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Hypernatremia and miliaria crystallina: a rare association

Hipernatremi ve milarya kristalina: nadir bir ilişki

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Abstract
Miliaria crystallina is a common, transient and self-limited cutaneous disorder, caused by blockage within the eccrine sweat duct. Herein, we report a neonatal case with miliaria crystallina developed during treatment of severe hypernatremic dehydration. The effective treatment and prevention of miliaria crystallina is to avoid further sweating. On the basis of the presented patient, we consider that miliaria crystallina may be rarely developed in newborns with severe hypernatremia but this condition is transient and self-limited.

Key words: Hypernatremia, miliaria crystalline, newborn, treatment.

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Öz
Milarya kristalina, ekrin ter kanalındaki tıkanıklığın neden olduğu yaygın, geçici ve kendi kendini sınırlayan bir kutanöz bozukluktur. Burada, ağır hipernatremik dehidratasyon tedavisi sırasında milarya kristalina gelişen bir yenidoğan olgusu sunulmaktadır. Milarya kristalinanın önlem ve tedavisi fazla terlemekten kaçınmaktır. Bu hasta sunumuyla, milarya kristalinanın ağır hipernatremisi olan yenidoğanlarda nadiren gelişebileceğini, ancak bu durumun geçici ve kendi kendini sınırlandırdığını düşünüyoruz.

Anahtar kelimeler: Hipernatremi, milarya krsitalina, yenidoğan, tedavi.

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Introduction

Miliaria is a common, transient cutaneous disorder triggered by hot and humid conditions and caused by blockage within the eccrine sweat duct. There are three main types of miliaria: crystallina, rubra, and profunda [1, 2]. Miliaria crystallina (MC) is a superficial form of miliaria when sweat glands are obstructed within the stratum corneum [1]. Although there are few adult cases of MC associated with hypernatremia, there is only one neonatal case of MC associated with hypernatremic dehydration (HD) in the literature [3-5]. Herein, we report a second neonatal case with MC developed during treatment of severe HD.

Case report

A female term baby, large for gestational age with birth weight of 4150 gr was born to a second gravida, 27-year-old mother by cesarean section. When she was 13-day-old, she was brought to the emergency department with complaints of severe weight loss and dehydration. There were no any features in her pre/perinatal or postnatal history except for poor sucking. Also, no history of maternal or neonatal drug use and infection was reported. In her physical examination, general status was as moderate-good; consciousness: clear; restless; respiratory rate: 56/min; cardiac heart beat: 144/min; blood pressure: 64/44 mmHg; and body temperature: 36.9°C. She had a body weight of 2900 gr (10-25p, 30% weight loss), a

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height of 50 cm (50-75p), and a head circumference of 34 cm (50-75p). She had severe decreased skin turgor, dry mucous membranes, sunken anterior fontanelle and eyes. Other physical systems' signs were normal. The laboratory tests were as follows: blood urea nitrogen 336 mg/dl, serum creatinine 3.1 mg/dl, serum Na 187 mEq/L, glucose 137 mg/dl, urine density 1024 and fractional excretion of sodium (FeNa) 0.8%, and plasma osmolality 437 mOsm/kg/H_2O. Other biochemical parameters, complete blood count, blood gases, cranial and urinary ultrasound examinations, and acute phase reactants for infection were normal. The patient was diagnosed with severe hypernatremic dehydration and acute renal failure. Intravenous electrolyte and fluid treatment was started appropriately and promptly. A few days later, a gradual decrease in serum sodium and creatinine levels was observed. On her clinical follow-up, no any seizure activity was observed, and cranial magnetic resonance imaging was normal. When she was 18-day-old, small, clear, superficial vesicles began to appear on her fore-head and neck, covering her whole body the following day (Fig. 1a and b). Skin biopsy was performed to the patient with negative serology for TORCH (toxoplasmosis, other agents, rubella, cytomegalovirus, herpes simplex) infections. Histopathologic examination revealed superficial perivascular lymphocytes infiltration, edema and several red blood cells in the dermis. The vesicles resolved spontaneously 3-4 days later, along with resolution of the hypernatremia (Fig. 2). On the basis of typical cutaneous lesions, the patient was diagnosed with MC. Informed consent was obtained from the patient's father.

