



# Vascular Access Device Thrombosis

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Central venous vascular access devices (VADs) are used for the delivery of chemotherapy, antibiotics, blood products, and nutritional support, as well as for withdrawal of blood samples. One of the complications associated with VAD placement is thrombus formation. In large outcome analysis studies of VADs, the incidence of thrombotic occlusion ranged from 3%–50% (Bodner et al., 2000; Glaser, Medeiros, Rollins, & Buchanan, 2001; Grove & Pevec, 2000; Moureau, Poole, Murdock, Gray, & Semba, 2002; Walshe, Malak, Eagan, & Sepkowitz, 2002).

Thrombotic complications are caused by fibrin buildup within and around the catheter. Catheter occlusions also can be caused by nonthrombotic events such as mechanical obstructions, drug or mineral precipitates, and lipid residue; however, 98% of VAD occlusions are caused by thrombosis (Herbst, Kaplan, & McKinnon, 1998). Potential consequences of catheter occlusions include loss of catheter use, venous thrombosis, and infection. Often, the catheter must be removed or replaced. These complications can result in an increase in hospital and treatment costs, patient length of stay, morbidity, or death (Luptak, 2001).

Three factors lead to thrombus formation: changes in blood flow, in coagulability, and in the blood vessel wall. Blood constantly is circulating in the human body. Any condition or disease state that contributes to the development of venous stasis increases the risk of thrombus formation. Venous stasis can occur with dehydration, hypotension, atrial flutter or fibrillation, immobility, or physical deconditioning. Certain types of cancers, such as breast cancer, lymphoma, lung cancer, and large solid tumors that involve the head and neck, are more likely to cause compression of the vessels in the upper chest, resulting in venous stasis or superior vena cava syndrome (Bagnall-Reeb, 2001; Knobf & Durivage, 1997).

The second factor, changes in coagulability, can result from several disease states. Coagulopathies can be hereditary but also can be related to malignancies, septicemia, or an inflammatory disease process. These coagulopathies alter antithrombin III and protein C (two naturally occurring anticoagulants), protein S, and factor V and result in abnormal thrombus formation. In addition, certain medications such as tamoxifen and estrogen-derived hormones can decrease anticoagulant proteins (Bagnall-Reeb, 2001; Knobf & Durivage, 1997).

Third, changes in the blood vessel wall result from trauma or injury to the vessel and, subsequently, the vascular endothelium. This damage can be initiated by the insertion of the central venous catheter. The damage to the vessel attracts platelets within seconds; in turn, these platelets are attracted and adhere to the subendothelial tissue of the blood vessel. At the same time, the coagulation cascade is activated in which the tissue factor is released because of cellular injury from the damaged vessels. The tissue factor then interacts with factor VII (proconvertin) and factor IV (calcium), resulting in increased coagulation activity. The extrinsic pathway meets the common pathway of the coagulation cascade in which prothrombin eventually is converted to thrombin and fibrinogen is converted to fibrin with subsequent clot formation. Therefore, once this extrinsic pathway is initiated via the initial vessel wall injury, blood clotting or thrombus formation occurs. The vessel walls can be injured in the presence of hypertension, diabetes, septicemia, or obstetrical complications. Various drugs or treatments, such as total parenteral nutrition mixtures, chemotherapy drugs, and steroids also can contribute to blood vessel injury. All of these factors, alone or in combination, can cause central venous catheter occlusion (Bagnall-Reeb, 2001; Knobf & Durivage, 1997).

Two main types of catheter-related thrombotic occlusions exist. Intraluminal clots can occur in the lumen of the catheter or the reservoir of an implanted port, and extraluminal occlusions are found on the exterior of the catheter and include fibrin tails, fibrin sheaths, or mural thrombi. These extraluminal thrombi occlusions can progress to venous thrombosis, leading to complete occlusion of the blood vessel if left untreated (Herbst et al., 1998).

Intraluminal thrombi occur when fibrin or blood products build up inside the lumen of the catheter, causing partial or complete occlusions. Partial occlusions cause resistance when flushing the catheter, decreased rates of infusion of IV products, and the inability to withdraw blood from the catheter. Portal reservoir thrombus occurs when a thrombus forms in the reservoir of an implanted port (Herbst et al., 1998). These types of occlusions may be caused by infrequent flushing, poor technique, frequent blood draws, low infusion rates to “keep the vein open,” reflux from changes in intrathoracic pressure (because of vomiting, coughing, or sneezing), lifting heavy objects, or congestive heart failure (Luptak, 2001).

A fibrin sheath is the buildup of fibrin that forms around the outside of the catheter. The fibrin sheath begins to form soon after the catheter is inserted into the blood vessel, with the origin of the fibrin at the vein entry site or where the infusate first comes into contact

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