QUIZ SECTION

Nasal Plaque with Central Eschar in an Infant: A Quiz

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A 2-month-old otherwise healthy male infant presented with a rapidly progressive erythematous plaque on the nose (Fig. 1A) for 10 days. He was treated with oral cefaclor and topical mupirocin while the lesion kept progressing. Physical examination confirmed an atrophic erythematous nasal plaque with central eschar and a scaly erythematous patch on the frontal angle. There was no fever, lymphadenopathy or hepatosplenomegaly. Family history was unremarkable, and maternal prenatal syphilis serology was negative. A punch biopsy was performed on the frontal lesion for histopathological examination and microbiological culture (Fig. 1B).

What is your diagnosis?
1: Mucormycosis
2: Insect bite dermatitis
3: Neonatal lupus erythematosus
4: Extranodal Natural Killer/T (NK/T)- cell lymphoma, nasal type
See next page for answer.

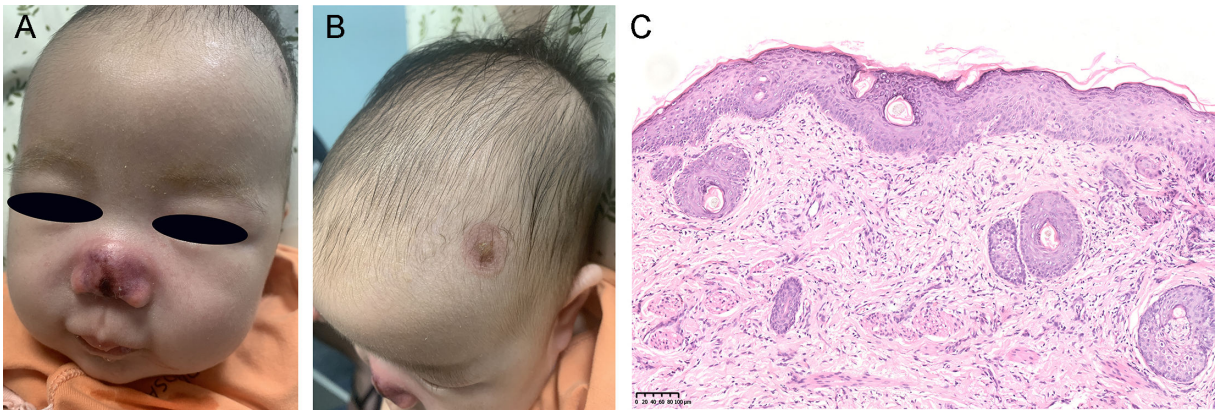

Fig. 1. (A) An atrophic erythematous nasal plaque with central eschar. (B) An erythematous patch on the left frontal angle with a central crust. (C) Histopathology from a punch biopsy demonstrated epidermal atrophy and superficial and deep periadnexal sparse inflammation infiltration, mild vacuolar interface and scattered lymphocytes in the superficial dermis (haematoxylin-eosin staining, original magnification × 200).

ANSWERS TO QUIZ

Nasal Plaque with Central Eschar in an Infant: A Commentary

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Diagnosis: **Neonatal Lupus Erythematosus**

Histopathology demonstrated epidermal atrophy and superficial and deep periadnexal sparse inflammation infiltration, a mild vacuolar interface and change with scattered lymphocytes in the superficial dermis (Fig. 1C), and periadnexal small numbers of lymphocytes and plasma cells in the deep dermis (Fig. 2A). Mucin deposition was present in the papillary dermis on Alcian blue staining (Fig. 2B). Both the infant and mother were positive for Sjögren syndrome–related antigen A (SSA/Ro) (+++) and Sjögren syndrome–related antigen B (SSB/La) (+++) antibodies, supporting a diagnosis of neonatal lupus erythematosus (NLE).

Laboratory studies showed neutropenia (absolute neutrophil count, ANC 0.95×10^9/L; reference range 3.4–4.8×10^9/L) and mildly elevated aspartate aminotransferase (51.81 U/L; reference range 5–34 U/L). Tissue microbial stains (Gram, acid-fast, periodic acid-Schiff) were negative. Other liver and renal function tests, syphilis serology, tissue cultures and electrocardiogram were normal. After 1 week of treatment with 0.1% mometasone furoate cream, the skin lesions resolved, leaving only hyperpigmentation and a mild atrophic scar.

NLE is a rare immune-mediated disorder with an estimated incidence of 1 in 20,000 live births (1). It results from the transplacental passage of maternal anti-SSA/Ro and anti-SSB/La antibodies. Approximately 1%–2% of mothers carrying these antibodies give birth to infants with NLE (1). While women with autoimmune diseases such as systemic lupus erythematosus, Sjögren's syndrome, mixed connective tissue disease or undifferentiated connective tissue disease are at increased risk, the mother in this case was asymptomatic and had not been previously diagnosed with any autoimmune disease. Given her serological profile, she was referred to rheumatology for further evaluation (1).

