

TRANSIENT NEONATAL PUSTULAR MELANOSIS

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ABSTRACT

The presence of vesicular or pustular lesion in the neonatal period evokes justifiable concern in a clinician caring for this vulnerable population. Both life-threatening bacterial and viral infections may present in this fashion.

We present a white male neonate who was diagnosed as transient neonatal pustular melanosis. It is important to differentiate this type of benign and transient dermatosis from life-threatening pyoderma, because its recognition may spare the healthy neonate from extensive sepsis work-up, antibiotic therapy, and prolonged hospitalisation.

Key Words: Neonate, Dermatitis, Pyoderma, Transient neonatal pustular melanosis

INTRODUCTION

The occurrence of transient neonatal pustular melanosis (TNPM) is reported as high as 4.4 % in all full-term black and as high as 0.6% in white neonates (1). Merlob, et al. reported an incidence of 0.24% (n=5267) and found no difference between infants of Ashkenazi and Oriental ancestry (2). This disorder presents at birth as a benign and transient dermatosis. The etiology of

TNPM is unknown and it does not require any medical treatment (3, 4).

CASE REPORT

This white, male infant was born after a normal term pregnancy with a birth weight of 2880 gr. No history of exposure to maternal infection or intake of drugs has been described. 23 hours after birth, he was hospitalised because of skin lesions and jaundice present since birth. He was the first child of the family and the family history was without any special problem. On physical examination the vital signs were as follows: axillary temperature= 36.3, respiratory rate= 46/min, heart rate= 136/min, weight= 2900 gr, length= 53 cm, head circumference= 35.5 cm. sucking (+), grasping (+), Moro (+). Fundoscopic examination was normal. No anomalies have been observed. The skin examination revealed 20 elevated, hyperpigmented, ruptured vesiculopustular lesions, 2-8 mm in diameter. These lesions were located at the right lower eye lid, back, axillary region, hands and feet, gluteus region and extremities (Fig. 1). The skin appeared yellowish. The anterior fontanel measured 2.5 x 2.5 cm. Examination of the chest, lungs and heart revealed no pathology. The liver was palpable 3 cm below the costal margin. The umbilicus appeared normal. The genital area was apparent male and normal. The four extremities moved well and approximately symmetrically. On



Fig. 1: Vesiculopustular lesions of TNPM

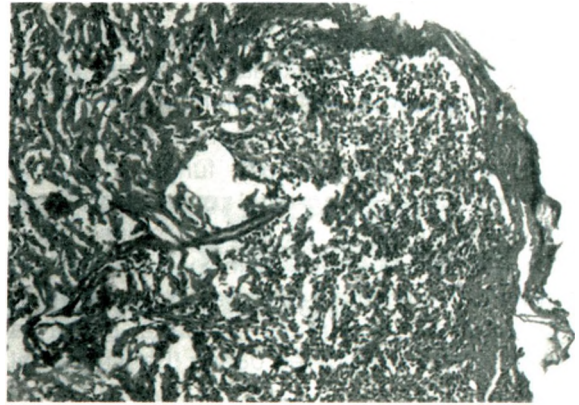


Fig. 2: Histopathologic examination: subcorneal collection of polymorphic neutrophils and a few eosinophils and lymphocytes.

DISCUSSION

TNPM should be considered, when skin lesions exist at birth, although it is rarely observed in white neonates. Three types of lesions might be recognised in TNPM: 1. rapidly-disappearing superficial pustules, 2. ruptured pustules with central hyperpigmented macules, 3. hyperpigmented macules. These types of lesions may appear separately or altogether. The number of lesions may vary from a few to many. While the pustules are usually apparent only during the first days of life and generally rupture in with two-three days, they leave macules, which persist for up to three months (3). The macules are predominantly located under the chin and the nape of the neck, and less frequently on the forehead, upper back, scalp, trunk, extremities, palms and soles. During the active phase on histopathologic examination, vesicular pustular lesions show thickening of the stratum corneum and subcorneal collections of polymorphic neutrophils and a few eosinophils. The macules are characterized by increased melanisation of edipermal cells (1, 2, 5).

