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ORIGINAL PAPER

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Transient neonatal hair loss: a common transient neonatal dermatosis

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Abstract For many years the aetiology of neonatal occipital alopecia (NOA) has been reported to be friction. We have made a retrospective check to see if the incidence of NOA has increased since the new paediatric tendencies of putting children back to sleep in the safest way have been used (APP guidelines). The results of this study in 301 neonates demonstrated that it has not. The aetiology of this phenomenon is the physiological shedding of hair in the first weeks of life. The pillow, which is often blamed, only aids this shedding. Parents should be informed that there is no relationship between the sleep position and the onset of this problem, to prevent them from changing the position of the sleeping child, which could lead to a fatal outcome.

Keywords Neonates · Neonatal occipital alopecia · Transient neonatal hair loss · Sudden infant death syndrome · Halo ring alopecia

Introduction

During the second month of life a number of neonates (NNs) develop an alopecic patch on the occipital region. This very well-known neonatal manifestation normally has a clearly delimited, elongated, oval aspect. (Fig. 1) The main diameter of the alopecic patch is usually from one auricular pavilion to the other. Several authors have proposed the term "neonatal occipital alopecia" (NOA) to describe this transient hair loss [5].

For many years the aetiology of NOA has been thought to be friction, caused by the NN's sleeping position. This hypothesis, usually supported by parents and the majority of paediatricians, has been strengthened by the APP guidelines (*Back to sleep*) campaign, an attempt to prevent sudden infant death syndrome (SIDS). Most paediatricians now recommend that neonates should sleep on their backs as the safest way to avoid SIDS [7].

We have made a retrospective check to see if the incidence of NOA has increased since these new tendencies have been put into practice.

It should first be commented that NOA is not always occipital, so we propose the term "transient neonatal hair loss" (TNHL) to describe this phenomenon.

Patients and methods

Prospective study

We studied 301 neonates born in the neonatal unit at the Umberto I hospital in Italy. Among this main group, 101 were visited at birth during 2003 and revisited 12 weeks later. Reports of TNHL were found in 12 , an 11.88% incidence.

Retrospective study

Considering that after the AAP guidelines in 1992, [6] paediatricians in Italy started to recommend the upward position only around 1998, we investigated 200 records of controls on healthy 3-month-old babies in the period prior to the "back to sleep" tendencies (1985–1995) when children were lain on their stomachs or on their sides to sleep. The only charts taken into account were those from 1985. The incidence found was 9% (18 babies).

We declare that there is no potential conflict of interest, real or perceived

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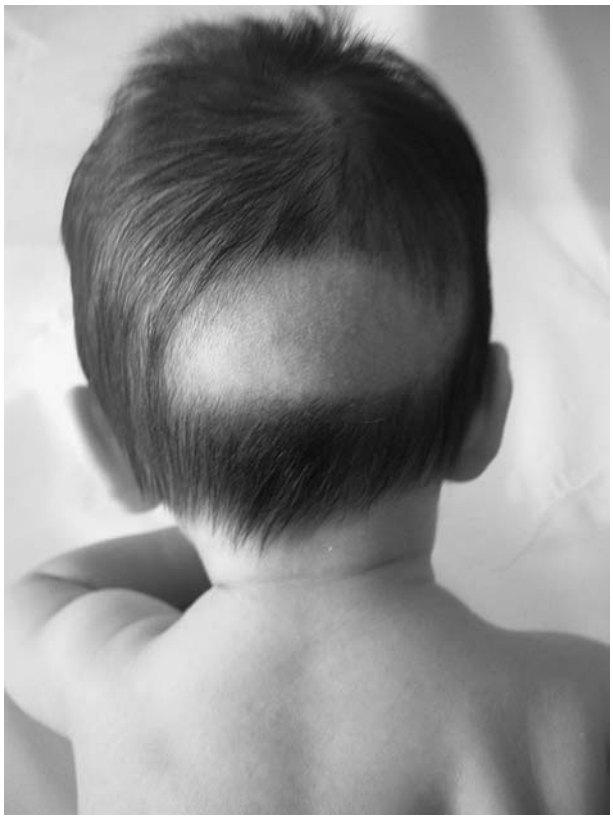

Fig. 1 Transient neonatal hair loss: a typical aspect with the oval area of hair loss in a 2-month-old infant

