Histologic Mimics of Basal Cell Carcinoma

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• **Context.**—Basal cell carcinoma (BCC) is the most common human malignant neoplasm and is a frequently encountered diagnosis in dermatopathology. Although BCC may be locally destructive, it rarely metastasizes. Many diagnostic entities display morphologic and immunophenotypic overlap with BCC, including nonneoplastic processes, such as follicular induction over dermatofibroma; benign follicular tumors, such as trichoblastoma, trichoepithelioma, or basaloid follicular hamartoma; and malignant tumors, such as sebaceous carcinoma or Merkel cell carcinoma. Thus, misdiagnosis has significant potential to result in overtreatment or undertreatment.

**Objective.**—To review key features distinguishing BCC from histologic mimics, including current evidence regarding immunohistochemical markers useful for that distinction.

**Data Sources.**—Review of pertinent literature on BCC immunohistochemistry and differential diagnosis.

**Conclusions.**—In most cases, BCC can be reliably diagnosed by histopathologic features. Immunohistochemistry may provide useful ancillary data in certain cases. Awareness of potential mimics is critical to avoid misdiagnosis and resulting inappropriate management.


Basal cell carcinoma (BCC) is the most common human malignant neoplasm, with an estimated overall lifetime risk of 30% in the United States.\(^1,2\) Although BCC may cause extensive local tissue destruction if not adequately managed, metastasis is exceedingly rare.\(^2\) The diagnosis of BCC is usually straightforward on the dermatopathology service. However, BCC may display overlapping histopathologic findings with benign follicular tumors as well as other cutaneous carcinomas. Misdiagnosis may lead to overtreatment or undertreatment. Therefore, awareness of the potential histologic mimics of BCC is critical.

**CLINICAL FEATURES AND COURSE**

Typically, BCC arises on sun-exposed parts of the body, such as the face, neck, and head, with the remainder on the trunk and lower limbs.\(^3\) Patients are usually middle aged or elderly.\(^4\) Although these slow-growing tumors rarely metastasize, delayed or incomplete treatment may result in significant morbidity from local invasion leading to destruction of skin and deeper tissues, including cartilage and bone, especially in relation to the nares, eyes, and ears.\(^5\) Treatment of BCC can be surgical or nonsurgical. Surgical techniques range from curettage and cautery to cryosurgery, excision, and Mohs micrographic surgery. Photodynamic therapy and topical treatments, such as fluorouracil and imiquimod, have been investigated for superficial lesions.\(^3,4\) Metastatic or locally advanced tumors may be managed with vismodegib or other inhibitors of Hedgehog pathway signaling.\(^5\)

Increased risk of BCC may occur in the setting of certain inherited syndromes. The key role for the sonic hedgehog (SHH) signaling pathway in BCC is highlighted by Gorlin-Goltz syndrome (basal cell nevus syndrome), an autosomal-dominant condition related to germline patched 1 (PTCH1) mutation, characterized by multiple BCCs with a young age of onset, keratocystic odontogenic tumors, medulloblastoma, cardiac and ovarian fibromas, and other skeletal anomalies.\(^6\) Other tumor syndromes that predispose patients to BCC are associated with decreased skin pigmentation or epidermal genomic instability, including xeroderma pigmentosum, Rombo syndrome, Bazex-Christol-Dupre syndrome, Rasmussen syndrome, and albinism.\(^2\)

**HISTOPATHOLOGY**

Microscopically, BCC is composed of cells with large, elongated nuclei that display variably prominent palisading at the edge of tumor nodules (Figure 1, A). Cytoplasm may be inconspicuous, pale, or lightly eosinophilic. Mitoses and single-cell apoptoses are usually present and may be prominent. Intratumoral mucin may form large pools or cystic spaces (Figure 1, B). The BCCs display characteristic clefiting between the stroma and edges of tumor nodules, which may be extensive or focal (Figure 1, B). They are associated with a characteristic myxoinflammatory stroma that displays variable proportions of mucin, lymphocytic inflammation, prominent fibroblasts, and collagen thickening (Figure 1, A through C).

