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Review Article

Management of bleeding in vascular surgery

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Abstract

Management of acute coagulopathy and blood loss during major vascular procedures poses a significant haemostatic challenge to anaesthetists. The acute coagulopathy is multifactorial in origin with tissue injury and hypotension as the precipitating factors, followed by dilution, hypothermia, acidemia, hyperfibrinolysis and systemic inflammatory response, all acting as a self-perpetuating spiral of events. The problem is confounded by the high prevalence of antithrombotic agent use in these patients and intraoperative heparin administration. Trials specifically examining bleeding management in vascular surgery are lacking, and much of the literature and guidelines are derived from studies on patients with trauma. In general, it is recommended to adopt permissive hypotension with a restrictive fluid strategy, using a combination of crystalloid and colloid solutions up to one litre during the initial resuscitation, after which blood products should be administered. A restrictive transfusion trigger for red cells remains the mainstay of treatment except for the high-risk patients, where the trigger should be individualized. Transfusion of blood components should be initiated by clinical evidence of coagulopathy such as diffuse microvascular bleeding, and then guided by either laboratory or point-of-care coagulation testing. Prophylactic antifibrinolytic use is recommended for all surgery where excessive bleeding is anticipated. Fibrinogen and prothrombin complex concentrates administration are recommended during massive transfusion, whereas rFVIIa should be reserved until all means have failed. While debates over the ideal resuscitative strategy continue, the approach to vascular haemostasis should be scientific, rational, and structured. As far as possible, therapy should be monitored and goal directed.

Key words: acute coagulopathy; bleeding management; vascular surgery

Editor's key points

- Much of the evidence on the management of major bleeding comes from studies conducted in trauma patients.
- Blood and FFP and platelets should be administered early in major bleeding.
- The use of fibrinogen concentrate is recommended in major bleeding where low circulating concentrations of fibrinogen have been demonstrated or are suspected.
- Data from the IMPROVE trial suggest that aggressive permissive hypotension (SAP<70 mm Hg) may be associated with worse outcome.

Acute coagulopathy with significant blood loss is frequent in major vascular procedures. The problem is confounded by the high prevalence of antithrombotic agent use among patients undergoing vascular surgical procedures and intraoperative heparin administration. The management of perioperative bleeding is constantly evolving with ongoing debate over several contentious issues such as optimal transfusion and fluid management strategies. Much of the existing evidence and guidelines are derived from studies on patients with trauma. This article aims to review the management of bleeding in vascular surgery. The approach to vascular haemostasis should be scientific, rational, and

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structured. As far as possible, therapy should be monitored and goal directed.

Pre-existing and acquired coagulopathy

Patients with vascular disease often pose complex haemostatic challenges. They are usually elderly with cardiovascular co-morbidities, are frequently taking antiplatelet or antithrombotic agents including new oral anticoagulants (NOACs), may have platelet dysfunction secondary to renal impairment, and often receive intraoperative heparin. Many, therefore, have pre-existing coagulopathy.

Acute coagulopathy acquired in vascular procedures has not been well studied. In a pooled analysis of seven studies of patients with ruptured abdominal aortic aneurysms[1] the prevalence of coagulopathy at presentation was 12.5%, but serum fibrinogen was normal in most (97%) of them. This contrasts with acute trauma coagulopathy (ATC) where one in four patients after major trauma had coagulopathy before resuscitation and this was associated with a four-fold increase in mortality.[2] ATC is usually multifactorial in origin with tissue injury and hypotension as the initial triggering factors, followed by dilutional coagulopathy, hypothermia, acidemia, hyperfibrinolysis and systemic inflammatory response syndrome (SIRS), producing a spiral of events that are self-perpetuating. Recognition of ATC as an intrinsic phenomenon has prompted the concept of early haemostatic treatment. Acute coagulopathy in vascular surgery may share similar pathophysiology. The severity of shock or tissue hypo-perfusion correlates with coagulopathy. In the absence of shock, patients often have a normal coagulation profile. How shock leads to coagulopathy remains unclear. Tissue injury activates both cellular and humoral elements of the immune system. Activation of coagulation proteases triggers complement release, platelet degranulation releases phospholipid mediators,[3 4] widespread endothelial disruption, increased thrombomodulin activity, and Protein C activation, and these have all been implicated.[5]

