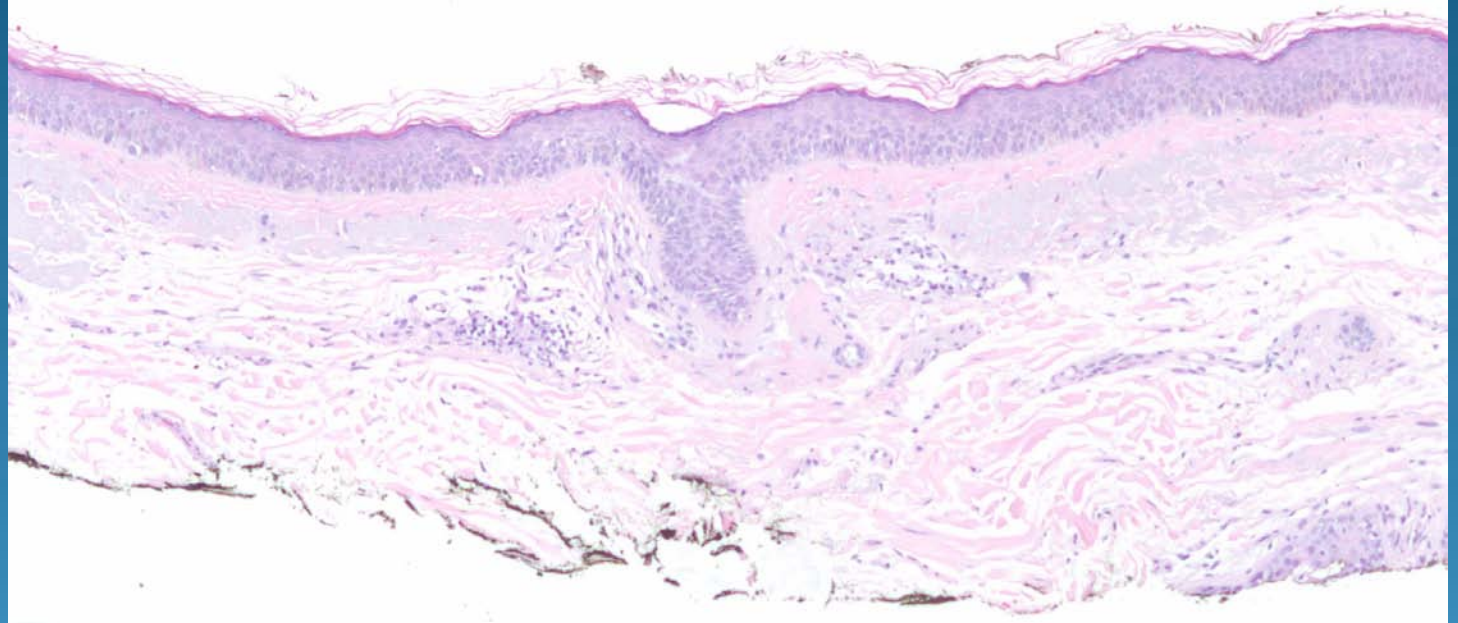