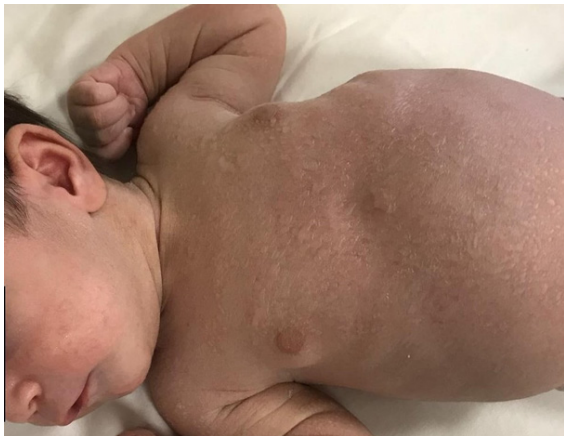

Figure 1. (a) Widespread lesions on the trunk

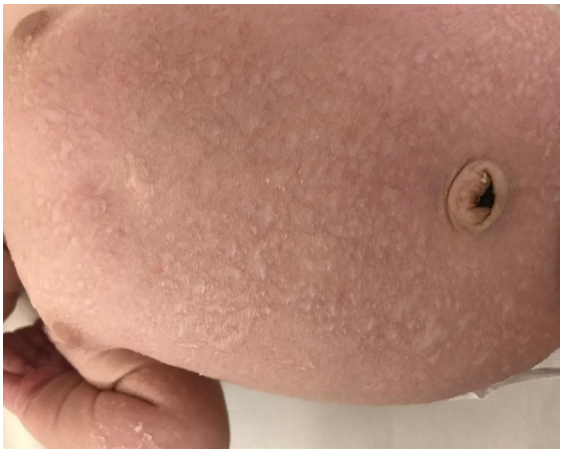

Figure 1. (b) Clear, vesicular lesions of miliaria crystallina

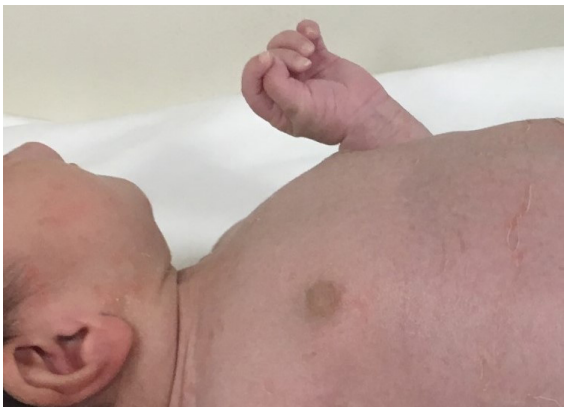

Figure 2. Complete resolution of the cutaneous vesicles a few days later

Discussion

It has been reported that MC develops because of a transient poral closure of the sweat duct opening, resulting in obstruction of free flow of eccrine sweat and retention in a vesicle below the skin surface [6]. Environmental heat, humidity and adrenergic/cholinergic drugs may be related to MC [1, 2, 6]. Clinical appearance of MC has translucent vesicle 1-2 mm in diameter, with an appearance of "drops of water", without an inflammatory halo [6]. Diseases of vesicular and pustular disorders such as herpes simplex, varicella, erythema toxicum neonatorum (ETN), neonatal pustular melanosis, infantile acropustulosis, and staphylococcal infectious are important for the differential diagnosis of MC [7-9]. Erythema toxicum neonatorum presents with multiple erythematous macules and papules that rapidly progress to pustules on an erythematous base,

and the rash usually resolves in five to seven days. Histopatholgical findings of this disease are of numerous eosinophils and occasional neutrophils. Neonatal pustular melanosis is less common than ETN and consists of three types of lesions. The first one is small pustules on a non-erythematous base; these usually are present at birth. The second one is erythematous to hyper pigmented macules with a surrounding collaret of scale; these develop as the pustules rupture and may persist for weeks to months. The third one is hyper pigmented macules that gradually fade over several weeks to months. Lesions in different stages may be present at the same time and microscopic examination of lesions demonstrates numerous neutrophils and, in contrast with ETN, rare eosinophils. Infantile acropustulosis is a benign vesiculopustular condition and characterized by recurrent crops of intensely pruritic vesiculopustules, mainly on the palms and soles but sometimes involving the dorsal aspect of the hands and feet and the limbs. Skin biopsy of this disease demonstrates a sub corneal pustule filled with neutrophils and eosinophils. Miliaria crystallina is characterized by small, thin-walled vesicles resembling dewdrops without inflammation and histopathological examination demonstrates sparse squamous cells and lymphocytes. The diagnosis of all these vesicular and pustular disorders is usually made based on the clinical appearance [9].

The most important differences include that, in MC, the sweat in the blisters is not colored, but clear like water, therefore; the color and the form of the lesions allow a definite clinical diagnosed [7]. The presented patient had similar cutaneous lesions of MC, however; she had no history of drug use, fever, environmental heat or humidity. In addition, TORCH serology and acute phase reactants for infectious are negatively for her, and skin biopsy demonstrates lymphocytes infiltration, therefore; the patient was diagnosed with MC. The only effective treatment and prevention is to avoid further sweating [7].

In 2012, Engür et al. [5] reported for the first time that high levels of sodium in sweat in hypernatremic dehydration might have caused eccrine duct damage, predisposing to MC. Afterward, Chao CT reported that findings of MC were observed in a male adult patient with hypernatremia, and that the vesicles were resolved spontaneously days later, along with resolution of the hypernatremia [3]. Miliaria crystallina was also developed in our neonatal case with severe hypernatremic dehydration during therapy, and the lesions were also resolved spontaneously a few days later, along with resolution of the hypernatremia. No any local or systemic treatment procedure or any other therapy modalities were used for MC in presented patient.

In conclusion, we agree with Engür and co-workers [5] that high levels of sodium in sweat due to hypernatremic dehydration may induced eccrine ductal damage. We consider that this condition is rarely occurred and that MC, transient and self-limited, may developed in newborns with severe hypernatremia.

Conflict of interest: The authors declare that there is no any conflict of interest.

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Informed consent: Written informed consent was obtained from the patient.

Author Contributions

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Analysis: Ö.M.A.Ö., T.A., C.Ç., H.E.
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Critical Review: Ö.M.A.Ö., T.A., C.Ç., H.E.
All co-authors take full responsibility for all aspects of the study and the final manuscript.