral PE. His platelet count on admission was 400,000/mm³ and he was treated with UFH for the thrombosis. The next day his platelet count had fallen to 160,000/mm³ and a heparin antibody test was ordered. The heparin drip was immediately discontinued and a DTI started. The test returned positive, confirming a diagnosis of HIT. Following recovery of his platelet count, Mr. Green was transitioned to warfarin and was discharged home without further complication.

Mr. Green exhibited the hallmark signs of rapid-onset HIT. He had a recent history of cardiac surgery, which carries a high incidence of HIT antibody formation. His platelet count fell rapidly after reexposure to heparin, indicating the presence of a pre-existing circulating HIT antibody.

Case 3: PICC line with heparin flushes

Mr. Brown, 60, was brought to the ED with complaints of tenderness and swelling in the left upper arm. One month ago, Mr. Brown had been hospitalized for an infectious process and subsequently discharged to a skilled nursing facility to receive I.V. antibiotics via a left upper arm PICC line. Heparin flushes were used to maintain the patency of the PICC line. Mr. Brown was found to have a DVT in the left upper arm. LMWH 1 mg/kg subcutaneous twice a day was ordered. His platelet count on admission was 66,000/mm³. Due to the low platelet count and a history of heparin exposure, a heparin antibody blood test was ordered. The test returned positive. The LMWH was discontinued after two doses and a DTI started. When his platelet count had risen to 116,000/mm³, warfarin therapy was initiated. It was titrated until the INR was 2.38, at which point the DTI was discontinued. Mr. Brown was discharged home on warfarin and oral antibiotics.

Critical care patients frequently have PICC or other invasive lines with heparin flushes used to maintain line patency. This case highlights the possibility of the heparin flushes triggering HIT and the need to monitor these critical care patients closely.

The future of heparin and HIT

In general, patients with a history of HIT should receive an alternative anticoagulant when anticoagulation is needed in the future.⁵ However, for many cardiac and vascular surgical procedures,

UFH remains the drug of choice and no approved alternative agents are available. If a patient has a history of a positive heparin antibody, it's prudent to perform an appropriate test to determine whether the antibody is still present in the patient's circulating blood. It's recommended that a highly sensitive test, such as the ELISA, be performed for this purpose. If the test is now negative, it appears to be safe to briefly reexpose the patient to heparin, for example during cardiac surgery.^{1,9} However, if additional anticoagulation is needed, for example in the postoperative period, an alternate (nonheparin) anticoagulant is recommended.

Nursing considerations

Being knowledgeable about HIT and maintaining a high level of suspicion and vigilance for it are critical keys to the early recognition of HIT. Your role as the critical care nurse puts you on the frontline to observe for it. Key areas to be aware of are the patient's history, current heparin exposure, platelet count values, observing for signs and symptoms of thrombosis, what to do if HIT is suspected, and the nursing care of a patient with HIT.

HIT develops after exposure to heparin; therefore, assessing the patient's history of UFH and/or LMWH is essential. The patient may recall having subcutaneous injections of LMWH, however, he may be unaware of I.V. UFH administration. For this reason, it's important to take careful history of prior procedures that may involve heparin use, such as cardiac and/or vascular surgery, cardiac catheterization, and previous treatment for thrombosis. In

addition, make note of recent hospital admissions for any reason, as approximately 50% of all hospital inpatients are exposed to some form of heparin. The timing of possible previous exposure is important since the antibody is transient and may no longer be present. Therefore, patients with a more recent history have a higher risk of developing HIT than when the history is remote. In addition, be aware of any heparin sources during the patient's current admission.

As mentioned earlier, the baseline, preheparin platelet count is the value future platelet counts are compared with. For this reason, be sure the platelet count blood sample is drawn before heparin therapy is initiated.

Thrombosis may occur anywhere with signs and symptoms corresponding to the location. Thrombosis of an extremity includes discoloration, pain, tenderness, redness, swelling, and temperature changes in the extremity. Myocardial infarction, PE, thrombotic stroke, and ischemic bowel are also possible manifestations of HIT.