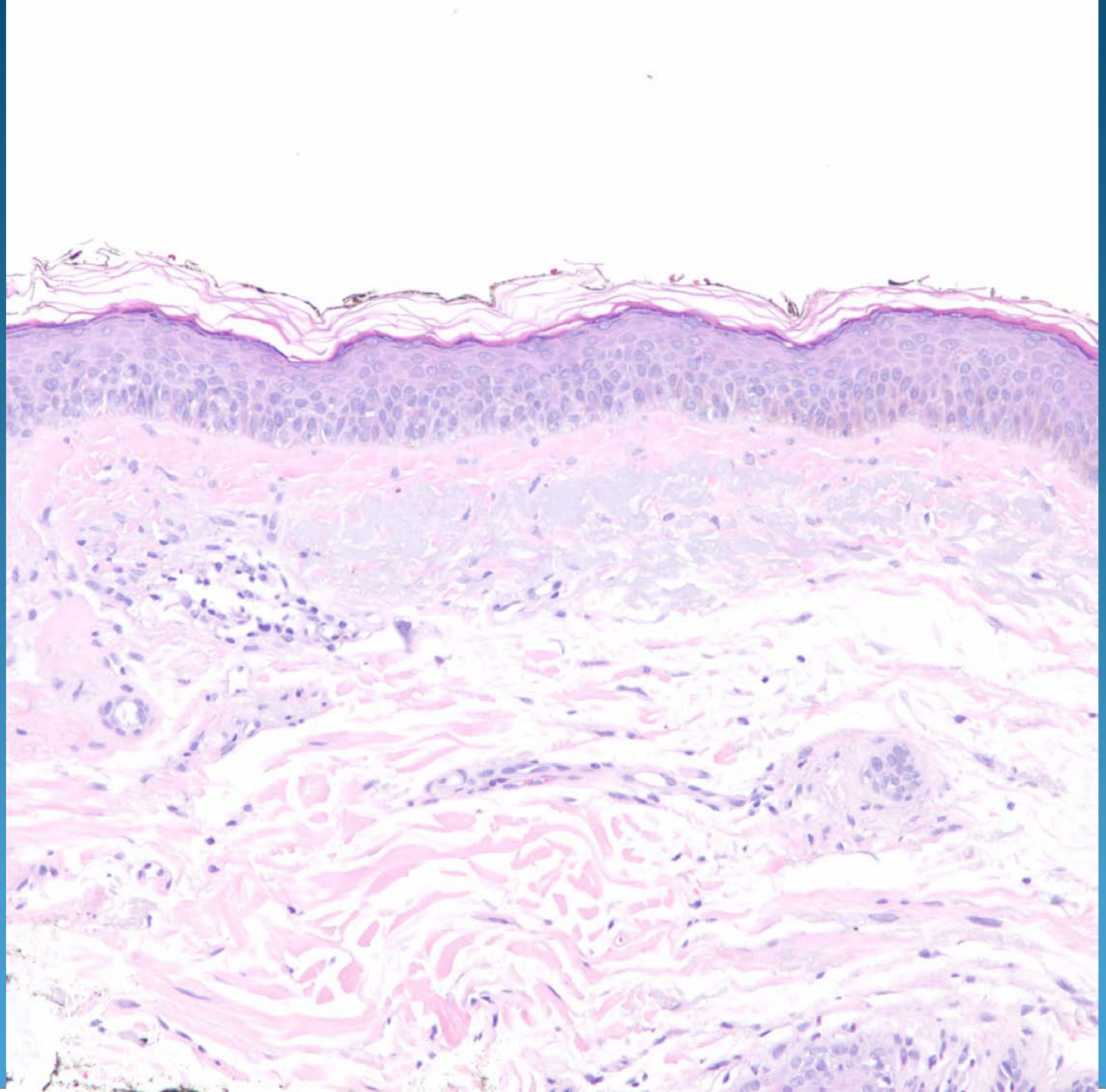
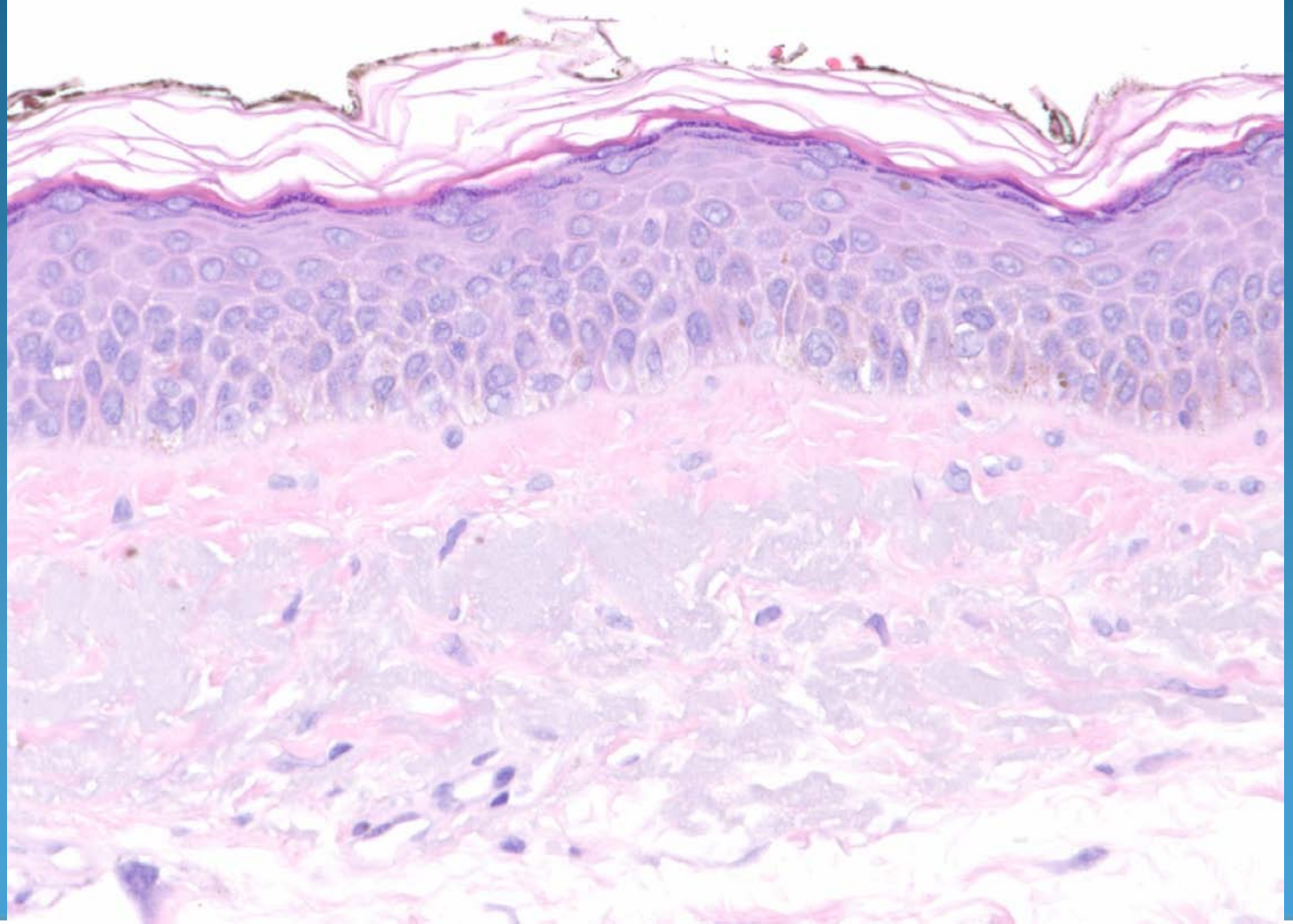