Cutaneous lesions of NLE may be present at birth but more commonly develop 4–6 weeks after sun exposure (2). The rash most frequently involves the face, particularly the periorbital region, as well as the perioral, zygomatic, temporal and nasal areas (1, 2). The scalp and neck may also be affected, with less frequent truncal or extremity lesions (1). Typical lesions consist of annular or polycyclic erythematous plaques with fine scaling. Targetoid forms with central clearing and discoid-like forms have also been described (1, 3). Although erosive, atrophic, bullous and papulosquamous morphologies have been reported – often presenting at or shortly after birth – an atrophic erythematous plaque with central necrosis is a rarely recognized manifestation of NLE (3–6). Weston et al. (7) described "thick crusted" lesions in 3 of 18 infants with NLE, though eschar was not explicitly mentioned. Such an escharotic lesion is uncommon in NLE and prone to misdiagnosis or delayed diagnosis. In this case, intense sun exposure may have contributed to lesion development. Consistent sun protection was advised, and both the atrophic scar and laboratory abnormalities resolved within 10 months.

Beyond cutaneous involvement, NLE can affect multiple organ systems, including cardiac, hepatobiliary, haematologic and neurologic systems, either in isolation or in combination. The most serious manifestation is congenital heart block (CHB), which carries a mortality rate as high as 20% (1, 4). Hepatobiliary involvement, seen in 10%–25% of infants, may include asymptomatic transaminase elevation, hepatomegaly or elevated gamma-glutamyl transferase (1). Hematologic abnormalities (10%–20%) include anaemia, thrombocytopenia and, less commonly, neutropenia or aplastic anaemia (1). Neurologic manifestations such as macrocephaly, with or without hydrocephalus, occur infrequently (1). The overall mortality of NLE is 5.6%, but it increases significantly with CHB (1, 4). Our case also exhibited neutropenia and elevated aminotransferases,

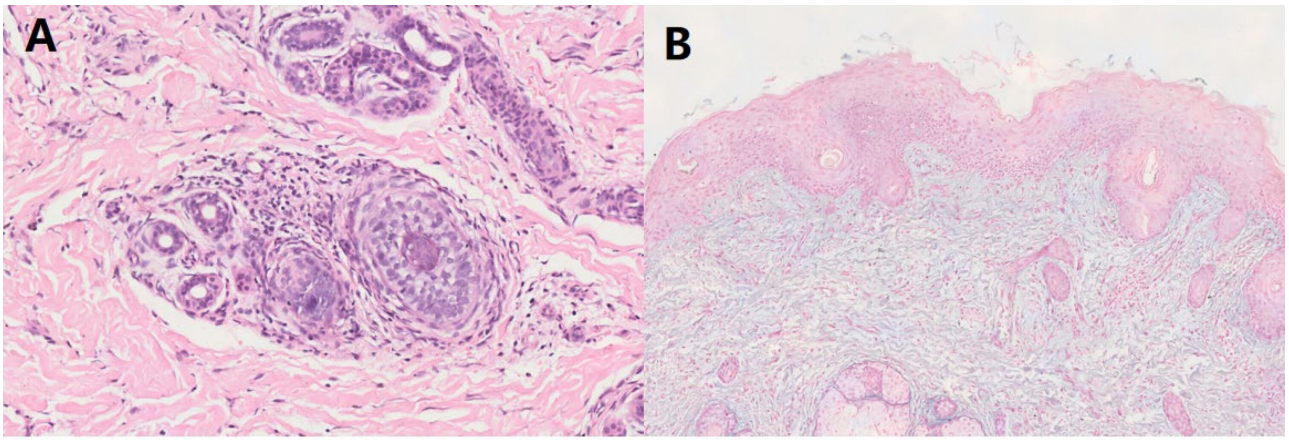

Fig 2. (A) Periadnexal small numbers of lymphocytes and plasma cells in the deep dermis (haematoxylin-eosin staining, original magnification × 400). (B) Alcian blue stain showed remarkable mucin in the papillary dermis (Alcian blue staining).

both of which were self-limited; no evidence of cardiac involvement was detected.

In the differential diagnosis of neonatal necrotic plaques, several entities warrant consideration. Cutaneous mucormycosis is a rare, angioinvasive fungal infection that generally arises following skin breakdown – often traumatic – and presents with necrotic eschars (8). Nasal involvement, which may indicate the rhino-orbito-cerebral form, represents a life-threatening concern in immunocompromised individuals, such as those with diabetes or haematologic malignancies (8). Diagnosis relies on histopathologic and mycologic culture; in this infant, however, both were negative. Insect bite reactions may simulate eschars due to excoriation and secondary infection, yet they typically heal spontaneously with hyperpigmentation over several weeks (9). Finally, extranodal NK/T-cell lymphoma, nasal type, is an aggressive Epstein-Barr virus-associated malignancy causing destructive midfacial necrosis (10). Importantly, it is a systemic disease with a poor prognosis, capable of disseminating to the skin, gastrointestinal tract and central nervous system (10), which distinguishes its clinical course from the typically self-limited nature of NLE.

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