Neonatal Pressure Ulcer

8

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8.1 Introduction

The structure of a premature newborn's skin is very different from that of the adult.

In the adult, the stratum corneum is composed of about 20 layers and has an important protective function. However, in the premature baby, this stratum is non-existent or minimal (0–3 layers). The thinness of these external layers is one of the principal sources of vulnerability (see Fig. 8.1).

The stratum corneum normally protects the body from toxins and infections, enables thermoregulation and controls transepidermal water loss.

In the premature newborn, in addition to the thinness of the external layers, there is also a lack of collagen and elastin in the superficial dermal layers. This considerably increases the risk of pressure ulcers, with spontaneous oedema increasing the cutaneous ischaemia.

In the hospital environment, the incidence of neonatal pressure ulcers (NPUs) is significant, affecting one in four babies in the neonatal intensive care unit (NICU) [1, 2].

Four per cent of babies who have been treated in a NICU are left with a scar [3]. In the majority of cases, these skin lesions are minor [4].

The significant incidence of NPUs in the NICU can be explained by the following risk factors [5]:

- The immaturity of the skin
- The restriction of the baby's voluntary movements (e.g. due to a central venous line, mechanical ventilation, etc.)

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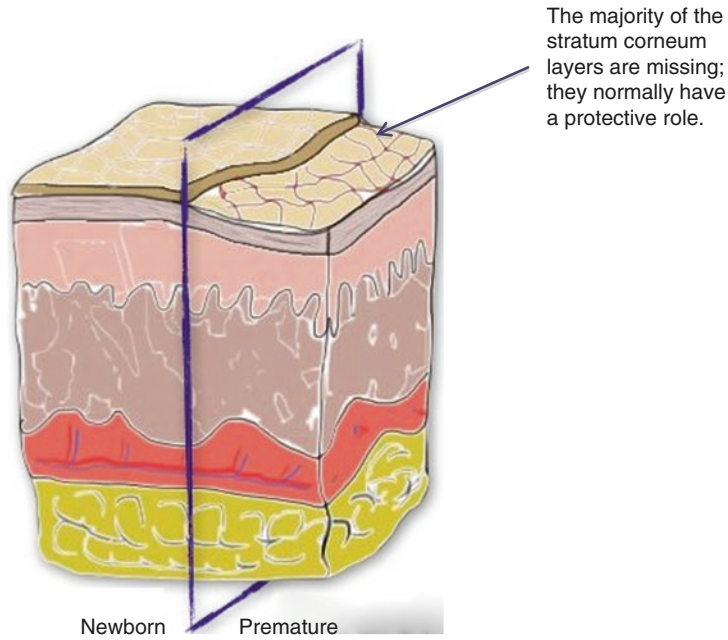


Fig. 8.1 Comparison of normal newborn and premature skin. The stratum corneum is absent or very underdeveloped in the premature skin

- The hot and humid environment
- The use or not of invasive ventilation

This chapter describes the main sources of pressure ulcers in the newborn and premature baby, their characteristic features, the therapeutic principles, and the potential sequelae.

8.2 Risk Assessment Scales

There are many different types of risk assessment scales, most of which have not been tested on a large scale. The Braden Q Scale [1] and the Neonatal Skin Risk Assessment Scale [6] seem to be reliable tools in terms of sensitivity and specificity.

8.3 Principles of Treatment

8.3.1 Topic Treatment

For Stage 2 pressure ulcers, the use of hydrocolloids seems to us to be appropriate in the majority of cases.

For Stage 3 and 4 pressure ulcers, treatment is very similar to that of the adult, but the possibilities available in terms of wound cleaning are often reduced as the baby cannot be moved.

Throughout the liquefactive necrotic stage, the use of silver sulphadiazine as an alternative to hydrogels seems to us to be a possibility. The dressings need to be substantial, thus allowing for additional discharge.