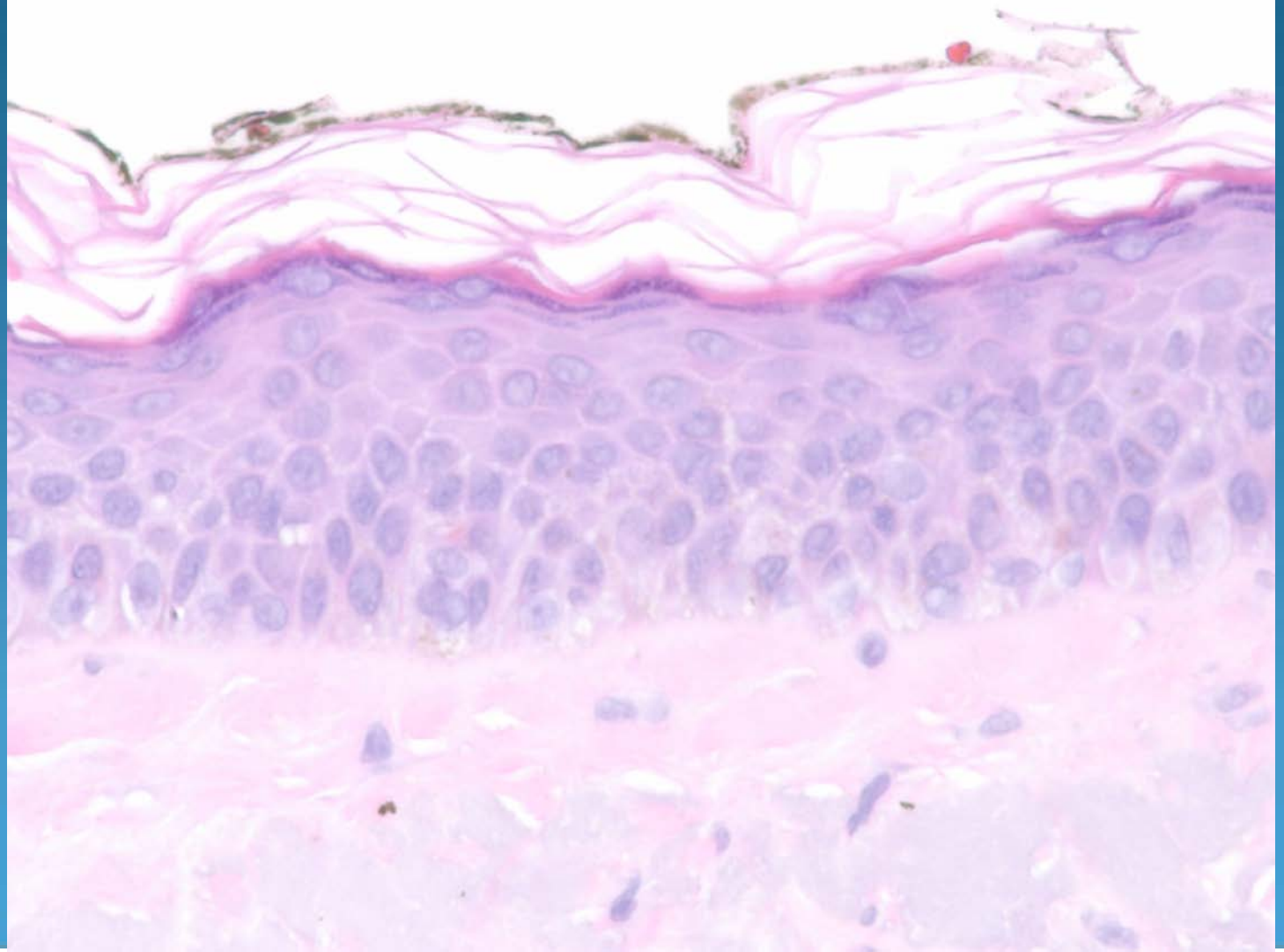
