International Surgery Journal
Lopez J et al. Int Surg J. 2026 Oct;13(10):1991-1996
https://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20263358

Case Report

A case of heparin induced thrombocytopenia requiring an open arterial thrombectomy

Jose Lopez[1]*, Patrick Kiarie[2], Bhakti Patel[3], Christina Montesano[3], Christopher Dachowski[4], Martine Louis[1], Manikyam Mutyala[1]

[1]General Surgery, Flushing Hospital Medical Center, Flushing, New York, United States
[2]St. George's University, School of Medicine, Grenada, West Indies
[3]New York Institute of Technology College of Osteopathic Medicine, Old Westbury, New York, United States
[4]Ross University School of Medicine, Barbados

Received: 19 August 2026
Accepted: 16 September2026

***Correspondence:**
Dr. Jose Lopez,
E-mail: jlopez2887@gmail.com

ABSTRACT

Heparin-induced thrombocytopenia (HIT) is an immune-mediated complication of heparin exposure characterized by thrombocytopenia and a paradoxical prothrombotic state. Although venous thrombosis is more common, arterial thrombosis can result in severe limb-threatening ischemia. A 69-year-old man admitted with new-onset atrial fibrillation and acute systolic heart failure received unfractionated heparin followed by enoxaparin. He subsequently developed a marked decline in platelet count and acute bilateral lower-extremity ischemia. Computed tomography angiography demonstrated extensive thrombi involving the abdominal aorta and bilateral iliac arteries, while venous duplex demonstrated no venous thrombosis. HIT was confirmed by positive anti-PF4 antibodies and serotonin release assay. All heparin products were discontinued and argatroban was initiated. Progressive limb ischemia required catheter-directed EKOS thrombolysis followed by open right common femoral artery thrombectomy, resulting in restoration of lower-extremity perfusion and avoidance of amputation. This presentation was particularly challenging because of concomitant atrial fibrillation and peripheral arterial disease, which provided an alternative explanation for the arterial thrombosis. HIT should be considered in patients with recent heparin exposure who develop thrombocytopenia and unexplained arterial thrombosis, even in the absence of venous thrombosis. Prompt heparin cessation, alternative anticoagulation, and multidisciplinary endovascular and surgical intervention can achieve successful limb salvage.

Keywords: Heparin-induced thrombocytopenia, HIT, Acute limb ischemia, Arterial thrombosis, Catheter-directed thrombolysis, Thrombectomy

INTRODUCTION

Heparin-induced thrombocytopenia (HIT) is an antibody-mediated adverse reaction to heparin, occurring in approximately 5% of patients exposed to heparin products. This reaction typically develops 5–14 days after initial exposure to heparin. Patients with HIT form auto-antibodies to the platelet factor 4 (PF4)/heparin complex, which binds to and activates platelets, creating a prothrombotic state.[1] Most patients who experience thromboses present either before or on the same day that platelet counts decrease by more than 50% within 24 hours.[2] Symptoms include life-threatening thrombocytopenia and an increased risk of thrombosis, with thromboembolic events in 55% of patients, with 25% experiencing at least three distinct thrombotic events.[2,3] Venous thrombosis is predominant, occurring in 90–97% of cases, with pulmonary embolism in 40% of

patients.[2,4] Arterial thrombosis in HIT is rare, primarily affects limb arteries, followed by stroke and then myocardial infarction (MI), in contrast to the typical order of atherothrombosis unrelated to HIT.[2] Given the high risk of thrombus formation, HIT is associated with a mortality rate of approximately 16%, underscoring the importance of early recognition and intervention.[5] Here, we report the case of a 69-year-old male with a severe and unusual presentation of HIT complicated by arterial thrombosis.

CASE REPORT

A 69-year-old Caucasian male with a past medical history of psoriasis presented to the emergency department (ED) with two weeks of worsening shortness of breath and chest tightness. His history was also notable for a 40-pack-year smoking history. Physical examination revealed tachycardia with an irregularly irregular rhythm, a right-sided pleural rub, decreased breath sounds, bilateral lower leg edema, and a scaly erythematous rash. Laboratory studies showed a hemoglobin level of 12.8, platelet count of 321, BUN of 27, and potassium of 5.3. Troponins were negative, but BNP and D-dimer levels were markedly elevated at 5,920 and 2,717, respectively. An EKG showed atrial fibrillation with a right bundle branch block and right axis deviation. A chest X-ray (CXR) revealed moderate right pleural effusion with underlying atelectasis. The patient was started on diltiazem, Lasix, and nebulizers.