8.3.2 Surgical Treatment

It is reserved for severe cases. It uses the classic reconstruction ladder (skin graft, local and distant flaps). Taking account of the healing capacity of the newborn, the flap realization is often performed in a second step during infant growth.

8.4 Main Areas Affected

1. *The nose*

   The nose is the main area affected, representing half of all NPUs [5]. This has been seen since the 1980s, with the development of nasal continuous positive airway pressure (NCPAP).

   According to different studies, nasal lesion incidence is between 20 and 60 % in premature treated with NCPAP [7, 8]. The majority of occurrence is due to the technique being incorrectly used and/or poor monitoring of skin tolerance [9].

The pressure ulcer most often involves the philtrum, the tip of the nose, the soft triangle, or the nasal septum.

A deformation or a change in function results, which is usually temporary. However, cases have unfortunately been reported in which columellar necrosis has led to a significant disorder in growth.

Figure 8.2 shows a typical lesion due to excessive pressure from an endotracheal tube, which, in bending the soft triangle, has resulted in ischaemia.

Figures 8.3 and 8.4 show two similar cases of columellar necrosis, at different ages. In Fig. 8.4, note the asymmetrical character of the lesion and the consequences on the development of the tip of the nose.

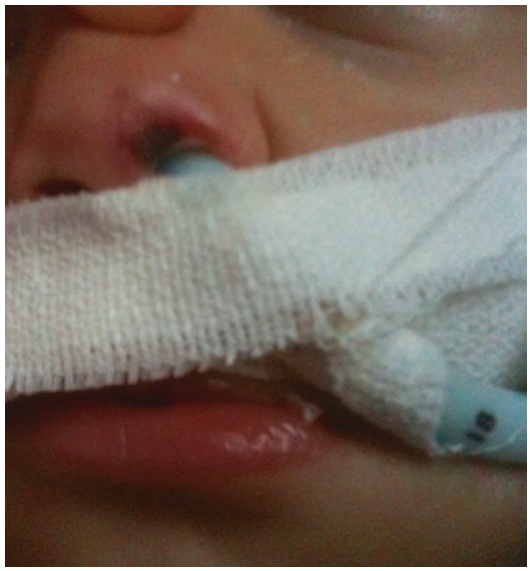


Fig. 8.2 Soft triangle pressure ulcer caused by excessive pressure from the endotracheal tube

2. *The foot and leg*

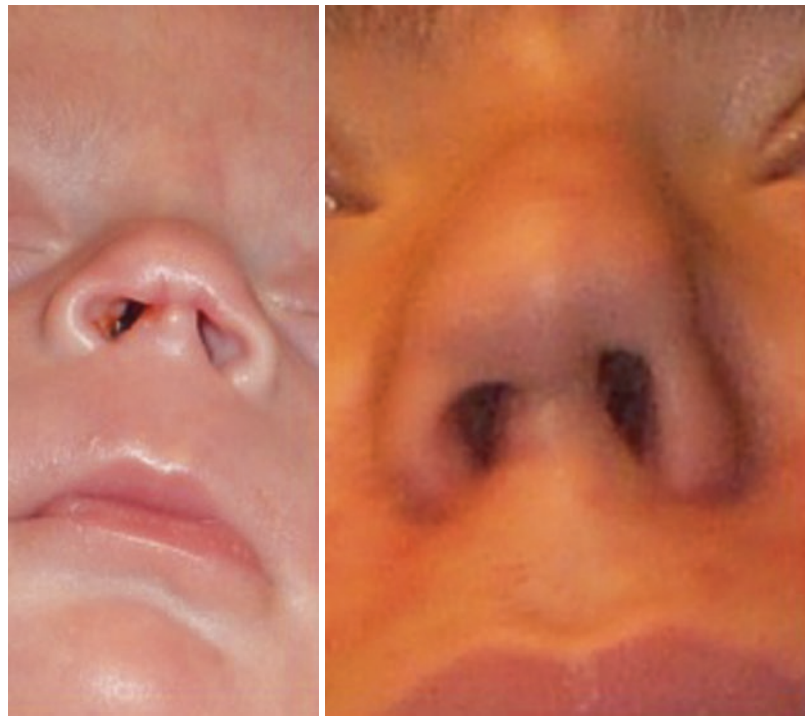
Here, the lesions are genuine pressure ulcers as seen in the bedridden or the paralysed. The main areas affected are the malleoli and the heels. Pressure ulcers have also been reported on the toes, due to the use of oximetry sensors. On the legs, they are often due to the premature use of postural splints.