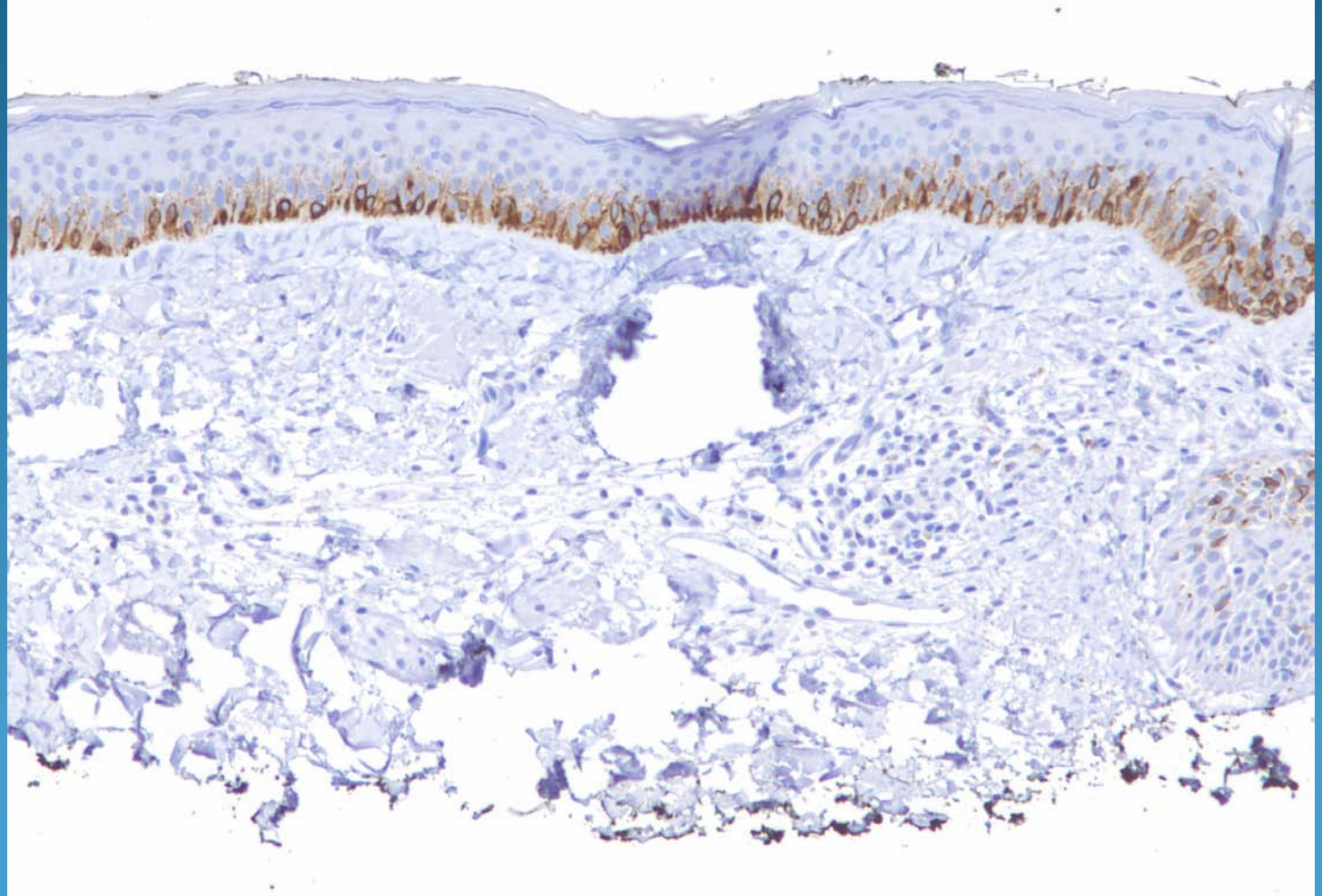








