Collodion-like membrane in a newborn exposed to infliximab

To the Editor: A 2-day-old black female newborn, born via cesarean section after premature rupture of membranes (34 weeks’ gestation), had a collodion membrane. Stellate cracking was present over the entire cutaneous surface (Fig 1). There were no nail abnormalities, bullae, ectropion, or eclabium. The mother was treated for severe psoriasis and psoriatic arthritis with a 7.5-mg/kg infusion of infliximab every 6 weeks throughout pregnancy, and for 6 months before conception. She took no other medications during her pregnancy. Newborn genetic screening, complete blood cell count, and comprehensive metabolic panel test results were within normal limits, and there was no history of consanguinity. There was no family history of bullous congenital ichthyosiform erythroderma or lamellar ichthyosis. Treatment was initiated with a petrolatum-based ointment (Aquaphor, Beiersdorf Inc, Wilton, CT). The collodion membrane shed completely and the skin normalized over 2 months. The skin was normal-appearing at age 1 year.

The mean age of onset of psoriasis is during the 20s and 30s, the peak childbearing years. Although the hormonal changes of pregnancy can improve psoriasis, approximately 20% worsen. Psoriatic arthritis, severe in 5% of patients, can be quite destructive. Inflammatory cytokines such as interleukin 17 and tumor necrosis factor (TNF)-alpha inhibit the T-helper cell type 17 axis inducing inflammation and defects in keratinocyte differentiation producing psoriatic plaques. TNF-alpha inhibitors, such as infliximab, are remarkably effective in controlling both psoriasis and psoriatic arthritis.

Infliximab, a complete chimeric monoclonal IgG1 antibody, is a potent anti-TNF-alpha biologic agent delivered by infusion. It is designated Food and Drugs Administration (FDA) category B because animal reproduction studies did not demonstrate a risk but well-controlled studies in human pregnancy have not been done. The Fc receptors on trophoblasts help to actively transport all biological agents with an IgG Fc region. Therefore, the transplacental passage of monoclonal antibodies such as infliximab and adalimumab is greater than in etanercept.

An FDA database review of 41 children born to mothers taking a TNF antagonist demonstrated congenital anomalies in 24 children (59%). Of the anomalies reported, 56% were part of the vertebral defects, anal atresia, cardiac defects, tracheoesophageal fistula, renal anomalies, and limb abnormalities (VACTERL) spectrum, but only 1 child was diagnosed with VACTERL. A retrospective analysis of pregnancy outcomes of 32 women exposed to anti-TNF-alpha inhibitors demonstrated no increased risk to the patients and fetuses, although the paucity of data precluded a recommendation for regular use in pregnancy.

Although collodion membrane associated with anti-TNF biologic agents has not been reported to our knowledge, there are several examples of paradoxical inducement of keratinization abnormalities. New-onset psoriasis has occurred in a series of patients with rheumatologic conditions treated with TNF-alpha inhibitors. The pathogenesis of these paradoxical reactions may be the result of overexpression of interferon because of the cross-regulation between TNF-alpha and interferon.
Perhaps a similar mechanism may explain the translucent, tight, and parchment paper–like “collodion membrane” in our patient after prenatal exposure to infliximab. Collodion membrane secondary to ichthyoses can be excluded because this child’s condition was self-limited. Considering potential side effects of alternative systemic agents, TNF-alfa inhibitors should remain in the armamentarium for pregnant psoriatic patients. Etanercept may be the safest of this class because of its lower level transplacental passage.

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Funding sources: None.

Disclosure: Dr Brodell is on the speakers bureau for Allergan, Galderma, PharmaDerm (a division of Fougera Pharmaceuticals Inc), and Veregen, a consultant/investigator/advisory board member for Galderma Laboratories LP; and has performed multicenter clinical trials for Genentech and Janssen Biotech Inc. Ms Offiah and Drs Campbell and Wyatt have no conflicts of interest.

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REFERENCES

http://dx.doi.org/10.1016/j.jaad.2014.01.856

Reticulated hyperpigmentation following chemotherapy for radiation-induced osteosarcoma

To the Editor: Chemotherapeutic agents have numerous cutaneous adverse effects, including hyperpigmentation, radiation recall, and extravasation injuries. Typically, hyperpigmentation reactions are either diffuse (eg, the “busulfan tan”) or localized (eg, streaking over veins in fluorouracil). We describe a patient that developed hyperpigmentation following chemotherapy for a radiation-induced osteosarcoma.

A 47-year-old African American man presented for management of a radiation-induced osteosarcoma. Intracranial meningioma was originally diagnosed and treated with a craniotomy in 1995, followed by resection of recurrent meningioma in 1996 and adjuvant radiation in 1999. In 2012, he presented with an enlarging right orbital rim mass that was treated with a craniotomy and orbitotomy. Pathology showed osteoblastic osteosarcoma, likely secondary to radiation therapy. His chemotherapy regimen included 2 months of Adriamycin (doxorubicin) and cisplatin, followed by methotrexate, ifosfamide, and etopoide. Seventy-two hours following his second dose of methotrexate, minimally pruritic cutaneous changes developed on his lower extremities, which spread to the extensor arms and trunk.

Physical examination demonstrated symmetric fine reticular and linear hyperpigmentation without excoriations on the dorsal ankles, medial arms, abdomen, and back, which on dermatoscopic exam appeared to be surrounded by a halo of erythematous macules (Fig 1, A). Punch biopsy demonstrated epidermal dysmaturity with numerous atypical keratinocytes and melanin deposition within the epidermis and stratum corneum (Fig 1, B). Given the mild inflammatory infiltrate and melanin deposition, we treated with 0.1% triamcinolone cream twice daily to reduce the inflammation and lighten the pigment by inhibiting melanocyte secretory function, similar to treatment of postinflammatory hyperpigmentation. The lesions resolved within 6 months.

Although a similar reticular, hyperpigmented pattern has not been reported in association with