These ulcers usually improve in a few days after removal of the pressure source and after mechanical or chemical debridement.

3. *The scalp and back*

The two main causes of scalp NPUs are birth trauma and excessive pressure on the occiput.

Scalp injuries are present in around 15 % of babies born using ventouse or forceps [7].



Figs. 8.3 and 8.4 Lesions linked to excessive pressure during NCPAP. The pressure ulcer especially affects the junction of the columella and the apex

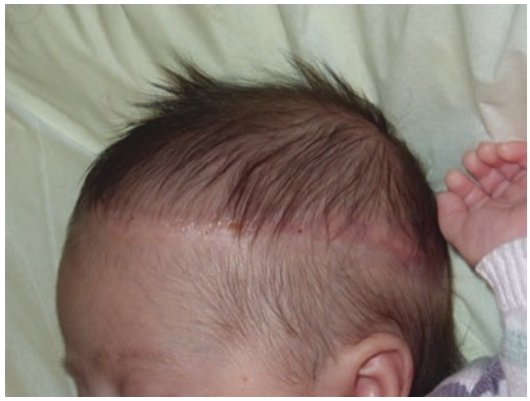


Fig. 8.5 Circular lesion caused by ventouse extraction

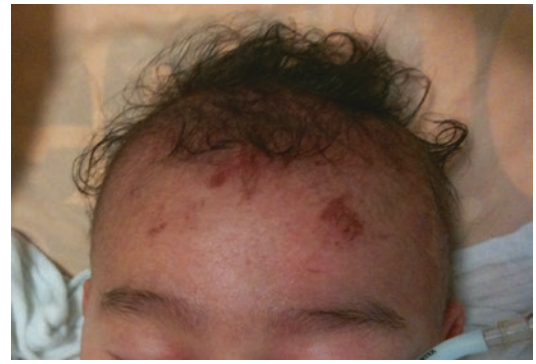


Fig. 8.6 Pressure ulcers on the forehead caused by EEG electrodes

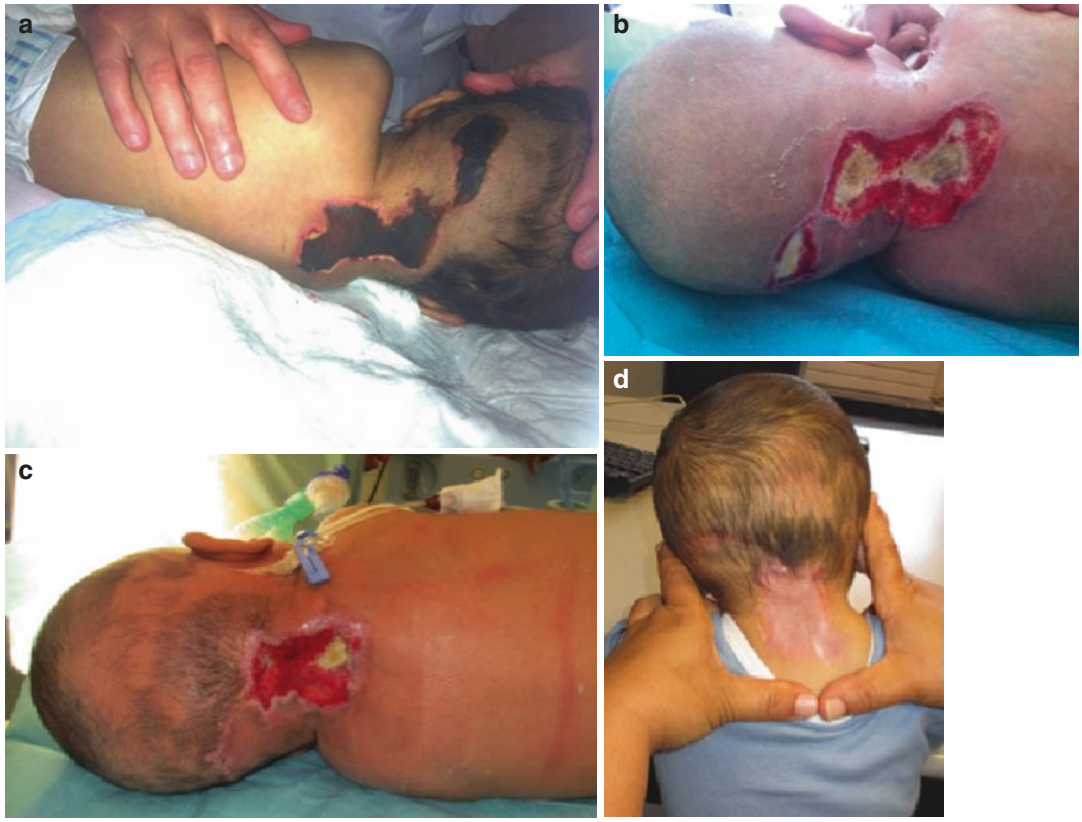


Fig. 8.7 (**a**) Extensive and deep pressure ulcer of the occipital and upper cervical region. (**b**) Result obtained after debridement and initiation of negative pressure therapy. The muscles and aponeurosis have been affected, with contact involving the cervical spine processes. (**c**) Closure of the superior area and repeated debridement (×5). (**d**) Results at 5 months after placement of artificial dermis and thin skin graft taken from the adjacent scalp

The lesions are often deep and semicircular, leaving a visible area of scarred alopecia.

Figure 8.5 shows a case of scalp lesion secondary to ventouse extraction, which affected a ¾-circular area. Wound healing was obtained after 3 weeks of closely supervised healing by secondary intention and involved additional surgical intervention.

Wound healing is usually attained in a few weeks under such supervision. Dressings need to be changed daily and use silver sulphadiazine or hydrogel.