The diagnosis of pustular dermatosis is usually based on clinical findings. Rarely, the appearance of TNPM legions may suggest another type of vesicular eruption. Then smears from skin lesions and bacterial cultures are imperative for the differential diagnosis of transient and infectious neonatal skin disorder. Skin infection can be detected fast by gram stain and definitively by bacterial culture method (1,2,4-6).

hospitalisation, phototherapy was applied due to hyperbilirubinemia (Tot. Bil.= 14.24 mg/dl, Dir. Bil.= 0.47 mg/dl). Cultures from the skin lesions and blood samples were obtained. On the postnatal second day a skin punch biopsy was performed. The biopsy specimens were stained with hematoxylin and eosin. The results showed subcorneal pustule formation in the epidermis and erosion in the areas adjacent to the pustules. Generally in the areas of erosion, polymorphic neutrophil, lesser eosinophil and lymphocyte containing inflammatory infiltration reached the hypodermis. The infiltrate was fused into the connective tissue cells (Fig. 2). No bacteria grew on the culture taken from the pustular specimen and the blood.

Most pustules disappeared within three to four days without recurrence, leaving behind a brown crust, which persisted for a few weeks if not actively removed by scratching.

The most common causes of infectious pustular skin lesions include bacterial infections, which may be septicemic (with *Listeria* the leading cause) or initially localized (staphylococcus); viral infections (varicella, herpes); fungal infections i.e. candidiasis (congenital or neonatal) or *Malassezia furfur*; or parasitic (scabies) (7,8). Other benign neonatal skin conditions (i.e. erythema toxicum neonatorum (ETN), acropustulosis of infancy, miliaria profunda) should be also considered during the differential diagnosis (6,9).

In our case, negative bacterial culture and typical skin biopsy results confirmed the diagnosis of TNPM. Pustular lesions turned into hyperpigmented macules on the third postnatal day. Because the duration of macules might exceed three months (3), the patient was discharged from hospital and invited for routine outpatient clinic examinations.

This case was presented to show the importance of differentiating this type of benign transient dermatosis from life-threatening pyoderma. The recognition of transient neonatal skin disease may spare the healthy neonate from extensive sepsis work-up, antibiotic therapy, and prolonged hospitalisation. Explaining to the parents, the transient nature of pustular melanosis and the rare possibility of the recurrence of the pustules will help to prevent unnecessary anxiety.

REFERENCES

1. Ferrandiz C, Coroleu W, Ribera M, Lorenzo JC, Natal A. Sterile transient neonatal pustular melanosis is a precocious form of erythema toxicum neonatorum. *Dermatology* 1992; 185: 18-22.
2. Merlob P, Metzker A. Transient neonatal pustular melanosis. *Am J Dis Child* 1982; 136: 521-522.
3. Schacher L, Press S. Vesicular, bullous and pustular disorders in infancy and childhood. *Ped Clin North Am* 1983; 30: 609-629.
4. Simon MW. Transient neonatal pustular melanosis: an uncommon rash (letter). *Am Fam Phys* 1995; 51 (suppl 6): 1401.
5. Lucky AW, McGuire JS. Infantile acropustulosis with eosinophilic pustules. *J Ped* 1982; 100: 428-429.
6. Ramamurthy RS, Reveri M, Esterly NB, Fretzin DF, Pildes RS. Transient neonatal pustular melanosis. *J Ped* 1976; 88: 831-836.
7. Moisson YF, Wallach D. Les dermatoses pustuleuses de la periode neonatale. *Ann Pediatr* 1992; 39: 397-406.
8. Brunkes A, Wallach D. Neonatal pustular disease. *Ann Dermatol Venereol* 1999; 126: 950-956.
9. Laude TA. Approach to dermatologic disorders in black children. *Semin Dermatol* 1995; 14: 14-21.