A man with a pigmented growth on his chest

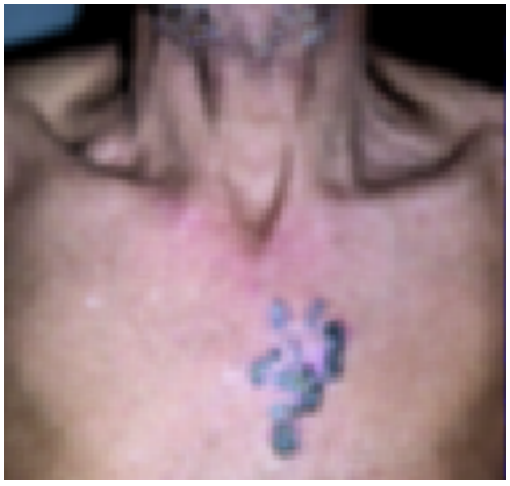
Amor Khachemoune, MD, CWS
SUNY Downstate Medical Center, Brooklyn, NY

A 50-year-old man came to the office with a 7-year history of a slowly growing lesion on his chest. The patient, an outdoor worker with excessive sun exposure for 20 years, had skin phototype IV (burns minimally, always tans well to moderately brown). He reported that this lesion has never healed, has bled on occasion, and that regular wound care over several months was ineffective. He had suffered no trauma, nor had he applied any caustic products to this area. The patient was otherwise healthy and was not taking any medications. His family history was not contributory.

Physical examination revealed a large plaque about 4.5 by 3 cm with irregular borders (**FIGURE 1**). It seemed to have been expanding centrifugally for a long time, with areas of skipping, individual nodules, and papules. Close exam showed central ulcerations and crusting on scattered papules and nodules (**FIGURE 2**). The borders were rolled up and had a somewhat pearly appearance with erythema and telangiectasia. The area immediately surrounding the plaque was also remarkable for faint erythema. The central part of the lesion was atrophic with pinkish discoloration; on finger pinching of the central, the skin seemed to be sclerotic. The rest of the skin, mucosal, adnexal examination and review of systems was unremarkable. No lymphadenopathy was present. A skin biopsy was done to confirm the clinical impression.

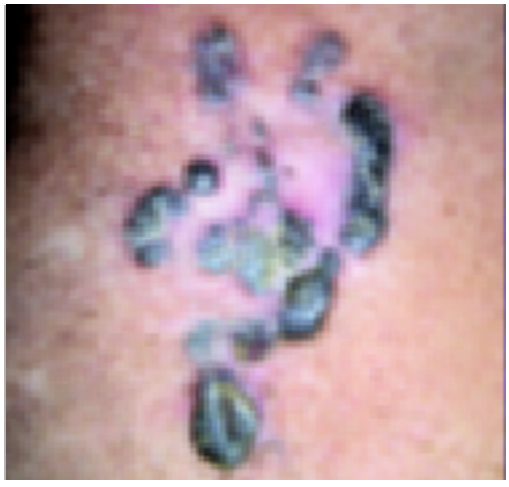
What is your diagnosis?

FIGURE 1 Pigmented plaque on chest



Pigmented plaque with area of skipping and centrifugal extension, atrophic center

FIGURE 2 Close-up



Close-up of the plaque, showing crusting on scattered nodules and papules.

FEATURE EDITOR
Richard P. Usatine, MD
University of Texas Health Sciences Center at San Antonio

CORRESPONDING AUTHOR
Amor Khachemoune, MD, CWS, SUNY Downstate Medical Center, Department of Dermatology Box 46, 450 Clarkson Ave, Brooklyn, NY 11203. E-mail: amorkh@pol.net

FAST TRACK

Speckled pigmentation is a distinguishing feature of BCC, and may be used to differentiate it from other skin tumors

Diagnosis: Basal cell carcinoma, pigmented subtype

Basal cell carcinoma (BCC) is the most common cancer among Caucasians[1] and accounts for approximately 75% of all skin cancers. Resultant mortality is very low, but it may cause destruction to local and surrounding structures. Metastasis to lymph nodes and other organs and subsequent death have been reported with BCC.[2,3] Other names for BCC are basalioma, basal cell epithelioma, rodent ulcer, and Jacobs' ulcer.

Clinical presentations: Location, subtypes, risk of recurrence

The face (particularly the nose) is the most common site for BCC. A small percentage of BCCs occur on the trunk; it is rare on areas not exposed to the sun, such as the penis, vulva, perianal areas and axilla. Many subtypes of BCCs exist, including nodular, superficial, cystic, micronodular, morpheaform, and pigmented. In addition to features seen in lesions of nodular BCC, the pigmented subtype contains increased brown or black pigment, and it is seen more commonly in persons with dark skin.

Histology and new investigations

BCC arises from the basal layer of the epidermis. There are many histologic subtypes. The basaloid cells form tumor aggregates or nests of varying sizes. Cells tend to align more densely in a palisade pattern at the periphery of these nests. In the morpheaform subtype, the cells are embedded in fibrous stroma.

In a recent article, Goldberg et al[4] used histopathology and special staining to show that BCC lesions had melanin pigment (positive for Fontana-Masson stain and negative for Perl's stain) within nests of tumor cells. The authors concluded that speckled pigmentation of a basal cell carcinoma is a distinguishing feature, which may be useful in differentiating this tumor from other discrete skin tumors.

