Synchronous skin lesions in mother and baby with neonatal lupus erythematosus

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Presentation

A 27-year-old woman, G1P1, gave birth to a baby girl with a widespread macular rash (Fig. 1a) with mild thrombocytopenia and hepatitis. There was no evidence of heart block or heart failure. The baby and mother were positive for serum anti-Ro60, anti-Ro52 and anti-La autoantibodies.

The mother did not complain of any symptoms of a systemic autoimmune rheumatic disease (SARD) including sicca symptoms. She did, however, report intermittent skin lesions, with one appearing on the right lateral border of her right foot (Fig. 1b). Biopsy of the baby's lesions at 6 weeks confirmed cutaneous neonatal lupus erythematosus (NLE) which showed lymphocytic dermal interface inflammation (Fig. 1c). Remarkably, biopsy of the mother's lesions bore striking resemblance histologically (Fig. 1d). Maternal and neonatal lesions improved with maternal hydroxychloroquine and breastfeeding (Fig. 1e, f).

Discussion

NLE is a rare disease caused by the in utero passive transfer of pathogenic of Ro60, Ro52 and La autoantibodies. It may cause a constellation of symptoms from cutaneous lesions, cardiac block or hepatitis. Mothers of NLE babies are either asymptomatic or have SARD that includes systemic lupus erythematosus [1]. The autoantibodies are thought to be directly pathogenic [1–3], though it is likely a multifactorial process that relies on environmental triggers as well such as ultraviolet light. This may explain why there was a large phenotypic difference in mother and baby despite the same pathogenic autoantibodies. It is unclear which antibody, in particular, is responsible for cutaneous manifestations [1]. Certainly, pregnancy itself may be a contributing agent in the modulation of lesion severity. In a study of new-onset SLE in pregnant and non-pregnant women, however, there appeared not to be a difference in frequency of cutaneous manifestations [4].

Mothers who give birth to children with NLE, though do not have any overt SARD, need ongoing follow-up for the development of symptoms which may take 10 or more years

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Fig. 1 Synchronous skin lesions demonstrating annular, coalescing lesions in the baby (a) and a non-painful, poorly-demarcated plaque lesion in the mother (b). Associated haematoxylin and eosin stain of the lesions are shown in c and d respectively, taken at $\times 200$ power. These demonstrate patchy interface dermatitis, vacuolar damage and

lymphocytic exocytosis (c and d) and additional dermal lymphocytic perivascular inflammation (d). Direct immunofluorescence was negative (c and d). Progress images at 12 months (6 months into hydroxychloroquine treatment) demonstrated near resolution of lesions in the baby (e) and complete resolution in the mother (f)

to develop [5]. Cutaneous NLE recurrence rate in subsequent pregnancies may occur in up to 1 in 3 [1]. Understanding the pathogenic autoantibody role will help to guide future therapies that may be of use in ameliorating or preventing the manifestations of NLE.

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Declarations

Ethics Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflicts of interest The authors declare no competing interests.

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