The patient was admitted to the coronary care unit (CCU) with a diagnosis of new-onset atrial fibrillation and acute systolic heart failure (HF). A heparin drip was initiated for 48 hours. A chest CT scan ruled out pulmonary embolism but confirmed right heart dysfunction, a large right-sided pleural effusion, and right lower lobe atelectasis. Surgery was consulted, and a chest tube was inserted, draining 2 liters of exudative fluid as per Light's criteria. Cytology and cultures of the fluid were negative.

By hospital day 4, the patient remained tachycardic (116-130 BPM) with an irregular rhythm despite diltiazem therapy. A transthoracic echocardiogram (TTE) revealed a reduced ejection fraction (EF) of 20-25% with severe global dysfunction. Diltiazem was discontinued, and oral metoprolol tartrate and amiodarone were initiated. Cardiac catheterization was planned once the patient was stable and euvolemic. On hospital day 6, follow-up imaging showed improved pleural effusion but a new small pneumothorax. The patient was transitioned from a heparin drip to subcutaneous enoxaparin sodium for DVT prophylaxis.

On hospital day 8, the patient experienced worsening tachycardia (140s BPM) and hypotension (77/58 mmHg), and vasopressors were initiated. A repeat chest CT revealed a new peripheral right-sided effusion. Fluid cultures grew *Streptococcus dysgalactiae*, prompting a switch to ampicillin.

On day 13, the patient developed severe thrombocytopenia with a sharp decline in platelets from 219 to 56. HIT antibody testing was sent, enoxaparin was discontinued, and argatroban was started.

On day 15, the patient developed acute right lower extremity (RLE) ischemia with pain, numbness, cyanosis, and loss of pulses. A 4T score of 4 indicated possible HIT. Duplex ultrasound showed no venous thrombi; however, CTA confirmed multiple stenotic segments and thrombi in the abdominal aorta and iliac arteries bilaterally, with near-occlusion of the distal abdominal aorta just distal to the inferior mesenteric artery (Figure 1). Additionally, a CTA of the head and neck revealed a thrombus in the descending aorta, a potential source for the lower extremity emboli (Figure 2). The platelet count increased from 56 to 98.

On day 16, HIT antibodies came back positive. On day 18, the patient's right foot developed cyanotic toes although warm to touch, without palpable pulses, and no sensation of plantar surface. The left foot was also dusky without palpable pulses although with some sensation of plantar surface. The platelet count had normalized to 180. The serotonin release assay (SRA) came back positive, further confirming HIT. The chest tube was removed.

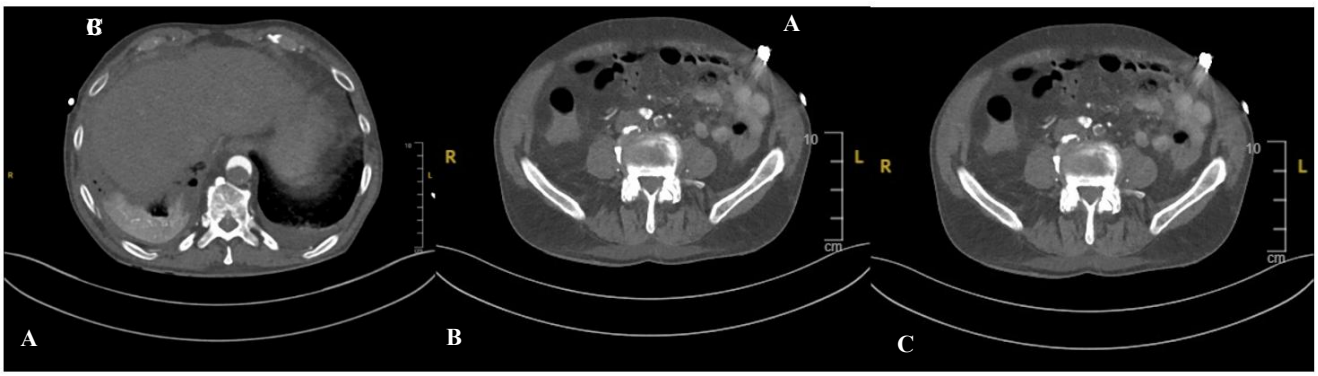

Figure 1: (A) CTA with contrast showing stenosis and thrombosis in the aorta at the level of the diaphragm, (B) near occlusion of the proximal right common iliac artery and (C) moderate to severe stenosis of the right popliteal artery.

