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Case Report

Subcutaneous fat necrosis requiring plastic surgical intervention in an infant treated with whole-body cooling

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ABSTRACT

We report an infant who developed subcutaneous fat necrosis of the newborn (SCFN) secondary to cooling treatment for hypoxic ischaemic encephalopathy (HIE). While SCFN is usually self-limiting, this patient went on to develop a large haematoma on his back with overlying skin necrosis necessitating debridement and split thickness skin grafting. Initially, the area affected on his back showed a number of small fluctuant swellings. By day 16 after birth, theses swellings coalesced to form a large 15 cm × 19 cm haematoma with a tense, shiny skin overlying it. On day 17, the large swelling was drained in theatre and a drain was left in situ. Total calcium blood level was raised at 4 mmol/l and he was managed with Pamidronate infusion. Postoperatively, examination of the back showed a 5 cm necrotic area in the centre of the back, and affected area was debrided along with a split skin graft applied to the exposed area.

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Introduction

Subcutaneous fat necrosis of the newborn (SCFN) is a rare form of panniculitis occurring in newborns. They are characterised by single or multiple erythematous-violaceous plaques or nodules, with the potential to evolve into calcifications.[1] These lesions tend to develop at the cheeks, shoulders, buttocks, thighs and calves. Common risk factors associated with SCFN include cord prolapse, perinatal asphyxia and sepsis. A common iatrogenic cause is therapeutic hypothermia which involves whole-body cooling of neonate diagnosed with hypoxic ischaemic encephalopathy (HIE). Whole-body cooling is considered to be an effective and safe therapy but secondary side effects such as bradycardia, hypotension, thrombocytopaenia, and intracranial haemorrhage have been documented.[2] SCFN is a rare dermatological complication of therapeutic hypothermia with a report stating 12 cases of SCFN was identified among 1239 treated infants. It is not entirely clear if the occurrence of SCFN is a result of the underlying asphyxia or the hypothermia as research to date indicates that either may be the cause.[3] We encountered a case of SCFN in a neonate treated with whole-body cooling for HIE. Following treatment, he developed multiple plaques and went on to develop haematoma, fat and skin necrosis over one of the plaque found on his back. This complication necessitated treatment with debridement and split thickness skin grafting.

Case report

A male infant was delivered at 39 + 2 gestational weeks by emergency caesarean section for reduced foetal movements and a reversed end-diastolic flow. At birth he had an Apgar score of 4, weighed 4535 grams and was intubated at 2 minutes. He cried spontaneously when he was extubated at 8 minutes but was re-intubated later that day due to deterioration of arterial blood gas results. Due to low Apgar scores and clinical diagnosis of grade 1–2 hypoxic ischaemic encephalopathy (HIE), the infant was cooled on a cooling blanket at 33.5 °C from 6 hours of birth for 72 hours. An MRI showed a parenchymal haemorrhage in the left occipital lobe, consistent with the diagnosis of HIE.

After three days of cooling, a large area of the infant's back was found to be erythematous. Diagnosis of subcutaneous fat necrosis (SCFN) was given by the dermatologist and he was then nursed in a prone position. He went on to develop smaller firm masses on both arms, lower legs and his cheek (Figure 1).

The area affected on his back showed a number of small fluctuant swellings. At day 16 after birth, theses swellings coalesced to form a large 15 cm × 19 cm haematoma with a tense, shiny skin overlying it. His haemoglobin level dropped from 16 g/dl to 9.9 g/dl with a low platelet count of 99×10^9/l. This was managed with blood and platelet transfusion.

On day 17, the large swelling was drained in theatre and 250 ml of blood stained fluid was removed.

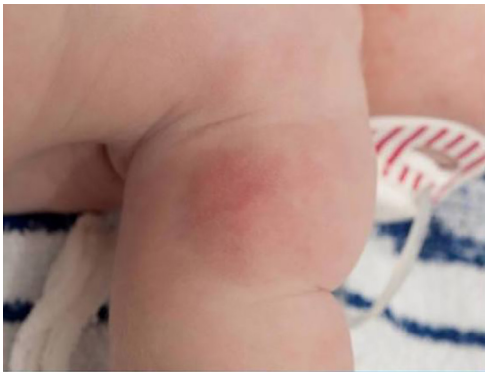

Figure 1. One of many indurated plaques, this one being on the posterior right shoulder.