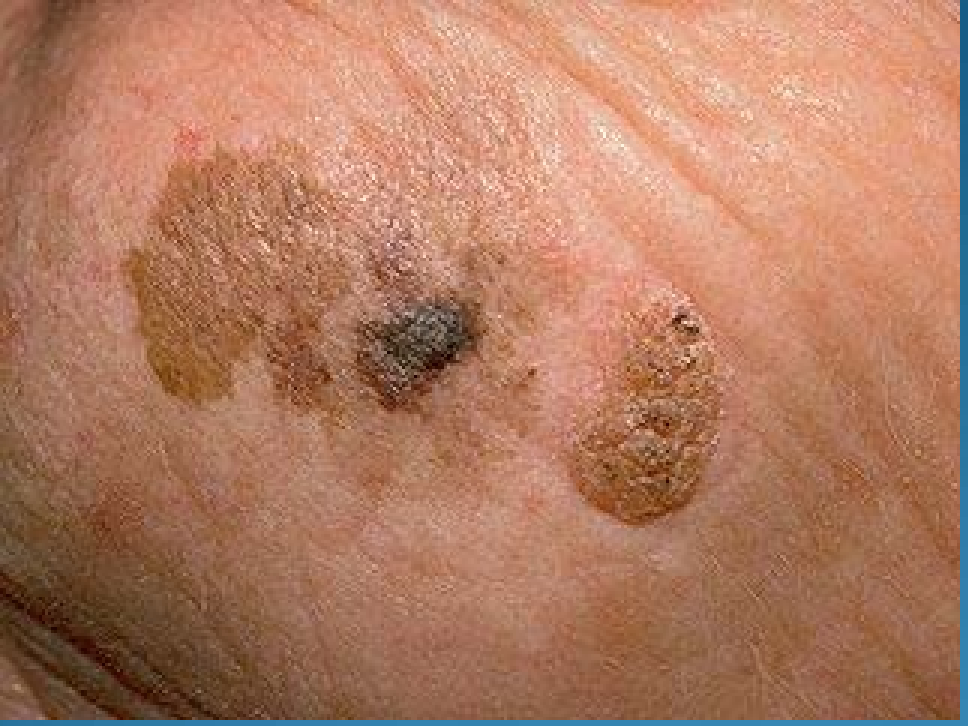
Melan-A

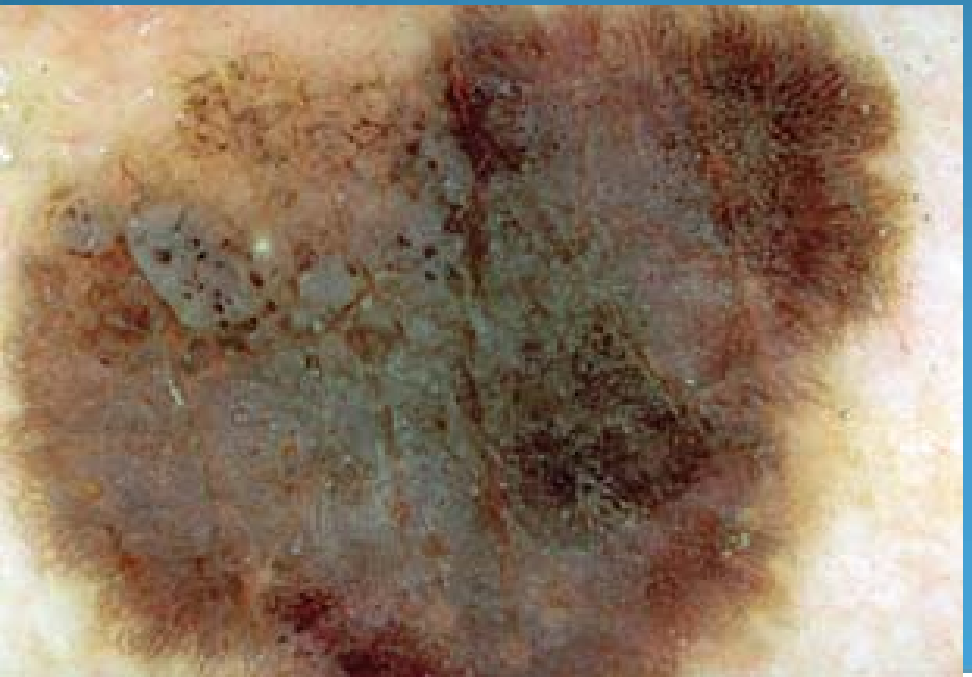


