

Congenital Erosive and Vesicular Dermatitis (CEVD): A Rare Case and Review of the Literature

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Abstract

Congenital erosive and vesicular dermatitis (CEVD) is a rare neonatal dermatosis characterized by erosive and vesicular lesions that develop at or shortly after birth and heal with reticulated scarring. The presented case highlights a localized erosive-vesicular eruption followed by cribriform scarring, clinically suggestive of CEVD. Recognition of the CEVD phenotype is important while maintaining a broad differential diagnosis that includes congenital infections, trauma, anetoderma of prematurity, and inherited blistering disorders.

Introduction

Congenital erosive and vesicular dermatitis (CEVD) is a rare neonatal condition characterized by erosive or vesicular lesions present at birth or developing shortly thereafter, which typically heal with reticulated scarring (1–4). Its pathogenesis remains unclear, though it is believed to represent an acquired in utero condition rather than a genetic disorder (1,5,6). Proposed mechanisms include intrauterine infection (e.g., herpetic infections), amniotic adhesions, or developmental defects in foetal skin healing (2,7,8).

Case presentation

A 3-month-old male infant was referred to our paediatric dermatology clinic for evaluation of several vesicular and erosive lesions located on the scrotum and lower abdomen. Shortly after birth, an erosion covered by some yellow vesicles was observed on the scrotum (figure 1). However, no swab was taken to confirm a herpes infection. Around the same period, a spontaneous abdominal erosion without preceding blister formation was also noted by the parents (figure 2). Both lesions were treated with chlorhexidine and healed within a few days. Physical examination revealed a linear, vertically oriented, non-inflammatory, atrophic macule on the lower abdomen (figure 3). The scrotal skin was completely healed, with no residual erythema or active lesions. The prenatal, perinatal, and postnatal courses were unremarkable. The

infant was born at term by vaginal delivery. There was no birth or instrumentational trauma during delivery. Growth and neurodevelopment were normal. There was no family history of dermatologic disorders. The patient's 3-year-old brother had a subtle hypopigmented linear scar on his hand, reportedly secondary to a healed infantile erosion, and a history of self-limiting autoimmune neutropenia (figure 4). Maternal serologic testing demonstrated positive IgG antibodies for cytomegalovirus (CMV), while toxoplasmosis serology was negative. A previous miscarriage had been reported, although no amniotic adhesions were observed on ultrasound. Maternal Herpes simplex virus (HSV) testing was negative.

Discussion

CEVD is present at birth and is frequently reported in premature infants, with erosions, vesicles, and bullae may affect a large body surface area (2). Lesions present in the neonatal period heal within 10 days to 3 months. The clinical presentation of a linear, reticulated, non-inflammatory macule following the early erosive lesions is characteristic, but should be interpreted in the context of the clinical course and exclusion of important mimics (1,3,4,9). The precise pathogenesis of CEVD remains uncertain; current evidence favours an acquired intrauterine process rather than a hereditary disorder (5,6,10). Proposed mechanisms include intrauterine infections such as Herpes simplex virus or *Candida albicans*,



FIGURE 1: Initial vesicular lesion with an underlying erosion observed on the scrotum shortly after birth.



FIGURE 2: A subtle erosive lesion was noted on the right thorax and abdomen shortly after birth, without preceding trauma.



FIGURE 3: Around 3 months of age a cribriform scar developed at the sites of the erosive lesions previously noted on the thorax and abdomen.



FIGURE 4: A similar cribriform scar on the right third digit of the patient's brother.

amniotic adhesions, or disturbances in foetal wound healing (2,7,11,12).

Approximately one-third of reported CEVD cases demonstrate features suggestive of intrauterine infection, such as chorioamnionitis or discoloured amniotic fluid (4,9). CEVD does not follow a Mendelian pattern of inheritance, and genetic analyses for *TP63* and *POCRN*, have not established a causative variant (5, 14, 16). Familial recurrence is rare (9, 14); however, the subtle scar observed in the sibling in the present report is nonspecific and cannot be taken as evidence of familial CEVD. Although cases in full-term infant have been reported, CEVD is strongly associated with prematurity (17).

The differential diagnosis of CEVD includes intrauterine or congenital infections (e.g., herpes simplex virus, varicella-zoster virus, bacterial infections), congenital or perinatal trauma, anetoderma of prematurity, and a range of developmental, inflammatory, and genetic dermatoses such as epidermolysis bullosa, aplasia cutis congenita, focal dermal hypoplasia or aplasia, incontinentia pigmenti, dyskeratosis congenita, congenital ichthyosis, linear scleroderma, popliteal pterygium syndrome, atrophoderma of Pasini and Pierini, and vascular or vasospastic disorders including acrodermatitis chronica atrophicans and poikiloderma congenitale (3,4,6,12,15-17). Accordingly, a thorough perinatal workup comprising targeted infectious testing, bacterial and fungal cultures when clinically indicated, and, when appropriate, histopathologic evaluation may assist in ruling out alternative diagnoses (4,8,12). In a premature infant, anetoderma of prematurity would warrant particular consideration when an atrophic lesion is already visible early in the clinical course.

Since the supple, reticulated scarring pattern is considered highly characteristic, the diagnosis is often established clinically after relevant alternative diagnoses have been excluded (1,3,9). Histopathologic findings early in the disease process of CEVD can include epidermal necrosis, subepidermal vesiculation, and an eroded epidermis (3). Older lesions demonstrate scar formation, decreased hair follicles, and absent eccrine glands. At subsequent follow-up visits, associated findings should be carefully assessed, including signs of hyperthermia, alopecia, nail abnormalities, ocular involvement, neurological development, and hearing function (18,19). Lesions resolve rapidly during the first month, and further episodes are uncommon. (1,2,9).

Our patient presented with atypical clinical features, including postnatal recognition of lesions (reported in only a few cases) and the limited skin involvement, suggesting a mild disease expression (15,17). Also a sibling that had similar scars is rare and only described in two other cases (4,10).

Conclusion

This case illustrates a localized presentation of CEVD. The observation of a similar lesion in a sibling may reflect shared perinatal or environmental influences rather than a hereditary predisposition. Recognizing CEVD is essential to distinguish it from congenital infections and genetic blistering disorders, thereby avoiding unnecessary investigations and providing appropriate reassurance to families regarding its typically benign course and favourable prognosis.

Statement

Informed consent for publication of this case report and the accompanying clinical photographs was obtained.

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