

Chapter 31

Management of Bleeding in the Postoperative Cardiac Patient

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ABSTRACT

Bleeding in the postoperative cardiac surgical patient can be multifactorial. This chapter examines the preoperative and intraoperative risk factors for having significant postoperative bleeding. It also discuss the advantages and disadvantages of standard laboratory testing as well as point-of-care tests, such as thromboelastography (TEG) and thromboelastometry (ROTEM), in their diagnostic capabilities. Finally, we conclude with different treatment strategies in this challenging patient population along with diagnostic criteria of clinically significant postoperative bleeding and when to return to the operating room for re-exploration.

DIAGNOSING THE COAGULOPATHY

Preoperative Coagulopathy Risk Factors in the Cardiac Surgery Patient

The cardiac surgical patient may have many reasons for having a coagulopathy prior to surgery and a complete history and physical examination preoperatively should reveal these risk factors. These include known coagulation factor deficiencies that are congenital or acquired, pharmacotherapy, concurrent systemic disease such as renal failure or severe liver compromise and rarely, infection causing disseminated intravascular coagulopathy (DIC).

The most common of these is pharmacotherapy since many of these patients have had recent non-ST elevated myocardial infarctions (NSTEMI) or ST elevated myocardial infarctions (STEMI) and had a loading dose of either thienopyridines, such as Plavix, or glycoprotein IIb/IIIa inhibitors, i.e., tirofiban. In acute coronary syndromes, atherosclerotic plaque is disrupted and activates platelet aggregation and intracoronary thrombus formation which can subsequently lead to further myocardial infarction territory

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and death (Boersma et al., 2002). Thienopyridines inhibit P2Y₁₂ receptors on platelets and given with aspirin, which inhibits cyclooxygenase-1 enzyme, constitutes the standard of care of dual antiplatelet therapy (Silvain et al., 2011). Dual antiplatelet therapy is recommended by the American College of Cardiology, American Heart Association, American College of Chest Physicians and European Society of Cardiology for patients undergoing percutaneous coronary interventions (PCI) with STEMI or with unstable angina/NSTEMI (Hirsh et al., 2008).

Other pharmacotherapies that may interfere with a balanced coagulation system prior to cardiac surgery are vitamin K antagonists, such as warfarin, direct IIa (thrombin) inhibitors, i.e. dabigatran or direct Xa inhibitors, i.e. rivaroxaban. These drugs can be used to prevent stroke in those patients with atrial fibrillation. Unfractionated heparin is a class I recommended treatment after the diagnosis of unstable angina/NSTEMI or STEMI is made (Jneid et al., 2012). Heparin exerts its effect by accelerating the action of antithrombin, a proteolytic enzyme that inactivates IIa (thrombin), IXa and Xa. The use of unfractionated heparin can lead to an acquired factor deficiency or heparin resistance.

A final category of drugs that potentially interferes with normal clotting mechanisms are the herbal supplements and vitamins. These have become increasingly popular in recent decades, from 22% in the 1990s to approximately 60% currently (Eisenberg et al., 1998; Heller et al., 2006). Fish oil is one of the leading vitamins due to its beneficial effects in preventing cardiovascular diseases. Decreased thromboxane A₂ production, prolonged bleeding time, and attenuated platelet aggregation have been demonstrated to be a dose-dependent complication of fish oil (Marsh & Coombes, 2006; Schmidt, Varming, Ernst, Madsen, & Dyerberg, 1990). Even though these effects on the hemostatic pathway are known, the controversy lies in if these mechanisms cause clinically significant bleeding. Two recent studies in patients undergoing lumbar decompression surgery demonstrated no increase in intraoperative and postoperative bleeding (Kepler, Huang, Meredith, Kim, & Sharma, 2012; Meredith et al., 2012). Other popular herbal and vitamin supplements that may increase bleeding in the surgical patient are garlic, ginkgo biloba, ginseng, saw palmetto, chondroitin and glucosamine, and vitamin E (Wong, Gabriel, Maxwell, & Gupta, 2012).

Uremia due to renal failure can cause a coagulopathy that is multifactorial and includes intrinsic platelet defects as well as abnormal platelet-endothelial interaction (Weigert & Schafer, 1998). Most importantly is platelet dysfunction which is due to both decreased platelet aggregation and impaired platelet adhesiveness. Diminished platelet adhesiveness is most likely caused by intrinsic dysfunction of glycoprotein IIa/IIIb in uremia, a platelet membrane glycoprotein which actions include platelet aggregation and adhesion by its interaction with fibrinogen and von Willebrand factor (Gawaz et al., 1994). Severe liver disease can cause most coagulation factors to be present in decreased quantity. Liver dysfunction also results in additional sialic acid residues on fibrinogen and other factors that impair coagulation. Splenomegaly, in these patients, can cause thrombocytopenia. Impaired clearance of endogenous plasminogen activators accentuates fibrinolysis and decreased levels of coagulation inhibitors induce a consumptive coagulopathy in cirrhotic patients (Marengo-Rowe AJ, 1988). In patients suffering from endocarditis, a coagulopathy can occur in the preoperative period due to low coagulation factors. Low coagulation factors may be caused by DIC, as well as by loss of plasma and impaired hepatic synthesis in the course of sepsis (Levi, Toh, Thachil, & Watson, 2009).

The preoperative cardiac surgical patient may have congenital or acquired coagulation factor deficiencies. von Willebrand disease is the most common inherited coagulopathy since it is an autosomal dominant disorder with variable expressivity. It has an incident rate of 1.4 to 5 cases per 1000 population (B. D. Spiess, Jay Horrow, Joel A. Kaplan 2011). Heyde's syndrome has recently been recognized as a type IIa, acquired von Willebrand disorder, due to a deficiency of high-molecular-weight von Willebrand

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