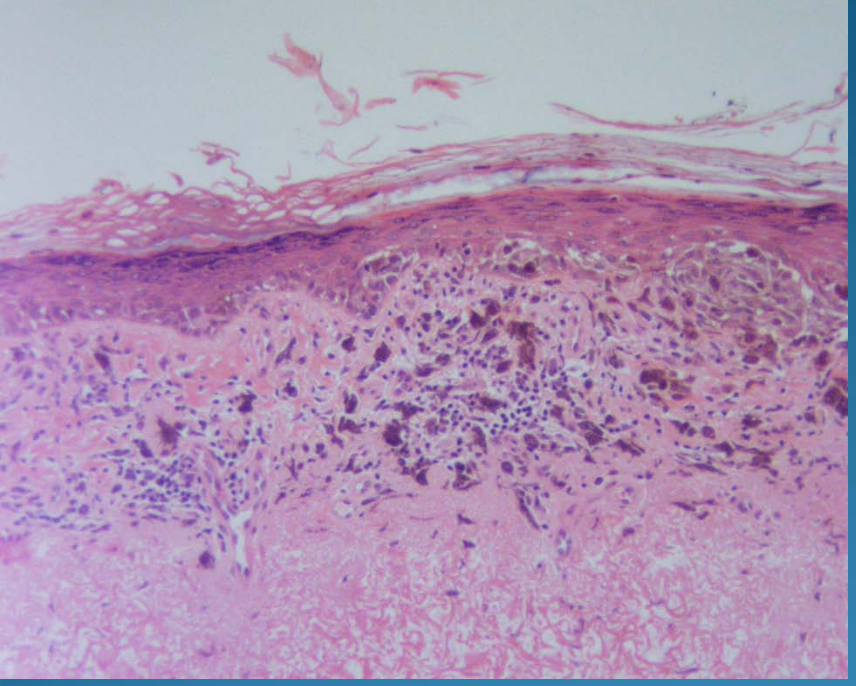
Amelanotic Melanoma in Situ: A histopathologic mimic of benign lesions