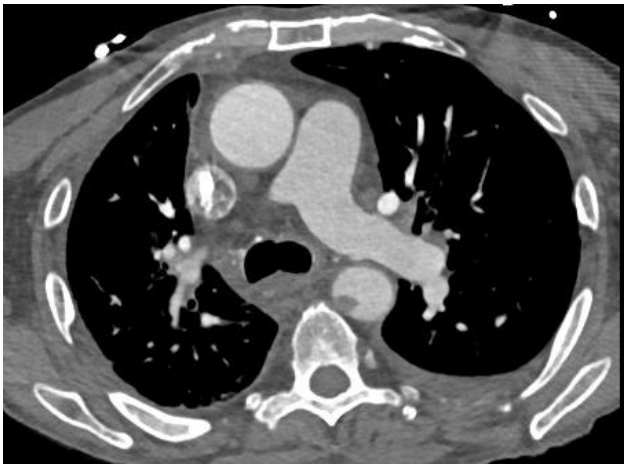

Figure 2: CTA of the head and neck showing a thrombus in the descending aorta.

On day 20, the discoloration worsened with the right fifth toe turning black, the left toes were dark red and vascular surgery was consulted. A preoperative nuclear stress test showing ischemic dilation with EF of 25%, placed the patient into the moderate risk group for surgery.

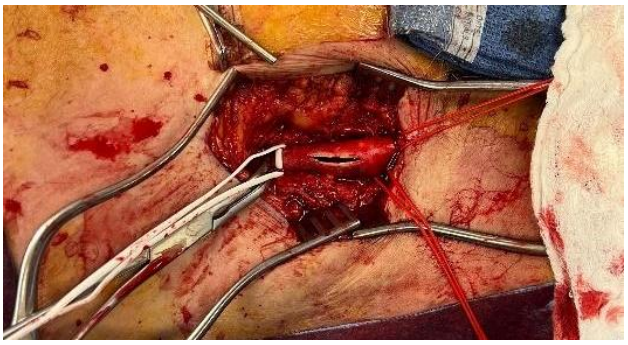

Figure 3: Incision made over the right common femoral artery. The skin, subcutaneous tissue and femoral sheath were cut along the line of incision. Common femoral artery, superficial femoral and profunda femoral arteries were exposed and vessel loops were placed.

Over the next few days, he required multiple procedures consisting of an aortoiliac and bilateral lower extremity angiograms, two endovascular thrombolysis (EKOS) procedures and lastly a right common femoral artery open thrombectomy. A 3 cm longitudinal incision was made in the right common femoral artery near the bifurcation. The skin, subcutaneous tissue, and femoral sheath were dissected along the line of incision, exposing the common femoral, superficial femoral, and profunda femoral arteries, and vessel loops were placed (Figure 3). Fogarty catheters were used for thrombectomy, removing multiple white and fibrinous clots (Figure 4).

A completion angiogram showed restored antegrade and retrograde flow. The patient tolerated the procedure well and was sent to the recovery room in stable condition. Postoperatively, all lower extremity pulses were restored except for the left popliteal pulse. Left leg color and capillary refill were normal.

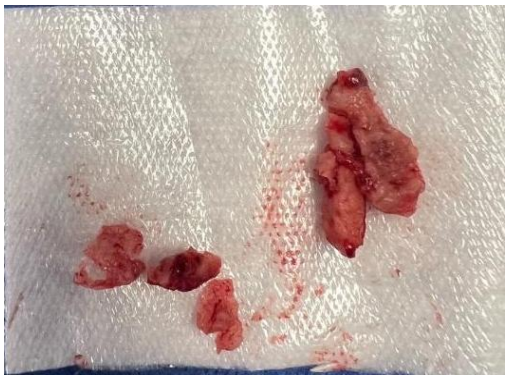

Figure 4: The clots removed from the superficial and Profunda femoral artery.

