Hemorrhagic Shock Every other Day

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Abstract

Catheter dysfunction is an important cause of catheter loss. In order to prevent this, locking solutions with minimal risk of systemic anticoagulation are used to ensure catheter patency. At present the most used solutions are either heparin or sodium citrate. According to the literature use of sodium citrate may be advantageous in reducing bleeding events. We report a case of hemorrhagic shock following hemodialysis catheter lock with heparin, reversed after switching solution to sodium citrate.

Keywords: Hemorrhagic shock, heparin, catheter lock, sodium citrate

Abbreviations: HB: Haemoglobin; APTT: Activated Partial Thromboplastin Time; INR: International Normalized Ratio; NE: Norepinephrine; FFP: Fresh Frozen Plasma; PC - Platelet Concentrate; TT: Thrombin Time; CVVHDF: Continuous Venovenous Hemodiafiltration.

Introduction

Use of a catheter lock solution plays a decisive role in hemodialysis access patency. At present the most commonly used solutions are either heparin or sodium citrate. There are different prospective studies comparing these two sealing solutions, however the authors only found one clinical report describing the risks of adopting one of the strategies [1]. We report a case of hemorrhagic shock following hemodialysis catheter lock with heparin.

Case Report

We present a 56-year-old black woman with end-stage renal disease secondary to autosomal dominant polycystic kidney disease. Seven years after she started on hemodialysis she was admitted to a Regional Transplant Centre for kidney transplant from a deceased donor. Besides past event of thrombotic occlusion of arteriovenous fistula, she had no history of other thrombotic episodes. Also, despite heparin exposure three times a week, no history of bleeding diatheses was reported.

Before surgery, three hours of hemodialysis without anticoagulation were done, and each lumen branch of her tunneled cuffed catheter placed on internal jugular vein was locked with high concentrated heparin.

Surgical implantation of donor’s kidney occurred without technical difficulties, although hemostasis was hard to perform. She presented immediate diuresis and no major problems were reported. After her transfer to a medical ward, she started severe abdominal pain; the surgical drain presented with highly hematic content and the patient evolved to hemorrhagic shock. Blood tests revealed hyperlactacidemia (5.6 mmol/L), acute anemia (hemoglobin (Hb) 6.7 g/dL), thrombocytopenia (76×10\(^9\)/L) and clotting disorder (activated partial thromboplastin time (aPTT) 47 seconds, international normalized ratio (INR) 1.3, fibrinogen 1.85 g/L). Vasopressor support with norepinephrine (NE) was initiated and a total of four red blood cell (RBC) concentrate, six fresh frozen plasma (FFP) units and one platelet concentrate (PC) were administered, ineffective. Repeated blood tests showed Hb of 6.6 g/dL, platelets of 66×10\(^9\)/L, aPTT of 83.6 seconds and undetermined thrombin time (TT); and so, an exploratory laparotomy was performed. Multiple bleeding points were found but no evidence of dots or a clear hemorrhagic point. The intraoperative thromboelastogram revealed normal HEPTEM with prolonged INTEM; admitting heparin poisoning from the pre-transplant haemodialysis, 25mg of protamine sulphate were administered with...
improvement of bleeding status and clot formation. Immediately after surgery she developed anaphylactic shock, attributed to protamine sulphate. She was invasively intubated and ventilated, started NE for haemodynamic stability and continuous veno-venous hemodiafiltration (CVVHDF) without anticoagulation. She remained dependent of multiple blood products and vasopressor support but bleeding dyscrasia was stabilized.

Due to coagulation of the extracorporeal circuit CVVHDF was suspended after 24 hours (day 3) and each lumen branch of the catheter was locked with high concentrated heparin. Blood tests collected hours after revealed undetermined TT, aPTT of 170.9 seconds, INR 7.1 and fibrinogen 1.86 g/L. Increasingly doses of NE were needed as the patient progressively presented with higher hematic surgical drainage and refractory hypotension. Since protamine sulphate could not be administered, blood products were given for hemostasis support. On the following morning (day 4), she had no evidence of active hemorrhagic events, and her coagulation tests were normal (aPTT 27 seconds, INR 0.9). Due to hypervolemia she started sustained low-efficiency dialysis without anticoagulation. The treatment was uneventful, with no need for vasoactive agents. After treatment the lumen branches were again locked with heparin. On the next day, she re-started uncontrolled bleeding from nose, mouth, upper digestive tract and surgical site. Coagulation tests confirmed a prolonged aPTT (125 seconds) with normal INR. Further coagulation studies revealed a nonspecific decrease in XIII factor. After excluding technical human errors and given the timeline of the events it was considered that the coagulopathy was caused by the presence of heparin, although in small quantity and only placed on the lumen branches of catheter. Sodium citrate was used as sealing solution since then and no further bleeding episodes or alteration of coagulation blood tests were found. After a total of sixteen RBC, fourteen PC, 12 FPP and 3g of fibrinogen, and from that day on, only one more RBC unit was administered.

Discussion

Preservation of hemodialysis access is of utmost importance. Catheter dysfunction is a frequent and common cause of hemodialysis inadequacy and catheter loss. High concentrations of heparin or other anticoagulant solution, as sodium citrate, are routinely used to fill the catheter and to maintain his patency after the hemodialysis session [2,3]. Heparin improves the affinity between antithrombin II and thrombin, antagonizing anti-Xa and anti-IIa activities [4], inhibiting adhesion and aggregation of platelets, enhancing the activity of protein C and stimulating vascular endothelial cells to release anticoagulant and fibrinolytic substances [5]. Heparin overflow can increase the risk of systemic anticoagulation and heparin-induced thrombocytopenia [5,6]. Despite the small volumes of heparin used, equivalent to the volume of the catheter lumen, significant elevations of aPTT values measured after catheter locking with this solution have been observed [2,6]. Moreover, in vitro studies demonstrated that filling the dialysis catheter lumen with its volume results in considerable leakage and some authors reported measurable plasma concentrations of gentamycin when it is used as catheter sealing solution [2]. Concentrated heparin is associated with increased bleeding complications compared with low-dose heparin [7]. This risk of serious bleeding is particularly detrimental in postoperative and intensive care unit patients [2,8]. On the other hand, citrate inhibits the activation of the coagulation cascade by acting as a calcium chelate [3], reducing blood circulation of active calcium ions and preventing thrombin production. According to the literature, citrate catheter sealing with sodium citrate is associated with significantly fewer systemic bleeding complications than heparin [5,8]. It also seems to reduce the occurrence of catheter blockage and catheter-related bacteraemia making it a potentially better agent and the sealing solution of choice for patients at high risk of bleeding [2,5,8].

Conclusion

The risk of heparin locks related bleeding when using cuffed tunnelled catheters is underestimated in our daily practice. Complex hemostatic derangements resulting from endothelial stress, circulating anticoagulant and fibrinolytic factors, consumption coagulopathy or massive hemodilution can occur after surgery, making perioperative patients a special risk group for major bleeding. Sodium citrate is an accessible and cost-efficient agent with similar catheter patency rates when compared to heparin. Its use avoids inadvertent systemic anticoagulation which can be devastating in susceptible individuals as surgical and UCI patients.

Conflict of interest

The authors declare that there is no conflict of interest.

References

