ELSEVIER

J. of Ambulatory Surgery 11 (2004) 47–48

Ambulatory Surgery

www.elsevier.com/locate/ambsur

Short communication

Heparin-induced CVA

R. Dattani*, H. Reece Smith

Department of Surgery, Royal Berkshire Hospital, London Road, Reading RG1 5AN, UK

Received 13 December 2003; accepted 13 January 2004
Available online 19 March 2004

Abstract

Surgeons commonly use heparin as prophylaxis against post-operative venous thromboembolism. Heparin-induced thrombocytopenia and thrombosis syndrome (HITTS) is a rare but potentially fatal complication of heparin therapy. We describe a case of HITTS in a 49-year-old woman, after elective cholecystectomy, which resulted in a CVA. The purpose of this case report is to increase the awareness of this phenomenon among health care professionals involved in day-care surgery and to discuss its management.

Keywords: Heparin; Thrombocytopenia; Thrombosis; HITTS; CVA

1. Introduction

Surgeons commonly use heparin as prophylaxis against post-operative venous thromboembolism. Heparin-induced thrombocytopenia and thrombosis syndrome (HITTS) is a rare but potentially fatal complication of heparin therapy. We describe a case of HITTS in a 49-year-old woman, after elective cholecystectomy, which resulted in a CVA. The purpose of this case report is to increase the awareness of this phenomenon among surgeons and to discuss its management.

2. Case report

A previously healthy 49-year-old woman underwent an elective laparoscopic cholecystectomy as a day case. She had no risk factors for thrombophilia except for smoking. One preoperative subcutaneous 5000 U injection of unfractionated heparin (UFH) was administered as thromboprophylaxsis. Seven days later, she was admitted with biliary peritonitis, which was treated with intravenous antibiotics and a percutaneous drain. The patient was discharged after 14 days once all symptoms had resolved. During this admission, she received twice-daily subcutaneous 5000 U injection of UFH and the platelet count remained within the normal range.

Two days later, she was re-admitted with a right subphrenic collection that was once again treated with a percutaneous drain. Once more the patient was commenced on twice daily 5000 U UFH. Two days following this admission, she developed a left-sided hemiparesis. The platelet count had fallen to 39×10^9 cells/l. MRI confirmed a right middle cerebral artery infarct. MRI angiography revealed a thrombus in the right internal carotid artery.

A diagnosis of HITTS was made which was confirmed by detection of pathogenic HIT antibodies. Heparin was stopped immediately and replaced with lepirudin. Five days later, the platelet count improved and warfarin was commenced. Abdominal ultrasound confirmed the subphrenic collection had resolved.

There was no improvement in the patient's neurological status at the time of transfer to a rehabilitation unit.

3. Discussion

Although heparin is widely used as the anticoagulant agent of choice in surgical patients, it has several potential adverse effects. The most hazardous of these is HITTS, also known as the white clot syndrome in view of the gross appearance of platelet-rich clots at thrombectomy [1]. The syndrome is usually caused by IgG antibodies against platelet factor 4 and heparin [2]. Patients typically develop

* Corresponding author. Present address: 27 Galloway Road, London W12 0PH, UK. Tel.: +44-7970-171814.
E-mail address: rupendattani@hotmail.com (R. Dattani).

0966-6532/$ – see front matter © 2004 Published by Elsevier B.V.
doi:10.1016/j.ambsur.2004.01.002

heparin-induced thrombocytopenia (HIT) 5–14 days after initiation of heparin therapy although, it may occur immediately in patients previously sensitised to heparin [2], as in our case. Studies of thrombocytopenia during subcutaneous heparin therapy have shown an incidence of HIT as high as 3% and a 0.9% incidence of HITTS [2]. Delayed onset HITTS has been reported up to 3 weeks after cessation of heparin [3].

Clinical warning signs of HITTS include thrombocytopenia, any unexplained thrombotic event (venous or arterial), skin lesions or finding of white clot at thrombectomy [2]. One of the unique features of HIT compared with other drug-induced thrombocytopenias is that it typically presents with thrombosis rather than bleeding [2]. Venous thrombosis (deep venous thrombosis being the most common) is more common than arterial thrombosis [4]. Pulmonary embolism is the most common life-threatening presentation [4]. Neurological complications caused by HITTS are uncommon but once developed have a poor prognosis [5].

HITTS should be considered as a clinicopathologic syndrome and the diagnosis can be made when clinical symptoms are associated with pathologic HIT antibodies, detected using either a functional or serological assay [2].

American College of Chest Physicians Consensus on Antithrombotic Therapy recommends all patients receiving heparin should have a baseline platelet measured [6]. Platelet count should then be monitored either daily or every second day, during the high-risk period for HIT, i.e. 5–14 days after starting heparin therapy. Persistent decrease in the platelet count of less than 100×10^9 cells/l or a 30% reduction from the baseline should prompt a diagnosis of HIT.

If HITTS is suspected, heparin should be stopped immediately and alternative forms of anticoagulation such as lepirudin, argatroban or danaparoid sodium commenced until resolution of thrombocytopenia, which typically takes 4–7 days [6]. We used lepirudin, which is a recombinant protein that directly inactivates thrombin. Low molecular weight heparin (LMWH) is contraindicated in the treatment of HITTS [6]. Warfarin can be considered once the platelet count rises above 100×10^9 cells/l. The patient with a history of HITTS should never be re-exposed to heparin unless absolutely necessary [2].

The use of LMWH is now becoming increasingly popular in some surgical units. Compared with UFH, LMWH has been shown to be both superior as a thromboprophylaxsis agent and is associated with a much lower incidence of HITTS [6]. There are limited reports in the surgical literature describing this entity. All health care professionals should have a greater awareness of HITTS.

References

[1] Towne JB, Bernhard VM, Hussey C. White clot syndrome: peripheral vascular complications of heparin therapy. Arch Surg 1979;114:372–7.
[2] Leopold MAJ, Cancio C, Cohen DJ. Heparin-Induced Throbocytopenia and Thrombosis. J Am Coll Surg 1998;186:77–89.
[3] Warkenstein TE, Kelton J. Delayed-onset heparin-induced throbocytopenia and thrombosis. Ann Intern Med 2001;135:502–6.
[4] Warkenstein TE, Kelton JG. A 14-year study of heparin-induced thrombocytopenia. Am J Med 1996;101:502–7.
[5] Pohl C, Klockgether T, Greinacher A, Hanfland P, Harbrecht U. Neurological complications in heparin-induced thrombocytopenia [letter]. Lancet 1999;353(9165):1678–9.
[6] Hirsh J, Warkentin TE, Raschke R. Heparin and low molecular weight heparin: mechanisms of action, pharmacokinetics, dosing considerations, monitoring, efficacy and safety. Chest 1998;114:489–510.