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Neonatal Extravasation: An Overview and Algorithm for Evidence-based Treatment

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ABSTRACT

The peripheral intravenous (PIV) catheter is the most used vascular access device for the administration of medications in hospitalized neonates, however 95% of PIV catheters are removed due to complications. Infiltration and extravasation are one of the most destructive complications to the neonate's fragile skin. This article reviews multiple aspects of infiltration and extravasation injury. First, starting at the cellular level the role of vesicants in vascular injury and its role triggering inflammation will be discussed, followed by a comprehensive review of vesicants and their mechanism of injury, by pH, osmolality or chemical composition, then an overview of the NICU nurses knowledge and actions to prevent infiltration and ending with the use of an evidence-based algorithm that was developed at one children's hospital to minimize injury caused by extravasations through targeted, prompt treatment.

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Many medications given to neonates have the potential to injure when an extravasation occurs. An extravasation is described by the Infusion Nurses Society (INS) as the inadvertent administration of a vesicant solution or medication into the surrounding tissues.[1] A vesicant is defined as a solution or medication that causes the formation of blisters leading to tissue necrosis and sloughing. Extravasation can result in varying degrees of localized tissue injury and can cause pain, infection, and partial to full thickness tissue loss involving muscles and nerves. If extravasation is severe and depending on the site, skin grafts, long hospitalization and high costs result. Not surprisingly, with disfigurement and loss of function, parents may try to recover payment for their loss by initiating lawsuits.[2,3]

Infiltration Is Common Among Neonates

The peripheral intravenous (PIV) catheter is the most used vascular access device for the administration of medications in hospitalized neonates; however 95% of PIV catheters are removed due to complications such as leaking, occlusion and infiltration.[4] Infiltration rates among neonates are as high as 57%–70% with extravasation occurring in 11–23%.[5] Both infiltration and extravasation are destructive, causing localized injury to the neonate's fragile skin.[6] Infiltration of non-vesicants can cause considerable tissue damage from pressure on internal structures as additional fluid collects around the vein, and in severe cases can result in compartment syndrome.[6] Extravasations have the potential to cause peripheral tissue injury depending on the type of vesicant, concentration of the vesicant, location, amount, and duration of exposure to the vesicant. Damage from a vesicant may progress over time and become evident 48–72 hours after the extravasation occurs.[7,8]

Neonatal Vulnerability to Vascular Injury

The preterm and sick neonate is more susceptible to skin injury and complications from extravasation injury than their mature, healthy counterparts. Their immature skin structures, flexible subcutaneous tissue, small blood vessels and poor venous integrity increase the risk of complication from venipuncture and IV infusions.[5,8] The goal in neonatal care is to prevent skin breakdown whenever possible. Similarly, attention to thermoregulation, pain and stress that infants endure as a result of repeated IV attempts or restarts, and infiltrations and extravasations must be considered and managed.[9,10] Multiple tools are available to score pain responses and enable the NICU (neonatal intensive care unit) nurses to manage this appropriately. Nonpharmacologic measures to decrease pain include the use of a pacifier, swaddling, or administration of sucrose during the insertion of an IV or for infiltration and extravasation injury.[10] Aside from non-pharmacologic interventions, treating infiltration and extravasation pain with analgesics should be considered.[8]

Inflammation in the Premature Infant

The neonatal immune system is poorly regulated compared to adults and dysregulation is magnified when neonates are born

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early.[11–13] While intravenous therapy is necessary in this population, it is not without its risks. Vesicants can harm the endothelial lining of the blood vessel, triggering production and release of oxygen free radicals that spur inflammation.[14,15] The release of the locally induced free radicals into the circulatory system when not controlled can trigger a system-wide response to the stressor and further free radical release. In a normal response, the infant's body mounts an anti-inflammatory release of free radical scavengers. When this anti-inflammatory response is inadequate, which is common in prematurity, or the inflammatory assault is severe, endothelial dysfunction leads to programmed cell death (apoptosis).[16] The load of oxidative stress in premature infants is especially of concern as it has been linked to various neonatal morbidities including necrotizing enterocolitis,[16,17] retinopathy of prematurity,[18–20] and chronic lung disease.[16,21–23]

Pathophysiology of Vascular Injury

In the human body, vascular injury, oxidative stress, and inflammation are intricately related. Infiltration and extravasation are both symptoms of vascular injury. When vascular injury occurs, the release of free radicals is stimulated and the energy producing ability of the cell becomes dysfunctional as mitochondria are damaged. As free radicals are released and the capacity of the damaged mitochondria to produce energy is limited, the balance between nitric oxide and superoxide is disrupted. This reactive oxygen species excess extends the damage.[24–26] A continual cycle of free radical production and endothelial apoptosis occurs leading to injury of cell membranes and vessels. Symptoms of this process include redness, swelling (inflammation with increasing vascular permeability), and visible tissue injury.[25] The inflammatory process involved in extravasation is depicted in Fig 1. Note how the interaction of the biological response to chemical and non-chemical risk factors when exposed to the harmful drug directly affects healthy endothelium, leading to vascular injury. The framework is a helpful tool in understanding the biological aspects of extravasation and action targets to prevent injury.

