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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, these 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, thrombocytopenia, 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, these 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 $99 \times 10^9/l$. 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.



Figure 2. Post debridement of necrotic skin and adjacent necrotic fat on the back.

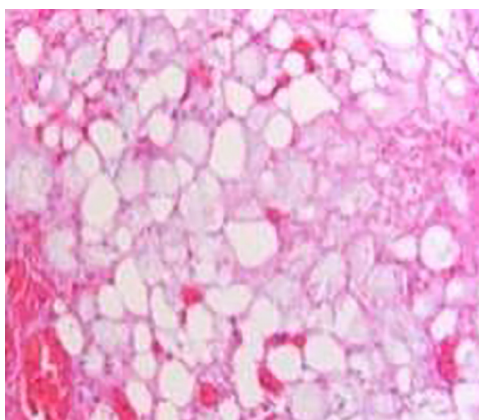


Figure 3. Fat necrosis with abundant foamy macrophages.

A drain was left in situ and this further drained 90 ml of fluid on the first postoperative day, with an average of 40 ml drained on subsequent days. Examination of the back showed a 5 cm necrotic area in the centre of the back, surrounded by what appeared to be viable skin. Postoperatively, a total calcium blood level was raised at 4 mmol/l and he was managed with Pamidronate infusion.

A week later, the necrotic area of his back was debrided and a split skin graft was applied to the exposed area (Figure 2).

The tissue excised was sent for histology and it reported fat necrosis with adjacent subcutaneous fat necrosis (SCFN). The slides showed full epidermal necrosis with massive haemorrhage in the superficial, mid and deep dermis with intense necrosis of the subcutaneous fat. The individual adipocytes were swollen and contained abundant radially eosinophilic crystalline spaces resulting from dissolved lipid. The crystals were largely composed of triglycerides (Figure 3).

This child was followed up in outpatient clinic for 2.5 years and reports no further problems with his back. Serial calcium monitoring was done up to 6 months post operation. (Figure 4)

Discussion

Neonatal hypoxic ischaemic encephalopathy (HIE) can lead to severe permanent disability or death. One method of reducing the severity of HIE is by total body cooling to 33.5 °C from 6 hours of birth for 72 hours.



Figure 4. Graft on the back two weeks post discharge from hospital.

SCFN is well documented as a secondary sequela to the treatment of neonate with a whole-body cooling blanket. The presumed pathogenesis of SCFN is cooling and hypoperfusion of the subcutaneous fat causing necrosis and granulomatous inflammation. Direct contact with the cooling blanket along with damage to cutaneous perforators can result in skin necrosis. Tissue trauma and haematoma formation compounded pre-existent HIE related electrolyte disturbances and thrombocytopenia respectively.

To our knowledge, this is the first case of a SCFN lesion that went on to develop an underlying haematoma, overlying skin necrosis which needed operative intervention by plastic surgery. Two case reports describe an abscess-like swelling in the back associated with whole-body cooling and SCFN. A punch biopsy of this swelling revealed fat necrosis on histological analysis and a chalky white fluid drained out after biopsy. No further operative intervention was required in these cases.^{4,5} There is another case reported where the neonate was ventilated for respiratory distress and went on to develop SCFN and thrombocytopenia. This neonate did not undergo body cooling but one of the back lesions became cystic and blood stained viscous fluid was aspirated. No biopsy was taken in this case.⁶ The other documented surgical intervention for this usually self-limiting condition, was found in a 9 months old child whose SCFN lesion was not resolving. In this case the lesion was removed and the incision closed directly with no need for grafting.⁷

The infant in this report received the cooling therapy according to the published guidelines in a timely fashion. The nursing staff did notice erythema on the back of the neonate post treatment and wisely nursed the infant prone after the third day of birth. This facilitated reduction in pressure on the area after treatment thus preventing further development of necrotic skin.

Hypercalcaemia is the most serious complication and it can develop up to 6 months after the onset of the lesions.⁸ Hypercalcaemia was seen in our neonate at day 21 and this required close observation and Pamidronate infusion. Research has shown that hypercalcaemia develops due to overproduction of 1,25-dihydroxy vitamin D by macrophages in the granulomas, secretion of calcium from necrotic fat tissue and increased prostaglandin activity.⁹ Hypercalcaemia symptoms in a neonate include lethargy, irritability, hypotonia, vomiting, polyuria, polydipsia, dehydration and constipation.

Our report highlights the importance of careful inspection of the skin of any neonate undergoing whole-body cooling. If a rare fluctuant swelling does develop under any lesion, early interventions may be warranted to prevent development of skin necrosis. Our patient was nursed prone on day three after the back erythema was noticed. It may be worth in the future looking into placing the neonate prone intermittently during the 72-hour cooling period. Another documented preventive measure involves the use of pressure-relieving mattresses and pillows.¹⁰

Conclusion

Whole-body cooling is a necessary treatment of HIE but we recommend close vigilance, frequent observation of the skin during the treatment and regular turning of the neonate on the cooling blanket

during this treatment. Close calcium and potassium monitoring needs to occur in the early phase of treatment and up to six months after.

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Conflict of interest

None.

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