DISCUSSION

Heparin is a widely used anticoagulant but, in rare cases, it causes heparin-induced thrombocytopenia (HIT), paradoxically resulting in a prothrombotic state. HIT is classified into two forms, both involving a decrease in platelets. Type 1 HIT is a non-autoimmune reaction resulting in a mild decrease in platelets that resolves without treatment and often without discontinuing heparin.[1,2] This occurs in up to 10% of heparin users.[1,2] In contrast, type 2 HIT is an antibody-mediated adverse reaction that occurs in approximately 5% of those exposed to heparin products.[1] Unfractionated heparin is much more likely to cause HIT than low-molecular-weight heparin.[1,2]

Type 2 HIT typically occurs 5-14 days after initial heparin exposure, during which patients form auto-antibodies to the PF4/heparin complex. These antibodies bind to and activate platelets, creating a prothrombotic state.[1,6] Signs and symptoms include life-threatening thrombocytopenia and an increased risk of thrombosis.[3] Most patients who experience thromboses present either before or on the same day as a >50% decrease in platelet count within 24 hours.[2] Thromboembolic complications occur in 55% of HIT cases, with 25% of patients developing thromboses at three or more sites.[2] Venous thrombosis is more common than arterial thrombosis in HIT (90-97%), with pulmonary embolism occurring in 40% of cases.[2,4]

Arterial thrombosis in HIT primarily affects limb arteries, followed by cerebral and coronary arteries, which is the opposite pattern of non-HIT related atherothrombosis.[2] Acute arterial thromboembolism typically presents with sudden onset of paresthesia, paralysis, absent distal pulses, cold and cyanotic extremities, severe pain and absent pulses distal to arterial blockage. Arterial thrombi have also been reported in less familiar locations, such as

the renal and mesenteric arteries.[1,3] Our patient presented with acute lower extremity ischemia due to arterial thrombosis, which may require amputation approximately 33% of the time.[1,3] With thrombus formation, mortality rates from patients treated for HIT are as high as 16.2%, emphasizing the importance of early recognition and intervention.[5]

HIT is diagnosed clinically and confirmed through serum anti-PF4 antibody testing. Clinical suspicion for HIT is raised by several factors, including a platelet count decrease of 30% to below 100, a >50% decrease in platelet count from baseline, thrombocytopenia occurring 5-10 days after heparin initiation, unexplained thrombocytopenia, and normalization of platelet levels upon discontinuation of heparin.[7] While awaiting antibody testing, the 4T score, an adapted clinical tool developed by Lo et al can help quantify the likelihood of HIT, with scores categorized as low (≤3), intermediate (4-5), and high (6-8).[8,9] A score of 0-3 effectively rules out HIT in most cases.[10] In this patient, the 4T score was 4, indicating an intermediate likelihood of HIT with a 10% probability, necessitating further evaluation. While functional assays are the gold standard for HIT diagnosis due to their higher specificity, false negatives can occur in the presence of antiplatelet drugs.[10] Thus, the most effective approach combines multiple tests for increased accuracy.[7] Notably, a negative functional assay does not definitively rule out HIT.[10] There is a subtype of autoimmune HIT characterized by platelet activation with a serotonin release assay, where anti-platelet factor 4 antibodies result in serotonin release from platelets without adding heparin.[11]

The majority of patients present with venous thrombotic events.[11] Our patient's presentation with negative venous Doppler studies, was atypical for HIT, but the distal limb ischemia, platelet count drop, and 4T score pointed toward arterial HIT, confirmed by CT angiogram and ELISA. Interestingly, the patient's symptoms appeared two days after the initial 50% platelet drop, whereas typical HIT symptoms present before or on the same day as the decrease. Prolonged heparin therapy (>5 days) is a significant risk factor for HIT, and this was the case for our patient, who had been anticoagulated with heparin for 11 days.[12] Women are at twice the risk of developing HIT compared to men due to heightened immune responses.[12] Surgical/trauma patients have a significantly higher risk of HIT (1-5%) compared to medical or ICU patients (<1%), placing this patient, a male medical patient, in a lower-risk category.[12]

The diagnosis was further complicated by two additional considerations in the context of his acute limb ischemia: peripheral arterial embolism due to new-onset atrial fibrillation and critical limb ischemia secondary to severe peripheral vascular disease (PVD). Atrial fibrillation increases the risk of embolic events, as supported by imaging showing multiple thrombi in the aorta, iliac arteries, and lower extremities (Figure 2). Additionally, the patient's extensive smoking history and imaging findings, such as near-occlusion of the distal abdominal aorta (Figure 1), suggested underlying atherosclerosis and PVD.