The dilution of coagulation factors is now recognized as a major cause of acute coagulopathy. Reduced intravascular hydrostatic pressure during shock causes fluid shift into the intravascular space and dilution. This is exacerbated by crystalloid use during resuscitation and more red cell (RBC) transfusion. Hypothermia inhibits coagulation protease activity and platelet function. Platelet activation is mediated through glycoprotein Ib/IX. At low temperature, effect of von Willebrand factor (vWF) on the glycoprotein is depressed. The activity of FVIIa and platelets decline linearly with reduced temperature.[6]

Acidosis occurs as a result of tissue hypoperfusion and excess chloride administration. It impairs plasma protease activities and coagulation factor complexes and cell surface interactions. The production of thrombin and the activity of clotting factors are all retarded. A decrease in pH from 7.4 to 7.0 reduces the activity of factor VIIa by 90%, factor VIIa/ tissue factor complex formation by 55% and factor Xa/Va complex formation by 70%. Administration of buffer solution does not seem to correct this coagulopathy. Other key changes in acute coagulopathy are reduction in thrombin generation, fibrinogen depletion and impaired fibrin formation. A reduced serum fibrinogen concentration to unstable clots is associated with increased transfusion requirement and mortality in patients with trauma.[7] Rapid restoration of serum fibrinogen has been shown to reduce transfusion and mortality.[8] Certain synthetic colloids with high molecular weight such as older generation hydroxyethyl starches, are large molecules that are difficult to be broken down. These high molecular weight colloids, although seldom used nowadays, can interfere with clot formation. Hyperfibrinolysis is common after endothelial injury and release of tissue plasminogen activator, has also been shown to play an important role in acquired coagulopathy.[9]

Perioperative management of antiplatelet and anticoagulant drugs

Drugs used for thromboembolic prophylaxis increase patients' risk of bleeding.[10] The decision to discontinue such therapy before vascular surgery is a dilemma between the risk of bleeding and that of ischaemic events.[11 12]

Anti-platelet agents

Current guidelines recommend that for elective procedures with high-to-very-high bleeding risk, non-aspirin antiplatelet agents should be discontinued five days before surgery, to minimize the risk of bleeding and need for allogeneic blood transfusion, while aspirin be continued throughout the perioperative period.[13] Aspirin is indicated for secondary prevention in cardiovascular disease, hence, should be stopped when it is taken for primary prevention (http://www.fda.gov/Drugs/ResourcesForYou/Consumers/ucm390574.htm#primary). Surgery results in an inflammatory and pro-thrombotic state with increased platelet aggregation. It is thought that the anti-inflammatory and antiplatelet effects of aspirin confer cardio-protection during the perioperative period. In the placebo-controlled trial POISE-2 that enrolled patients undergoing noncardiac elective surgery, the use of aspirin did indeed reduce cardiovascular mortality, but this advantage was offset by an increase in postoperatively bleeding, which itself can contribute to myocardial events.[14] Worth noting from the POISE 2 trial is the fact that only 23% of the patients enrolled had underlying ischaemic heart disease, and patients that had less than six weeks of bare metal stent (BMS) placement or within a year of drug eluting stent (DES) placement were excluded from the study. Furthermore, patients requiring aspirin for secondary prevention made up less than 36.3% of those assigned to the aspirin group. Therefore, one can argue that the high-risk group in the POISE 2 trial may have been diluted with lower risk patients.[15] The increase in bleeding with perioperative aspirin use demonstrated in the POISE 2 trial, has been demonstrated in several other studies.[16 17] In a recent randomized placebo controlled trial involving 5784 patients who underwent coronary artery bypass surgery, it was shown that the composite of death, thrombotic events, and rate of major bleeding were similar between the two groups.[18] Based on a review of studies, continuation of aspirin in patients with ischaemic heart disease, cerebrovascular disease or peripheral artery disease is still the current recommendation for vascular surgery.[19]

In the event of emergency surgery with significant intraoperative bleeding, platelet concentrates with or without desmopressin can be administered to reverse the anti-platelet effect. Based on limited evidence, it is recommended to maintain the platelet count above 50×10^9 cells litre^{-1} in mild to moderate bleeding, and above 100×10^9 cells litre^{-1} in severe or ongoing bleeding.[20]

Vitamin-K antagonists

Vitamin-K antagonists (e.g. warfarin), are still commonly prescribed but are progressively being superseded by more titratable drugs. They work by inhibiting the enzyme vitamin K epoxide reductase and, hence, the recycling of the inactive vitamin K

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