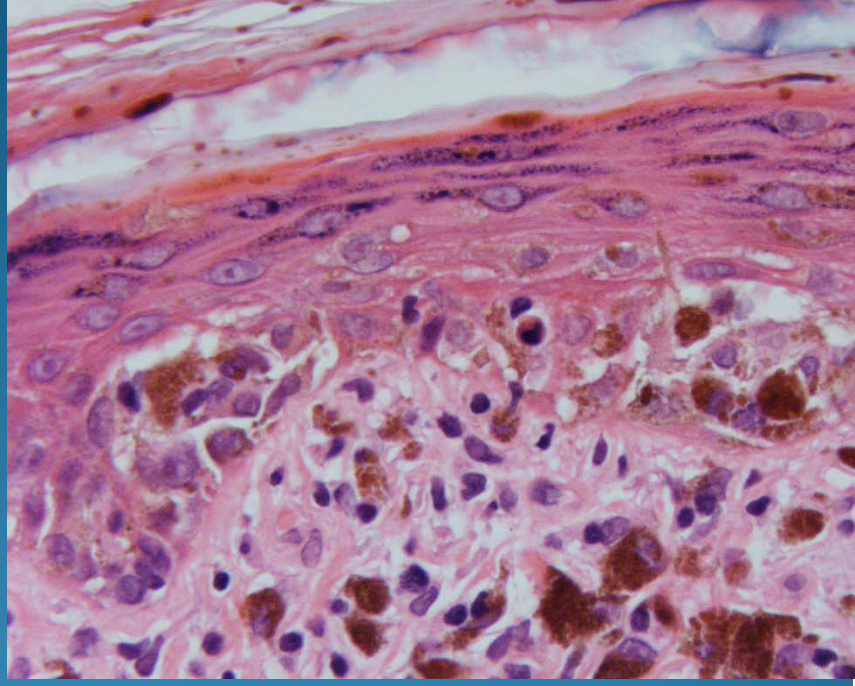
Tan BH and Shitabata PK

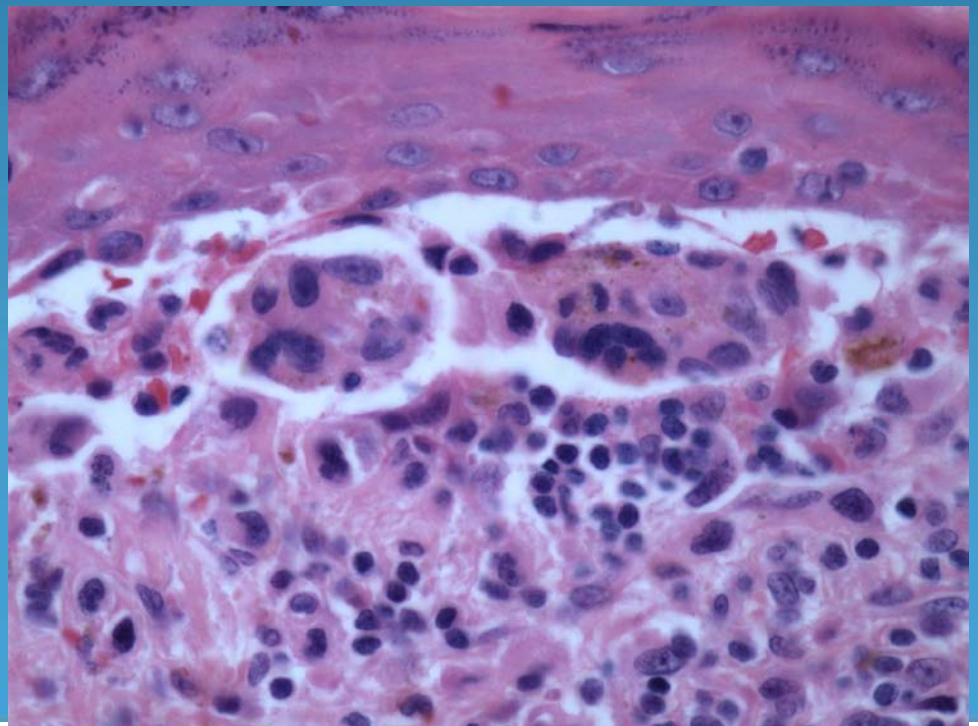
Presented at the International Society of Dermatopathology, March 5, 2009, San Francisco