If HIT is suspected or if the 4T score is >3, management begins with immediate cessation of heparin products and initiation of alternative anticoagulation, such as direct thrombin inhibitors (argatroban, bivalirudin) or indirect factor Xa inhibitors (danaparoid, fondaparinux).[10] In this case, enoxaparin was discontinued, and the patient was started on argatroban. Direct oral anticoagulants are gaining traction, but data about their comparative effectiveness remains limited.[10] Counseling on future heparin avoidance is critical, as prior HIT episodes increase the risk of recurrence upon re-exposure. Additionally, second-line treatments, such as plasmapheresis, IVIG, cangrelor, and imlifidase, are reserved for specific cases, and plasmapheresis is indicated for patients requiring emergent cardiovascular surgery with heparin anticoagulation or in refractory HIT.[10] Intravenous immunoglobulin (IVIG), while less commonly used, is beneficial in refractory HIT due to its inhibition of heparin-independent platelet-activating antibodies.[10] Platelet transfusions, despite thrombocytopenia, are contraindicated due to an increased risk of thrombosis.[13]

COVID-19 is a well-known predisposition to arterial and venous thromboembolic events, and thus, anticoagulation therapy is often administered to reduce the risk of clot formation. A proposed mechanism is the aberrant immune activation secondary to infection causing pathological IgG antibodies to heparin and platelet factor 4. Shiuan et al describe a case of spontaneous HIT complicated by multiple bilateral lower extremity arterial occlusions presenting as adult limb ischemia.[11] Additionally, an 86-year-old male receiving prophylactic heparin developed multiple pulmonary arterial emboli, a significant drop in platelet count, and a 4T score of 6. This illustrates the need for careful monitoring of patients on anticoagulation, particularly in the setting of COVID-19, where both the viral infection and anticoagulation therapy increase the risk of thromboembolic events.[14]

Malignancy is another well-known cause of hypercoagulability and, consequently, prothrombotic events, due to the release of procoagulant substances by tumor cells, interactions with the fibrinolytic system, and tumor-mediated platelet activation.[15] The use of heparin products for anticoagulant patients with malignancy should be done with caution as they are susceptible to HIT-related complications including arterial thrombi. Hirashita et al reported a patient undergoing hepatic arterial infusion chemotherapy (HAIC) for hepatocellular carcinoma who developed a stroke from an arterial thrombus originating at the subclavian bifurcation. The catheter was removed, and the patient was initiated on anticoagulation therapy with heparin; however, five days later, he developed thrombocytopenia, yielding a 4T

score of 6, and positive autoantibodies against platelet factor 4.[16]

Catheter-directed thrombolytic therapy has been successfully used to treat and prevent complications associated with acute arterial thrombi in the extremities. An early study reported the successful outcomes and the limb-salvaging potential of thrombolysis in patients with HIT-associated arterial thrombosis, especially in patients at high risk for surgical procedures.[17] Another case reported successful catheter thrombolysis in a patient with acute lower extremity arterial thrombosis highlighting the clinical benefit of minimally invasive interventions in the setting of HIT.[18] Given the burden of clots in our patient, EKOS thrombolysis was deemed appropriate as an adjunct to anticoagulation therapy. The EkoSonic endovascular system combines catheter-directed thrombolysis with ultrasound to dissolve venous and arterial clots.[19] While studies show mixed results regarding its superiority, EKOS uses fewer thrombolytic agents and is faster, which is beneficial for patients with significant comorbidities that increase the risk of thrombolytic complications.[19]

CONCLUSION

The case presented highlights the complexity of diagnosis and management of arterial HIT. Although arterial thrombi secondary to HIT are less common, they pose a high risk of mortality if not recognized and treated early. It is crucial to consider HIT as a potential diagnosis and initiate treatment promptly when symptoms remain unexplained or do not fit the 4T criteria. This patient's case was particularly atypical due to the absence of venous thrombosis and confounding conditions such as new-onset atrial fibrillation and PVD. The use of the 4T score, confirmatory testing of anti-PF4 antibodies, and vascular imaging can overcome these complexities, allowing for timely diagnosis and intervention. Immediate cessation of heparin therapy, initiating alternative anticoagulation, using EKOS thrombolysis, and open thrombectomy successfully mitigated further complications, such as limb amputation for this patient. This case also highlights the importance of multidisciplinary management and tailored treatment strategies to overcome the complexity of the patient's comorbidities with the diagnosis of HIT, ultimately improving patient outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: None required

