

Safe Management of Chemotherapy: Infusion-Related Complications

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Extravasation

1. Pathophysiology: Tissue damage secondary to vesicant infiltration or leakage outside of the vessel that occurs as a result of one of two major mechanisms

- a) The vesicant binds to nucleic acids in the DNA of healthy cells in the tissue, causing cell death. The dead cells release complexes, which are taken up by adjacent healthy cells. This process of cellular uptake of extracellular substances sets up a continuing cycle of tissue damage as the DNA-binding vesicant is retained and recirculated in the tissue for a long period of time (Luedke, Kennedy, & Rietschel, 1979). Examples of DNA-binding vesicants include anthracyclines (daunorubicin, doxorubicin, epirubicin, idarubicin), dactinomycin, mechlorethamine (nitrogen mustard), mitomycin, and mitoxantrone.
- b) The vesicant does not bind to cellular DNA. The vesicant has an indirect rather than direct effect on the cells in healthy tissue. It is eventually metabolized in the tissue and is more easily neutralized than DNA-binding vesicants (Ener, Meglathery, & Styler, 2004). Exam-

ples of non-DNA-binding vesicants include plant alkaloids (vinblastine, vincristine, vindesine, vinorelbine) and taxanes (docetaxel, paclitaxel, paclitaxel protein-bound particles for injectable suspension), which are mild vesicants.

2. Factors affecting tissue damage severity
 - a) Type of vesicant extravasated (DNA-binding or nonbinding)
 - b) Concentration and amount of vesicant in the tissue
 - c) Location of extravasation
 - d) Patient factors, such as older age, comorbidity (e.g., diabetes), and impaired immunocompetence (Ener et al., 2004; Schulmeister, 2011)
3. Risk factors for peripheral extravasation (Goolsby & Lombardo, 2006; Sauerland, Engelking, Wickham, & Corbi, 2006)
 - a) Small, fragile veins
 - b) Previous multiple venipunctures
 - c) Prior treatment with irritating or sclerosing drugs, such as chemotherapy
 - d) Sensory deficits
 - e) Limited vein selection because of lymph node dissection, lymphedema, or limb removal
 - f) Impaired cognition, altered mental status (impairs ability to detect

administration site sensation changes), or somnolence

- g) Probing during IV catheter insertion
 - h) Inadequately secured IV catheter
 - i) Administration site in areas with minimal overlying tissue (e.g., dorsum of the hand, wrist, or antecubital area)
 - j) Use of rigid IV devices (e.g., steel-winged “butterfly” needles)
4. Possible etiologies of peripheral extravasations (Sauerland et al., 2006)
- a) Vein wall puncture, piercing, or trauma
 - b) Dislodgment of the catheter from the vein
 - c) Administration of a vesicant in a vein below a recent (less than 24 hours) venipuncture site
 - d) Administration of a vesicant in a vein below a recent or nonhealed vesicant extravasation site
 - e) Inadvertent intramuscular (IM) or subcutaneous (SC) vesicant administration
5. Risk factors for extravasation from central VADs (Sauerland et al., 2006)
- a) Difficulty encountered during device insertion (e.g., probing during venipuncture, inability to advance guidewire or catheter)
 - b) Inadvertent slicing, piercing, or nicking of catheter prior to or during insertion
 - c) Device misplacement with catheter tip outside of the venous system
 - d) Inadequately secured noncoring needles (implanted ports)
 - e) Deeply implanted ports
 - f) Presence of a fibrin sheath or thrombus at the catheter tip
 - g) Catheter migration
 - h) Long dwell time of catheters inserted using a subclavian approach