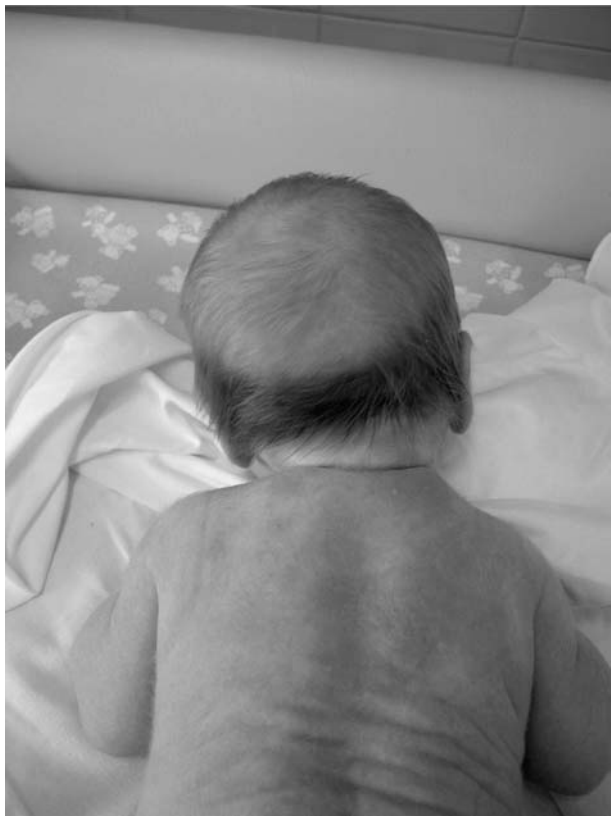

Fig. 2 Transient neonatal hair loss: sometimes the hairless patch shows a marked lineal aspect. The friction–pillow theory is totally unacceptable for this image

Discussion

Several authors hesitate to blame the aetiology of TNHL on the sleep position. From our point of view TNHL should be included among the common transient neonatal dermatoses (CTNDs). The baby's age at appearance, the levity of the lesions, the absence of accompanying symptoms and the spontaneous and rapid resolution of the lesions support the inclusion of TNHL among the CTNDs: ''Clinical manifestations during the neonatal period on a rapid evolution to a self resolution''.

It is now clear that this peculiar CTND has no relationship with the sleep position but with the physiology of hair-shaft shedding. (Fig. 2)

Compared with the period previous to the ''back to sleep'' campaign, when paediatricians suggested that babies should be lain on their stomachs, TNHL had an incidence of 9%; our current study (1988–2004) does not show any significant difference; a 12% incidence ($P = 0.4307$).

The physiological hair cycle on ''foetal hair'' is the real cause of the localised shedding of the occipital patch. In animals the hair cycle is synchronised so that the entire pelage grows continuously through winter [9]. When summer comes growth ceases abruptly, and a cephalocaudal moult ensues. This synchronised growth pattern also occurs with human hair in utero. Hair first appears on the scalp at 20 weeks of gestation and then grows over the rest of the body [4]. Initially, all hairs are in anagen (growing phase). Through a cephalocaudal spread of follicular differentiation, anagen hairs cover the entire scalp by 18 to 20 weeks of gestation. [3] These initial hairs are lanugo, unmedullated hairs [8]. At about the 5th month of gestation, over a period of 7 to 10 days, a wavelike transition of anagen to telogen (rest phase) hairs begins, from the frontal to the parietal area [1]. The hairs in the occiput do not change at this point. The telogen lanugo hairs in the frontoparietal areas are usually shed in utero at 7 to 8 months [3] and are replaced by a second wave of anagen vellus hair growth (second pelage) in the frontal to parietal areas [1].

The lanugo hairs in the first pelage of the occiput remain in anagen until near term, when they abruptly go into telogen.[1]. These occipital telogen hairs are generally shed approximately 8 to 12 weeks later [3], and, until they are replaced by the newly emergent anagen hair growth, this accounts for the decreased density of hair normally seen in this area in 2-to-3 month-old infants.

The pillow (which is often blamed) only aids the shedding of telogen hairs. [9] After the first two synchronised moults and towards the end of the first year of life, there is a change in the relationship of adjacent hair

such that a random mosaic pattern emerges in which each hair follows its own intrinsic rhythm.

From this pathogenic hypothesis every single NN should develop an identical pattern of TNHL between the 8th and 12th-week of gestation. However, in reality this is not so. Many children do not develop the typical occipital patch of transient neonatal hair loss. An explanation for this could be that, in some neonates, there are enough anagen hairs on the affected area to disguise the loss of telogen hairs.

The most important *differential diagnoses* to be considered are the following.

In the parietal pattern of hair loss, which is probably the reason for the confusion about its aetiology, this type of TNHL should be differentiated from *pressure alopecia*. [2] Those children who are very ill, lying on their backs with the occiput on the pillow and immobilised for long periods of time, are liable to develop pressure alopecia. The same type of hair loss can be observed after a prolonged stay in hospital. If the nursing is adequate and the children are periodically moved, there is no permanent damage and the hair grows again. Systemic illness, severe enough to keep the child immobile, can cause pressure alopecia and is a common event in very ill children. Pressure alopecia is not exclusive to neonates as it can also be seen in adults, where it is permanent.

Halo scalp ring is an uncommonly reported perinatal pressure hair loss [10]. Halo scalp ring is a distinctive form of hair loss attributed to caput succedaneum. In general the prognosis is good. From our point of view halo scalp ring is just an extended and elongated variation of TNHL and is not due to caput succedaneum.

Alopecia areata should be differentiated; it is extremely rare in this age group.

Conclusion

Transient neonatal hair loss in its classical occipital form is well known and easily diagnosed by paediatricians and neonatologists.

Parents should be informed of the absence of a relationship between the sleep position and the onset of this problem to prevent them from changing the baby's position (face to bed or lateral position) when it is asleep, as this could lead to a fatal outcome.

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