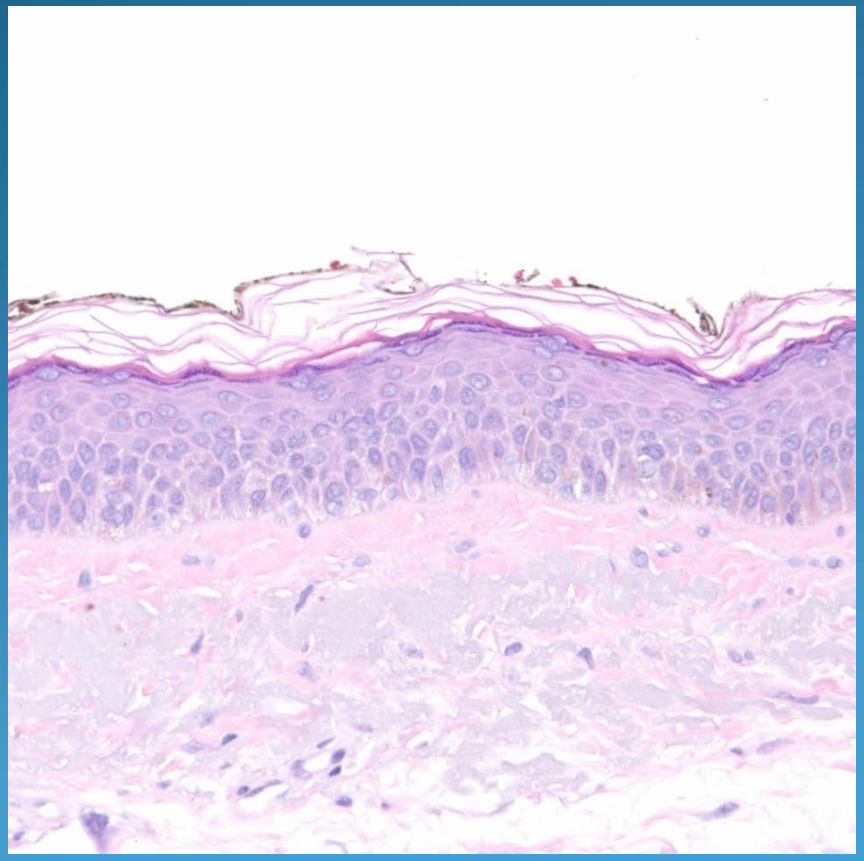




Retrospective Review

- Retrospective review of 444 cases of MMIS
- Ten cases identified
- MF 8:2
- Mean age 67 years (53-79 yrs)
- Location-Head and neck/upper trunk
- Clinical impression:
  - Atypical nevus 5/10
  - LM 3/10
  - Lentigo 1/10
  - BCC 1/10

Histopathology



- Low power architecture of sun damaged skin or lentigo
- No epidermal atrophy
- No bridging nests or discohesive clefting
- Junctional melanocytes small with minimal atypia
- Confirm by Melan-A staining

Characteristic	MMIS	AMMIS
Sun damaged skin	+	+
Epidermal atrophy	+	
Bridging/dyscohesive melanocytic nests	+	
Severe melanocytic atypia	+	
Starburst melanocytes	+/-	
Extension along adnexal epithelium	+	+

Amelanotic Melanoma In Situ: A Histopathologic Mimic of Benign Lesions

Belinda H. Tan, Paul K. Shitabata

Division of Dermatology, Harbor-UCLA Medical Center and the Dermatopathology Institute, Torrance, CA, USA.

The entity of amelanotic melanoma in situ (AMMIS) may be under recognized. Atypical cells confined to the epidermis and absent pigment granules are salient features that should prompt high power histopathologic examination. In this retrospective study, a review of 444 cases of malignant melanoma in situ yielded ten diagnoses of AMMIS. Of these, patients averaged 67 years old (ranging from 53 to 79 years) with the majority (8/10) being male. The clinical impressions varied and included atypical nevus (5/10), lentigo maligna (3/10), lentigo (1/10), and basal cell carcinoma (1/10). Lesions localized to the face, trunk, or extremities. Microscopically, these specimens demonstrated solar elastosis and resembled lentigos at low power. A bandlike infiltrate in some suggested lichenoid keratosis. In general, there was no epidermal atrophy, upward intraepithelial spread by atypical melanocytes, bridging nests or discohesive clefting. Junctional melanocytes were small, equal to, or less than the size of adjacent keratinocytes, with minimal atypia, and had little to no pigment. Taken together, the differential diagnosis included pigmented actinic keratosis, lentiginous melanoma, lentigo maligna and superficial spreading malignant melanoma in situ, Bowen's disease, and extramammary Paget's disease. Positive staining for melanocyte marker Melan-A and negative HMW cytokeratin supported AMMIS in cases. AMMIS may present a diagnostic pitfall when examined under low power and therefore, when suspecting this diagnosis, high power examination combined with Melan-A staining is strongly recommended.