Remember with both platelet count changes and thrombosis development, the classic time frame for HIT is 5 to 14 days after initiation of heparin therapy. However, both rapid and delayed onset may also occur, so be suspicious of HIT if there has been any heparin exposure history. Regardless of the platelet count, suspect HIT if a new, progressive, or recurrent thrombosis develops during or soon after heparin therapy.^{5,7}

If you suspect your patient is developing HIT, notify the prescriber immediately, including

Patient education

All educational information should be presented in an age-appropriate manner. Teach the patient about the HIT disease process. Explain to him that he should consider this an allergy, and to inform his healthcare provider and medical caregivers at all subsequent hospital admissions. The patient may wish to wear an allergy bracelet or necklace.

Educate the patient about the signs and symptoms of clotting (for example, discoloration of fingers and toes, new chest pain), especially when different than cardiac pain, which could signal a PE. Pain, redness, and swelling in an extremity, and neurologic symptoms, such as slurred speech, numbness, tingling of extremities, or paralysis, may also be significant for the development of HIT. The patient should seek medical attention immediately if he experiences any of these symptoms.¹⁶

information on current or recent heparin exposure. On the physician's order, discontinue all heparin products. Assist the prescriber in changing heparin-coated indwelling line. Note heparin as an allergy on the arm bracelet and medical record, and notify the pharmacist of a possible diagnosis of HIT. You might consider the use of a "no heparin products" sign over the patient's bed. Do not massage or rub an extremity where a thrombosis is suspected.

DTI's are powerful anticoagulants and should be monitored carefully after their initiation. Follow all rules of accurate medication administration, for example, correct identification of patient and drug, verification of dose, route of administration and timing. Verify the medication with a colleague before administration and dose change. Ensure that infusion pumps are functioning accurately, and be prepared for frequent medication changes based on aPTT laboratory results. Accurately document all dosage starts, changes, holds, and stops of anticoagulant therapy. Bleeding is a major concern with all anticoagulation medications,

therefore, observe the patient for any signs or symptoms of bleeding, such as hematuria, hemoptysis, or gastrointestinal bleeding, and report any observed bleeding to the physician immediately.¹⁹ (See [Patient education](#).)

Prevention

Prevention efforts center on reducing exposure to heparin. This can be accomplished by the routine use of NS instead of heparin to flush invasive lines, and the use of a DTI during PCI procedures.¹² The use of LMWH whenever possible instead of UFH is also helpful, as there is a reduced incidence of HIT with LMWH.⁶ HIT education for healthcare providers plays a significant role in prevention. In addition, catastrophes may be prevented by maintaining a high level of suspicion and acting promptly when HIT is suspected.⁸

The keys to improved outcomes in patients with HIT are early recognition and prompt and appropriate management. Critical care patients will benefit when the critical care nurse is knowledgeable and maintains a high level of suspicion and vigilance for HIT. ❖

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Managing the patient with HIT

GENERAL PURPOSE: To provide the registered professional nurse with an overview of the diagnosis and management of heparin-induced thrombocytopenia (HIT).
LEARNING OBJECTIVES: After reading this article and taking this test, you should be able to: 1. Discuss the pathophysiology of HIT. 2. Describe the diagnosis and management of HIT.

1. What causes heparin-induced thrombocytopenia (HIT)?

- a. The heparin dose is too high.
- b. Heparin products have been used long term.
- c. The patient is allergic to heparin.
- d. The platelet count falls below the normal range following heparin administration.

2. Typically, HIT results from the

- a. combination of platelet activation and removal of circulating antibody-coated platelets.
- b. combination of platelet deactivation and removal of circulating antibodies.
- c. inability of heparin to bind to receptor sites.
- d. inability of the heparin molecule to bind to platelet factor 4 (PF4).

3. Heparin-induced thrombocytopenia is defined as a fall of the platelet count

- a. below 200,000/mm³.
- b. by less than 20% from baseline.
- c. by 20% from baseline.
- d. by more than 50% from baseline.

4. After exposure to heparin, the platelet count in HIT starts to fall in

- a. 5 to 14 days.
- b. 15 to 20 days.
- c. 21 to 30 days.
- d. more than 30 days.

