A 16-year-old boy presented with an asymptomatic, hyperpigmented, hypertrichotic lesion on his right upper chest extending into the right axilla. The pigmentation, first noted 4 years earlier, had progressively enlarged. The coarse and dark hair confined to the hyperpigmented area appeared 2 years before.

The patient’s medical history was uneventful. A review of systems showed no abnormalities. There was no family history of similar skin lesions.

Physical examination showed a large patch consisting of numerous brownish macules with hypertrichosis on the right upper chest and right axilla (Figure 1). The rest of the physical examination was unremarkable.

What’s your diagnosis?
**Answer: Becker nevus**

Becker nevus (also known as Becker melanosis or Becker pigmentary hamartoma) is characterized by a circumscribed, brownish patch with geographical borders and sometimes hypertrichosis.\(^1\) This disorder is named after Samuel William Becker, who in 1949 reported 2 young men with focal, unilateral, acquired, hyperpigmentation and hypertrichosis.\(^2\)

**Epidemiology**

Onset of Becker nevi is usually around puberty, with a prevalence of 0.25% to 0.5% in adolescent boys and young men.\(^3\)\(^-\)\(^5\) The condition is rarely congenital, and the male-to-female ratio is approximately 5 to 1.\(^3\)\(^,\)\(^6\) Becker nevi occur in all races but are more common in nonwhites than whites.\(^4\) The majority of cases are sporadic, but familial cases have also been reported.\(^7\)\(^,\)\(^8\)

**Etiopathogenesis**

The exact etiopathogenesis of Becker nevus is not known. Some investigators consider it a hamartoma of ecto-mesodermal tissues, appearing sporadically by cutaneous mosaicism.\(^9\) An autosomal dominant mode of inheritance with incomplete penetrance has rarely been reported.\(^9\)\(^,\)\(^10\)

The fact that Becker nevi become more prominent after puberty, are more predominant in males, occur with colocalized hypertrichosis and acneiform eruption, and have a high affinity of pityriasis versicolor suggests that a target tissue increase in androgen receptors or sensitivity to androgens may be responsible.\(^11\) Studies have shown the extent of androgen receptor expression is higher in the epidermis of a Becker nevus and higher levels of androgen receptor messenger RNA within the Becker nevus compared with unaffected skin fibroblasts.\(^11\)

**Histopathology**

Histopathological features of Becker nevi include epidermal acanthosis, regular elongation and fusion of rete ridges, hyperpigmentation of the basal layer, and hyperkeratosis, as well as dermal smooth muscle hyperplasia.\(^6\) Although the lesion is
called a nevus, nevus cells are absent in the dermis. Thus, malignant transformation is not a concern.

**CLINICAL MANIFESTATIONS**

Although Becker nevi can be present at birth, the majority of them are first noticed in the second decade of life.\(^1,\)\(^7\) Clinically, a Becker nevus presents as an asymptomatic circumscribed light to dark brownish macule or patch that gradually enlarges in an irregular fashion, similar to a geographical configuration.\(^1\) The hyperpigmentation may appear as a single irregular hyperpigmented macule/patch or multiple blotchy hyperpigmented macules arranged in a checkerboard pattern as seen in the present case (Figure 1).\(^1,\)!\(^2\)\(^-\)!\(^3\) The lesion may become raised, presumably secondary to androgen stimulation.\(^1\)!\(^2\) Hypertrichosis usually develops a few years after the pigmentation.

Hairs generally appear in the region of the pigmentation but are not necessarily confined to that area; they become coarser and darker with time (Figure 1). Nonhairy Becker nevi account for approximately 50% of cases (Figure 2).\(^6\) Sites of predilection include the shoulder, scapula, and upper chest. Other less commonly affected areas include the face, neck, extremities, groin, and genitalia.\(^3\) The majority of cases are unilateral, but a few cases of bilateral occurrence have also been reported.\(^5\)!\(^8\) Multiple lesions are extremely rare.\(^1\)!\(^8\) Becker nevi are rarely segmental and follow Baschko lines.\(^1\)!\(^5\)

Cutaneous anomalies that colocalize with Becker nevus include: acne, granuloma annulare, neurofibromatosis type 1, lichen planus, vitiligo, discoid lupus erythematosus, and pityriasis versicolor.\(^3\)!\(^5\)!\(^1\)!\(^6\) Occasionally, Becker nevi may be associated with ipsilateral breast hypoplasia, supernumerary nipples, aplasia/hypoplasia of the pectoralis major muscle, smooth muscle hamartoma, lipoatrophy, and skeletal anomalies (such as: ipsilateral limb hypoplasia, pectus excavatum, pectus carinatum, scoliosis, segmental odontomaxillary dysplasia, hemivertebrae, and spina bifida).\(^3\)!\(^1\)!\(^8\) The term “Becker nevus syndrome” has been used to describe the association of a Becker nevus with noncutaneous anomalies.\(^3\)!\(^1\)!\(^8\)!\(^1\)!\(^9\)
DIAGNOSIS

The diagnosis of Becker nevus is primarily clinical based on its typical features. This can be aided by dermoscopy, which typically reveals network, focal, skin furrow and perifollicular hypopigmentation, hair follicles, and vessels. A biopsy of the lesion or referral to a dermatologist should be considered if the diagnosis is in doubt.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes congenital melanocytic nevus, café au lait patch, Albright syndrome, and nevus spilus. A congenital melanocytic nevus refers to a melanocytic nevus present either at birth or within the first few weeks of life. The color varies from light to dark brown. The lesion is palpable but reasonably flat at birth. It may be hairy or hairless. With time, the lesion tends to become darker and more elevated. Coarse dark hair may become prominent in late childhood.

Clinically, a café au lait patch is a flat, discrete, uniformly pigmented lesion with well-defined borders. The pigmentation is tan or light brown in white-skinned patients and dark brown in dark-skinned individuals. The color simulates that of “coffee and milk,” hence the name “café au lait.”

Albright syndrome, or McCune-Albright syndrome, is characterized by the triad of polyostotic fibrous dysplasia, hyperfunctioning endocrine system (notably precocious puberty and hyperthyroidism), and a hyperpigmented lesion on the same side as the body lesion; the skin lesion is present at birth and has a serrated border like the “coast of Maine.”

Nevus spilus classically presents as a light-brown circumscribed pigmentation that is stippled with dark brown punctate macules or papules. Although nevus spilus can be congenital, described pigmentation that is stippled with dark brown punctate macules or papules. Although nevus spilus can be congenital, the lesion is palpalable but reasonably flat at birth. It may be hairy or hairless. With time, the lesion tends to become darker and more elevated. Coarse dark hair may become prominent in late childhood.

PROGNOSIS AND MANAGEMENT

The lesion is benign and asymptomatic. Therefore, except for cosmesis, no treatment is necessary. Reassurance may be all that is needed. Affected patients should, however, be examined for associated noncutaneous abnormalities.

For those who desire treatment for cosmetic purposes, the hyperpigmentation can be treated with Q-switched ruby and Erbium:YAG lasers with selective damage of superficial melanocytes and with some beneficial results. The hypertrichosis can be treated with depilation, a diode, or Nd:Yag laser.

Alexander K. C. Leung, MD, is a clinical professor of pediatrics at the University of Calgary and a pediatric consultant at the Alberta Children’s Hospital in Calgary, Alberta, Canada.

Benjamin Barankin, MD, is a dermatologist and the medical director and founder of the Toronto Dermatology Centre in Toronto, Ontario, Canada.

REFERENCES