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“Reconstructive Management for Large Hematoma After Cooling for Neonatal Hypoxic Ischemic Encephalopathy”

Background: Neonatal hypoxic ischemic encephalopathy (HIE) is a brain injury that occurs due to insufficient oxygen to the brain at birth. The recommended treatment is whole-body cooling. Although cooling can prevent HIE’s detrimental effects, it has a few known, albeit rare, side effects, including development of subcutaneous fat necrosis (SCFN). We report an unusual presentation of SCFN that progressed to a large hematoma in a newborn undergoing whole-body cooling. He subsequently required surgical intervention, including hematoma evacuation, serial debridement, dermal substitute placement, and a split thickness skin graft.

Case Presentation: A term male infant, delivered emergently via C-section, began whole-body cooling due to respiratory depression and severe HIE. He developed severe thrombocytopenia and coagulopathy, likely secondary to cooling versus perinatal asphyxia and liver ischemia. He required several transfusions of platelets, packed red blood cells, and fresh frozen plasma. On DOL 2, the patient had redness with mild induration to the back and upper arms. The lesions progressed to erythematous coalescing plaques, favoring SCFN. Most notable was a fluctuant 15cm x 15cm nodule of the central upper back. It appeared primarily violaceous with focal areas of blanching and darker areas concerning for necrosis. When seen by plastic surgery, the skin overlying the hematoma had capillary refill along the periphery but a more stressed central area. The team determined that the patient’s coagulopathy and anemia would require correction, after which non-viable skin could be managed non-emergently. To minimize bleeding risk, general surgery evacuated the hematoma once counts had stabilized on DOL 10. Pathology showed necrotic adipose tissue with extensive hemorrhage. On DOL 13, the team performed an incision and drainage followed by debridement of non-viable areas. Once wound cultures were negative and the wound bed appeared healthy, the patient underwent dermal substitute placement followed by serial wound vac changes. On DOL 34, plastic surgery placed a split thickness skin graft harvested from the lower back. On DOL 45, his graft appeared well-adhered. He was discharged and followed up in clinic with excellent graft healing and stable calcium levels at 2 and 4 months.

Discussion: There are limited reports of SCFN lesions coalescing into a large hematoma requiring surgical intervention. The early onset and rapid expansion of our patient’s hematoma can likely be attributed to severe thrombocytopenia. Our report emphasizes the importance of careful attention to the skin of a neonate undergoing whole-body cooling. Following the discovery of progressively coalescing SCFN lesions, early intervention may be warranted. However, the risk of skin necrosis must be weighed against the risks of procedural intervention in coagulopathic patients. In conclusion, hematoma development secondary to SCFN is a rare complication requiring close monitoring and a multidisciplinary team.