Basal cell carcinoma displays a variety of growth patterns that may be broadly divided into nonaggressive (indolent) and aggressive types. Growth patterns exist on a histologic
Figure 1. Basal cell carcinoma (BCC). A, Superficial BCC displaying small basaloid nodules with prominent peripheral palisading, extending from the base of the epidermis into the papillary dermis. B, Nodular (circumscribed) BCC displaying large, well-defined dermal nodules. C, Basaloid cells with peripheral nuclear palisading and clefting between tumor and stroma. Characteristic BCC stroma displays inflammation, fibrosis, prominent large fibroblasts, and increased mucin. D, Aggressive (infiltrating) BCC displaying angulated strands of tumor cells in mucinous stroma. E, Micronodular BCC with small, round, and infiltrative nodules. F, Morpheaform BCC may be composed of deceptively bland tumor nodules and strands within sclerotic stroma. G, Fibroepithelioma of Pinkus, with thin anastomosing epithelial cords in a myxoid stroma. H, Focal mitotic activity and cleft retraction in fibroepithelioma of Pinkus (hematoxylin-eosin, original magnifications \( \times 200 \) [A and C], \( \times 20 \) [B, D, E, and F], \( \times 10 \) [G], and \( \times 23 \) [H]).
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**Histologic Mimics of Basal Cell Carcinoma (BCC)**

- **Nonneoplastic epidermal changes**
  - Superficial but-like, basaloid, epidermal proliferation; peripheral palisading (focal) retraction artifact
- **Benign follicular tumors**
  - Benign, hair-germ tumors (trichoeipithelioma, trichoblastoma)
  - Basaloid cells; peripheral palisading; mucin deposition within tumor nodules; apoptotic bodies; connection to epidermis (less common than in BCC)
- **Sclerosing basaloid neoplasms**
  - Basaloid cells; peripheral palisading; mucin deposition within tumor nodules; apoptotic bodies; connection to epidermis (less common than in BCC)
- **DTE**
  - Basaloid cells; peripheral palisading; mucin deposition within tumor nodules; apoptotic bodies; connection to epidermis (less common than in BCC)
- **Syringoma**
  - Basaloid cells; peripheral palisading; mucin deposition within tumor nodules; apoptotic bodies; connection to epidermis (less common than in BCC)
- **Microcystic adnexal carcinoma**
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continuum, and combinations of growth patterns in a tumor are seen in 40% to 75% of BCC specimens. Furthermore, there is an intrinsic error rate of shave and punch biopsies of approximately 20% in determining BCC subtype classification compared with complete excisions. Despite those limitations, growth patterns represent a major predictor of tumor behavior.

Indolent BCC variants, such as superficial and nodular (or circumscribed) BCCs, show small lobules extending into the papillary dermis from the basal layer of the epidermis or large nodules in the papillary and reticular dermis, respectively (Figure 1, A and B). Cleft retraction, peripheral palisading, and stromal mucin are often prominent.

Aggressive-growth BCC variants, including micronodular, infiltrative, morpheaform, and metatypical, share features of increased cell necrosis, mitotic activity, and stromal proliferation, with a decreased demonstration of stromal retraction, deeper growth, and less circumscription. These variants demonstrate higher rates of recurrence and metastasis (although metastasis is rare for BCC of any growth pattern). The infiltrative (or aggressive) growth pattern comprises angulated nests and strands of tumor cells in a prominent, fibroblastic stroma (Figure 1, D). Micronodular BCC shows round to oval tumor nests, but unlike nodular BCC, the nests are smaller and widely dispersed (Figure 1, E). Morpheaform or sclerosing BCC shows thin columns and small nodules associated with intensely collagenized stroma (Figure 1, F). Metatypical BCC has a component of squamous differentiation within angulated nests of tumor cells, imparting morphologic overlap with squamous cell carcinoma. Of note, the term basosquamous carcinoma is sometimes used synonymously for metatypical BCC but may also describe collision tumors between BCC and squamous cell carcinoma; therefore, this term is best accompanied by clarification of the intended definition.

The BCCs may display alternative differentiation, most commonly squamous differentiation. Although many BCCs display focal squamatization, tumors with more-extensive squamous differentiation may be designated as metatypical or keratotic. The metatypical pattern of squamous differentiation is described above. Keratotic BCC demonstrates central squamous differentiation and horn cysts within well-circumscribed basaloid nodules. Less-common forms of alternative/ divergent differentiation in BCC include matrical, eccrine, sebaceous, or myoepithelial differentiation. Further variant morphologies that may be seen in BCC include clear cell change, signet ring cells, granular cells, adamantinomatoid, adenoid cystic, and metaplastic carcinoma.