REFERENCES

1. Nicolas D, Nicolas S, Hodgens A, Reed M. Heparin-induced thrombocytopenia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2026. Available at: https://www.ncbi.nlm.nih.gov/books/NBK482330/. Accessed on 16 May 2023.
2. Greinacher A, Farner 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 Haemost. 2005;94(1):132-5.
3. Arepally GM, Padmanabhan A. Heparin-induced thrombocytopenia: a focus on thrombosis. Arterioscler Thromb Vasc Biol. 2021;41(1):141-52.
4. Linkins LA, Dans AL, Moores LK, Bona R, Davidson BL, Schulman S, et al. Treatment and prevention of heparin-induced thrombocytopenia: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e495S-e530S.
5. Ajitha S, Adla Jala SR, Verma R, Chennapragada SS, Pramudita AH, Dandwani M, et al. Trends in heparin-induced thrombocytopenia in the pre-pandemic and peak-pandemic era, and impact of COVID-19 on mortality: an analysis via the National Inpatient Sample. Blood. 2023;142(Suppl 1):5519.
6. Patriarcheas V, Pikoulas A, Kostis M, Charpidou A, Dimakakos E. Heparin-induced thrombocytopenia: pathophysiology, diagnosis and management. Cureus. 2020;12(3):e7385.
7. Ahmed I, Majeed A, Powell R. Heparin-induced thrombocytopenia: diagnosis and management update. Postgrad Med J. 2007;83(983):575-82.
8. Lo GK, Juhl D, Warkentin TE, Sigouin CS, Eichler P, Greinacher A. Evaluation of pretest clinical score (4 T's) for the diagnosis of heparin-induced thrombocytopenia in two clinical settings. J Thromb Haemost. 2006;4(4):759-65.
9. Crowther M, Pishko A. Management of heparin-induced thrombocytopenia. In: UpToDate. Waltham (MA): UpToDate; 2024. Available at: https://www.uptodate.com/contents/management-of-heparin-induced-thrombocytopenia. Accessed on 08 August 2024.
10. Marchetti M, Zermatten MG, Bertaggia Calderara D, Aliotta A, Alberio L. Heparin-induced thrombocytopenia: a review of new concepts in pathogenesis, diagnosis, and management. J Clin Med. 2021;10(4):683.
11. Shiuan E, Sharma D, Ely EW, Moodabagil N, Tillman BF. Limb ischemia due to spontaneous heparin-induced thrombocytopenia as the primary presentation of acute COVID-19 infection. J Thromb Thrombolysis. 2022;54(2):367-71.
12. Salter BS, Weiner MM, Trinh MA, Heller J, Evans AS, Adams DH, et al. Heparin-induced thrombocytopenia: a comprehensive clinical review. J Am Coll Cardiol. 2016;67(21):2519-32.
13. Gurbuz AT, Elliott WG, Zia AA. Heparin-induced thrombocytopenia in the cardiovascular patient: diagnostic and treatment guidelines. Eur J Cardiothorac Surg. 2005;27(1):138-49.
14. Zhang Y, Chen Z, Li J, Wang X, Liu Y. Heparin-induced thrombocytopenia during anticoagulation

therapy for COVID-19-related pulmonary embolism: a case report. Medicine (Baltimore). 2024;103(49):e40732.
15. Liu X, Huang L, Zhang X, Wu J. Blood hypercoagulability and thrombosis mechanisms in cancer patients: a brief review. Heliyon. 2024;10(19):e38831.
16. Hirashita K, Matsumoto A, Yabuno S, Kanda T, Yunoki M, Yoshino K. A case of posterior circulation ischemic stroke caused by heparin-induced thrombocytopenia after detaining hepatic arterial infusion catheter. J Stroke Cerebrovasc Dis. 2018;27(9):e196-200.
17. Calligaro KD, Kansagra A, Dougherty MJ, Savarese RP, DeLaurentis DA. Thrombolysis to treat arterial thrombotic complications of heparin-induced thrombocytopenia. Ann Vasc Surg. 1995;9(4):397-400.
18. Turba UC, Bozlar U, Simsek S. Catheter-directed thrombolysis of acute lower extremity arterial thrombosis in a patient with heparin-induced thrombocytopenia. Catheter Cardiovasc Interv. 2007;70(7):1046-50.
19. Cetingok Sr U, Akkoyun C, Isik Z, Gungor O. Treatment with EkoSonic™ Endovascular System (EKOS®) of massive pulmonary thrombosis following recovery from COVID-19 infection. Cureus. 2022;14(10):e30467.

Cite this article as: Lopez J, Kiarie P, Patel B, Montesano C, Dachowski C, Louis M et al. A case of heparin induced thrombocytopenia requiring an open arterial thrombectomy. Int Surg J 2026;13:1991-6.