5. HIT patients with low platelet counts typically

- a. hemorrhage.
- b. bleed only if complications are present.
- c. do not form thrombi.
- d. develop a generalized malaise.

6. Heparin antibodies usually remain present in most HIT patients for about

- a. 3 months.
- b. 6 months.
- c. 9 months.
- d. 1 year.

7. Patients who have rapid onset HIT usually

- a. start decreasing platelets after 5 days of heparin.

- b. do not test positive for the PF4-heparin antibody.
- c. have received heparin in the recent past.
- d. develop symptoms after heparin is stopped.

8. Which marker is the key to the recognition of HIT?

- a. platelet count
- b. thrombus formation
- c. international normalized ratio (INR) value
- d. activated partial thromboplastin time (aPTT) value

9. Which population of patients is most likely to develop PF4-heparin antibodies?

- a. orthopedic patients
- b. cardiac surgery patients
- c. obstetric patients
- d. low-molecular-weight heparin (LMWH) recipients

10. The first-line treatment when HIT is suspected is to

- a. begin warfarin with hourly lab monitoring of aPTT.
- b. continue the heparin and treat with direct thrombin inhibitors (DTIs).
- c. stop the heparin and substitute warfarin.
- d. stop the heparin and treat with DTIs.

11. The effects of DTIs can be reversed by administering

- a. protamine sulfate.
- b. vitamin K.
- c. bivalirudin.
- d. No reversal agent exists.

12. Which agent is cleared by the liver and should be used cautiously in patients with liver disease?

- a. heparin
- b. bivalirudin
- c. argatroban
- d. lepirudin

13. Which drug should not be used alone for the treatment of HIT?

- a. warfarin
- b. heparin

- c. lepirudin
- d. bivalirudin

14. Which statement is true about treating HIT patients?

- a. Inferior vena cava filters are used to provide protection against pulmonary embolus.
- b. Direct thrombin inhibitors will not affect the patient's INR and prothrombin time values.
- c. Direct thrombin inhibitors have short half-lives ranging from 90 to 120 minutes.
- d. The goal of treatment is to reduce the risk of thrombosis.

15. Which of the following is *least* likely to cause HIT?

- a. LMWH
- b. I.V. infusion of heparin
- c. heparin flushes
- d. subcutaneous heparin

16. Currently, which anticoagulant is the drug of choice for vascular procedures?

- a. LMWH
- b. unfractionated heparin
- c. warfarin
- d. DTI

17. Which is a key nursing action when caring for critical care patients?

- a. Obtain a history of the patient's prior exposure to heparin.
- b. Obtain informed consent for treatment with heparin.
- c. Draw a baseline platelet count within 1 hour following the first heparin dose.
- d. Monitor the patient for thrombosis formation on days 14 through 18.

18. Decrease the risk of harm to your patients from HIT by all of the following *except*

- a. using saline flushes.
- b. using nonheparin-coated invasive lines.
- c. using unfractionated heparin for thrombosis prophylaxis
- d. monitoring and trending frequent platelet counts.



ENROLLMENT FORM Nursing2007 Critical Care, November, Managing the patient with HIT

A. Registration Information:

Last name _____ First name _____ MI _____
Address _____
City _____ State _____ ZIP _____
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LPN RN CNS NP CRNA CNM other _____
Job title _____ Specialty _____
Type of facility _____ Are you certified? Yes No
Certified by _____
State of license (1) _____ License # _____
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Registration Deadline: December 31, 2009

Contact hours: 2.5 Pharmacology hours: 0.0 Fee: \$22.95

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B. Test Answers: Darken one circle for your answer to each question.

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C. Course Evaluation*

- 1. Did this CE activity's learning objectives relate to its general purpose? Yes No
- 2. Was the journal home study format an effective way to present the material? Yes No
- 3. Was the content relevant to your nursing practice? Yes No
- 4. How long did it take you to complete this CE activity? ___ hours ___ minutes
- 5. Suggestion for future topics _____

D. Two Easy Ways to Pay:

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