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Short Report

Wound Necrosis and Peripheral Microangiopathy due to Delayed-onset Heparin-induced Thrombocytopenia Following Arterial Bypass Surgery

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ABSTRACT

Introduction: Delayed-onset heparin-induced thrombocytopenia (HIT) is a rare side effect of heparin. This prothrombotic condition can present its first signs up to three weeks following heparin administration even if heparin use has been stopped.
Report: A 54-year-old claudicant patient underwent a suprageniculate limb bypass with heparin administration. Despite bypass patency, our patient developed recurrent wound necrosis and kept complaining of ischemic pain. The patient then developed toe necrosis and underwent leg amputation.
Conclusion: Delayed-onset HIT is a rare condition that should be looked for and promptly managed in patients with peripheral vasculopathy following heparin administration.

Introduction

Delayed-onset heparin-induced thrombocytopenia (HIT) is a rare side effect of heparin. In contrast to classical HIT, this prothrombotic condition can present its first signs up to three weeks following heparin administration even if heparin use has been stopped.[1]

We report the case of a claudicant patient who underwent supra
onset
aggres
distal toe and leg necrosis.

Report

A 54-year-old man with a past history of diabetes mellitus type II and hypertension presented with bilateral peripheral arterial disease. He could not walk more than 200 m without calf pain (Fountain IIB), with a significant stenosis of the left superficial femoral artery and an extensive occlusion of the right superficial femoral artery. We performed a right suprageniculate femoro-popliteal vein bypass as well as a percutaneous angioplasty and stent implantation of the left superficial femoral artery. Unfractionated heparin 3 500 UI was given during surgery. The initial evolution was uneventful and the patient was discharged at day 7. Postoperative angiological examination showed improved ankle plethysmography of 55 mmHg on the right and 65 mmHg on the left (35 and 50 mmHg in the preoperative setting respectively). Three weeks following hospital discharge, the patient developed bilateral limb claudication with a hanging livedo. All surgical scars of the femoro-popliteal bypass were necrotic and the 3 first toes

workups were done and were normal. After a multidisciplinary approach, delayed-onset HIT was suspected based on high anti-PF4 antibodies (4 times threshold values) and, retrospectively, a platelet count showed a biphasic profile with a decrease of more than 30 percent. Heparin administration was stopped and replaced by fondaparinux. The patient mostly complained of pain in both feet and legs that was difficult to control with morphine derivatives, pregabalin (Lyrica®) and haloperidol (Haldol®), and then clonidine (Catapresan®) and duloxetine (Cymbalta®) were also tested with no benefit. Five months following initial surgery, the patient presented with increased pain that predominated on the right side, with a vascular workup that showed partial thrombosis in the abdominal aorta and both iliac arteries. The patient underwent therapeutic doses of fondaparinux and a transmetatarsian amputation was undertaken because of critical ischemia of the right fore-foot. During this procedure, the patient developed cardiac ischemia and underwent a coronography that showed a severe bitroncular

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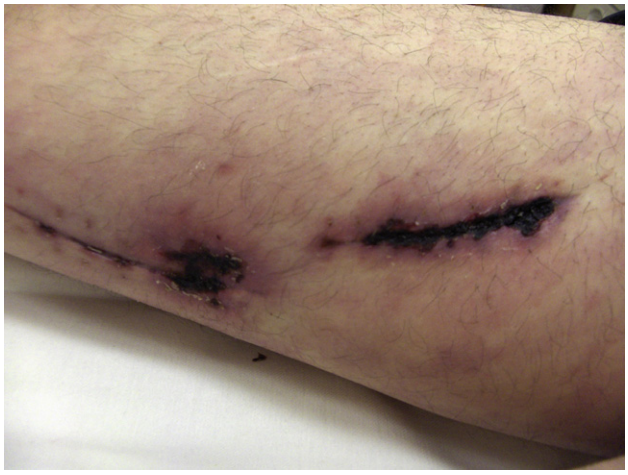

Figure 1. Wound necrosis at 9 weeks.