There are other rare locations, which are nevertheless worth mentioning. The use of EEG monitoring equipment can cause pressure on the forehead; this can be a source of ulcers, which are sometimes deep. Figure 8.6 shows an example of lesions seen in intensive care which caused visible scarring.

In a recent study [5], pressure ulcers on the back of the head were found to represent 14 % of all NPUs.

Excessive pressure can cause occipital ulcers, which are sometimes extensive. They affect children in intensive care or recovery. Catastrophic situations can result from the combined risk factors of mechanical ventilation, sedation, a central venous line, humidity, the warmth of the incubator, and the baby's state of shock (see Fig. 8.7).

In the baby seen in Fig. 8.7, the pressure ulcer was due to prolonged excessive pressure in the context of major cardiac surgery, with prolonged low cardiac output. The child had not been turned because of its haemodynamic instability. Healing was obtained after surgical debridement in the intensive care bed, dressings using negative pressure therapy, followed by skin graft on a thin one-layer artificial dermis (Matriderm®). One part was closed without tension.

The result was not perfect (see Fig. 8.7d), but it enabled rapid coverage, without adhesion.

Conclusion

Neonatal pressure ulcers are specific entities which require special attention from neonatal care teams. The thinness of the stratum corneum makes the skin of the premature and nursing baby particularly vulnerable to pressure. During the critical maturation phase, care teams must remain alert to the possibility of pressure ulcer formation, especially in an environment which can encourage such ulcer development.

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