The Neonatal Intensive Care Unit (NICU) Nurse's Role

NICU nurses monitor the PIV site with vigilance to aid in early identification of infiltration and extravasation and prevent this type of injury whenever possible. Identifying an infiltration may be difficult, even for the most experienced nurse.[10] The NICU nurse is aware of the subtle changes in heart rate, oxygen saturations, apnea, and the more obvious change in behavior such crying and agitation that may indicate problems with the PIV therapy.[4] Neonatal nursing entails not only basic knowledge of the anatomy and physiology of neonatal skin and how to prevent iatrogenic injury from routine nursing care but also knowledge of vascular anatomy and infection control for the safe delivery of infusion therapy.[4,8,10] Several organizations have developed standards and guidelines that assist nurses with the best practice in infusion therapy.[10] These evidence-based guidelines and grading scales for infiltrations can aid with standardizing documentation and protocols that guide nursing care and improve patient outcomes.[5,6]

Potential Origins of Infiltration

There is a supposition that an infiltration or extravasation is caused by IV catheter dislodgement or puncture of the vein during insertion or during handling of the infant. Chemical composition of medications also impacts risk of vein rupture.[5] The vein's tolerance to an infusion is affected by the osmolality and pH of the vesicant, the duration of the exposure, and irritation to the endothelial cells.[4] An additional factor in causing a cannulated vessel to rupture and leak is the pressure in which the medication is being delivered by the infusion pump.[3,5] Janet Pettit describes the following 3 theories of the mechanism of infiltration and extravasations. The first theory states that the irritation of the venous endothelium from the infusate causes vasoconstriction and diminished blood flow.[4] The second theory has been confirmed by dye studies. It shows infiltration of the infusate through the catheter insertion hole created with IV placement; this can occur when the flow proximal to the catheter tip becomes obstructed.[4] The third theory is that irritation of the venous endothelium caused by the osmolality, pH, or chemical composition

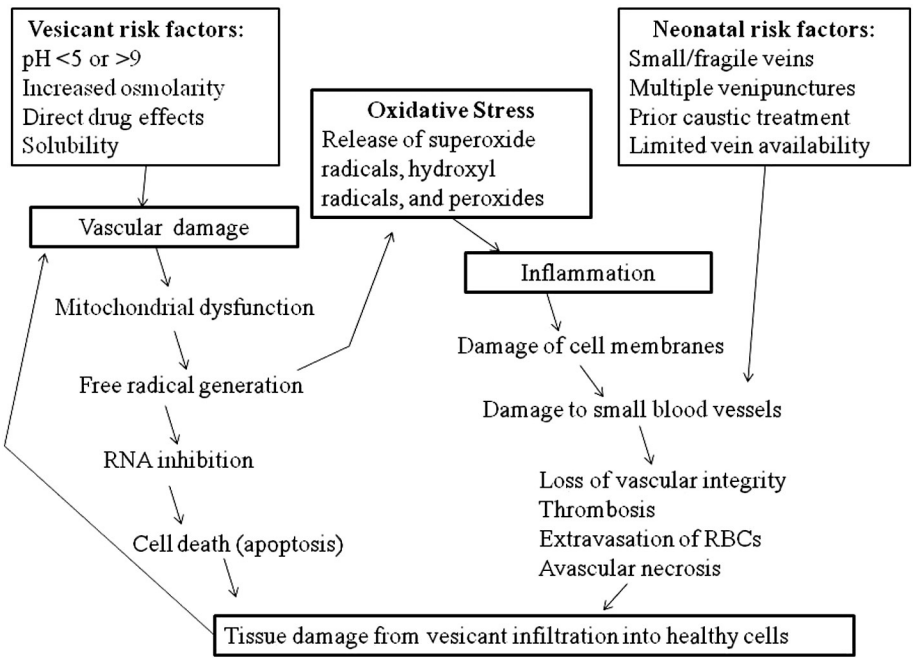

Fig 1. Model of vascular injury.

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