More recently, endothelins (ETs) have been implicated as participating in the pigmentation process of BCC. Enhanced ET-1 expression in pigmented BCC plays an important role in the hyperpigmentation of this tumor.[5]

Risk factors for BCC: Environment, genetics

Solar radiation is the chief environmental cause of BCC. Therapeutic radiation, such as PUVA for psoriasis or radiation for the treatment of acne or tinea capitis may result in BCC many years after exposure. Genetic characteristics such as fair skin, light-colored eyes, and red hair are also important risk factors. Another reported risk factor is chronic arsenic exposure, which may result from ingestion of contaminated water or seafood.

Basal cell nevus syndrome is an autosomal dominant genetic disorder with multiple characteristic clinical features. These features include the development of multiple BCCs at a relatively young age, macrocephaly, frontal bossing, hypertelorism, bifid ribs, palmar and plantar pitting, and bone cysts in the mandible. These patients have a mutation in the tumor suppressor gene patched (PTCH) on chromosome 9.[6] Xeroderma pigmentosum and other rare genetic diseases also increase the likelihood of BCC tumors. Risk of BCC recurrence is related to tumor location and size, histologic type, and treatment modality and history of UV exposure.

Differential diagnosis for pigmented BCC

- Malignant melanoma
- Melanocytic nevus
- Spitz nevus
- Pigmented seborrheic keratosis
- Pigmented dermatofibroma
- Squamous cell carcinoma
- Pigmented Bowen's disease (squamous cell carcinoma in situ)
- Keratoacanthoma
- Trichoepithelioma
- Sebaceous hyperplasia
- Fibrous papule of the nose

Treatment: Local destruction, photodynamic therapy, immune modulators

Treatment options available for BCC depend on characteristics of the tumor type, location, individual patient, and economic resources.

Cryosurgery, electrodessication and curettage, CO_2 laser destruction, surgical excision, Mohs micrographic surgery, radiation therapy, topical therapy such as 5-fluorouracil (5-FU) and imiquimod (Aldara), intralesional interferon, and photodynamic therapy are used to treat BCC.

Older treatments for low-risk BCCs in cosmetically less significant areas

Cryosurgery induces cytotoxicity by production of extracellular and intracellular ice crystals. It is rapid and cost-effective, and demonstrates high cure rates.

Electrosurgery can be used for superficial and nodular BCC on the trunk and extremities.

Surgical excision is an effective method for BCCs located on low-risk areas without aggressive histologic features.

Radiation therapy is an option for the treatment of both primary and recurrent BCCs.

Topical chemotherapeutic agent 5-fluorouracil (Efudex, Carac, Fluoroplex) is also effective against BCC.

Interferon (interferon alpha 2b) has also been used as intralesional injections to treat superficial and nodular BCCs.

Treatment for high-risk tumors

Mohs micrographic surgery is currently the standard of treatment for high-risk BCCs and BCCs located in cosmetically sensitive locations such as the nasolabial fold and periorbital areas. It has a higher cure rate than other modalities.

Relatively new methods

Imiquimod is used for primary superficial BCC (not on head or neck) in adults with normal immune systems. It is used for tumors 2 cm or smaller in diameter on certain areas of the body. Imiquimod treatment is indicated only when surgical methods are not appropriate.[7]

Photodynamic therapy includes the use of various intravenous and topical photosensitizers—ie, intravenous verteporfin, topical 5-aminolevulinic acid (ALA), topical methyl aminolevulinate (mALA)—combined with different sources of visible light. Photodynamic therapy is being used for some BCCs but is not recommended for pigmented BCC.[8]

Given the ill-defined nature of this lesion, and its size, we recommended surgical excision using either regular excision with staining or marking of margins, or Mohs micrographic surgery. Unfortunately, the patient was lost to follow-up. ■

ACKNOWLEDGMENTS

The author wants to thank Dr. Shahbaz A. Janjua with his assistance in the preparation of this manuscript.

REFERENCES

1. Marks R. An overview of skin cancers: incidence and causation. *Cancer* 1995; 75:607–612.
2. Robinson JK, Dahiya M. Basal cell carcinoma with pulmonary and lymph node metastasis causing death. *Arch Dermatol* 2003; 139:643–648.
3. Tilli CM, Van Steensel MA, Krekels GA, Neumann HA, Ramaekers FC. Molecular aetiology and pathogenesis of basal cell carcinoma. *Br J Dermatol* 2005; 152:1108–1124.
4. Goldberg LH, Friedman RH, Silapunt S. Pigmented speckling as a sign of basal cell carcinoma. *Dermatol Surg* 2004; 30:1553–1555.
5. Lan CC, Wu CS, Cheng CM, Yu CL, Chen GS, Yu HS. Pigmentation in basal cell carcinoma involves enhanced endothelin-1 expression. *Exp Dermatol* 2005; 14:528–534.
6. Blyumin ML, Khachemoune A. Self-assessment examination. Man with multiple papulonodules on the back. Identification no. 804-203. *J Am Acad Dermatol* 2004; 50:498–494.
7. Oldfield V, Keating GM, Perry CM. Imiquimod: in superficial basal cell carcinoma. *Am J Clin Dermatol* 2005; 6:195–200.
8. Kaviani A, Ataie-Fashtami L, Fateh M, Sheikhbahaee N, Ghodsi M, Zand N, Djavid GE. Photodynamic therapy of head and neck basal cell carcinoma according to different clinicopathologic features. *Lasers Surg Med* 2005; 36:377–382.

FAST TRACK

Treatment options available for BCC depend on characteristics of the tumor type, location, individual patient, and economic resources