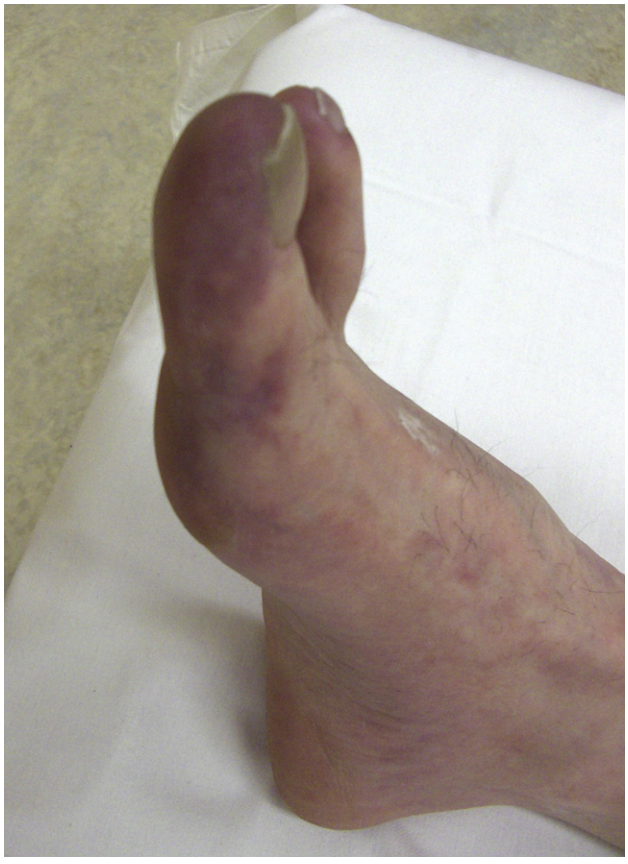

Figure 2. Fore-foot ischemia at 9 weeks with a hanging livedo.

disease treated by angioplasty. During the following weeks, the ischemic condition of the patient progressed. A Burgess amputation was performed on the right side because of critical ischemia progression and a transmetatarsian amputation on the left through wet necrosis of all toes.

Discussion

Delayed-onset HIT is a rare condition that shares some of the pathophysiology of the more classical HIT. However, unlike classical, which develops in 48 h, delayed-onset HIT becomes clinically detectable 9.2 days (range, 5–19 days) following heparin exposure. Most common complications are venous thromboembolism or thrombosis of arteries or arterial grafts.[2,3] Our patient presented a microangiopathy with partial thrombosis of the abdominal aorta and both iliac arteries and ischemic pain of the lower limbs. He also developed this condition much later after heparin administration. The outcome was dramatic with multi-organ vessel thrombosis. This atypical HIT presentation made the diagnosis difficult. Also, late recognition of delayed-onset HIT generated major morbidity with stump necrosis and major amputation. Our claudicant patient underwent major limb amputation 7 months after initial surgery because of atypical status and delayed diagnosis. Currently danaparoid is the recommended treatment of choice in situations of HIT. We used fondaparinux, which was easily available in our hospital and was an effective therapy, although rare cases of HIT during fondaparinux prophylaxis have been reported.[4,5] Our suspicion of delayed-onset HIT was based on the late onset of thrombosis, the positive anti-PF4 antibodies and the drop in platelet count.

Conclusion

Delayed-onset HIT is a rare condition which is difficult to diagnose and can cause dramatic outcomes. In this case report, a claudicant patient developed delayed-onset HIT after heparin exposition during bypass surgery, and developed severe ischemia in the limbs resulting in bilateral amputation and coronary disease. This syndrome should be part of the differential diagnosis of diffuse arteriopathy in all patients that have been exposed to heparin in previous weeks. Anti-coagulation therapy should begin with fondaparinux or danaparoid. Despite recognition, adequate therapies are very limited with a high risk of death, of about 20 percent.[2,3]

Conflict of Interest/Funding

None.

References

1 Cooney MF. Heparin-induced thrombocytopenia: advances in diagnosis and treatment. *Critical Care Nurse* 2006;**26**(6):30–6. quiz 7. Epub 2006/11/25.
2 Rice L, Attisha WK, Drexler A, Francis JL. Delayed-onset heparin-induced thrombocytopenia. *Annals of Internal Medicine* 2002;**136**(3):210–5. Epub 2002/02/06.
3 Warkentin TE, Kelton JG. Delayed-onset heparin-induced thrombocytopenia and thrombosis. *Annals of Internal Medicine* 2001;**135**(7):502–6. Epub 2001/10/02.
4 Modi C, Satani D, Cervellione KL, Cervantes J, Gintautas J. Delayed-onset heparin-induced thrombocytopenia type-2 during fondiparinux (Arixtra) therapy. *Proceedings of the Western Pharmacology Society* 2009;**52**:5–7. Epub 2009/01/01.
5 Linkins LA, Warkentin TE. Heparin-induced thrombocytopenia: real-world issues. *Seminars in Thrombosis and Hemostasis* 2011;**37**(6):653–63. Epub 2011/11/22.