An additional variant, the fibroepithelioma of Pinkus, typically presents as a solitary nodule on the trunk. Histologically, the tumor is characterized by a well-circumscribed nodule of anastomosing epithelial strands and basaloid buds in a myxoid stroma (Figure 1, G and H). Some have proposed this tumor may represent a fenestrated trichoblastoma. However, the immunophenotypic overlap with BCC, and association with a component of conventional BCC in some cases, support classification as a variant of BCC.

**ANCILLARY STUDIES**

Basal cell carcinoma displays a profile of cytokeratin expression similar to follicular germinative cells, characterized by CK5/6 and CK14 expression and the absence of CK20. Reports have been mixed regarding CK7 expression in BCC, Ber-E4 typically shows strong expression. There is diffuse nuclear expression of p63. Epithelial membrane antigen (EMA) and carcinoembryonic antigen (CEA) are typically not expressed, although EMA expression may be present in squamatized areas.

Most BCCs harbor SHH pathway-activating mutations affecting genes, including PTCH1 (the most common aberration), SMO, and SUFU. Sporadic BCCs frequently also harbor mutations in the tumor-suppressor TP53, Recent studies have identified additional, less-common mutations affecting genes, including MYCN, ERBB2, PIK3CA, NRAS, KRAS, HRAS, PTPN14, and RB1, which may contribute to BCC tumorigenesis in a few cases.

**DIFFERENTIAL DIAGNOSIS**

There are multiple benign and malignant dermatologic entities that may mimic BCC histologically, and misdiagnosis may lead to unnecessary excision or delayed workup of metastatic disease. Histologic mimics of BCC may include nonneoplastic processes (such as follicular induction over dermatofibromas), benign adnexal tumors (especially follicular tumors), or cutaneous carcinomas with basaloid or blue-cell features. Distinguishing these entities requires clinical correlation, identification of key histologic features, and possibly, ancillary tests, including immunohistochemistry. Here, we review situations in which BCCs may show significant histologic overlap with benign and malignant cutaneous lesions and provide a practical approach to distinguishing these entities.

**Follicular Induction over Dermatofibroma**

The epidermis above a dermatofibroma (DF) may be associated with epidermal hyperplasia, including basaloid proliferations with evidence of follicular differentiation, such
as follicular bulbs, dermal papillae, and papillary mesenchymal bodies. These types of proliferation are referred to as follicular induction (FI) (also known as follicular basal cell hyperplasia, superficial basal cell hyperplasia and basaloid epidermal proliferation). Estimates of the incidence of FI in dermatofibromas range from 2% to 23%. In contrast, the occurrence of BCC over DF is extremely rare, with few supporting case reports.

Distinction between FI and BCC can be challenging in superficial biopsies, in which the DF is not directly visualized (Table). Both BCC and FI show peripheral palisading and a superficial, budlike growth pattern. In contrast to BCC, FI may demonstrate evidence of follicular differentiation, including germinative buds and papillary mesenchymal bodies (Figure 2, C). One study suggested that clear cell hyperplasia is indicative of FI. Unlike FI, BCC lacks epidermal hyperplasia between nests. Cleft retraction can be seen in both, although extensive clefting is more typical of BCC. In challenging cases, immunohistochemistry may be considered. Studies have had mixed results regarding the utility of Ki-67 and p53 for distinguishing BCC from FI. Similar to benign follicular tumors, FI will demonstrate colonization by CK20+ Merkel cells in most cases, whereas BCC will not. As noted above, collision tumors between BCC and DF are exceedingly rare; however, the occurrence of FI in this context may represent a potential diagnostic confounder (Figure 2, D through F).

**Benign, Hair-Germ Tumors (Trichoepithelioma and Trichoblastoma)**

The benign, hair-germ tumors trichoblastoma (TB) and trichoepithelioma (TE) most commonly arise on the head and neck. Some consider TE to represent a subtype of TB. Most TE/S are sporadic and solitary tumors in younger patients; however, less commonly, they can be a manifestation of Brooke-Spiegler syndrome, associated with germ-line mutations in the tumor suppressor gene CYLD.

Histologically, TB is a large, circumscribed dermal tumor without epidermal connection, composed of small basaloid cells arrayed in numerous large nodules or micronodules (Figure 3, A). Similarly, TE is a well-circumscribed dermal tumor consisting of nests and strands of basaloid cells in a dense, concentric configuration. Constituent cells in TE appear relatively less primitive than those in TB. The TE may display intervillous horn cysts and epidermal connection in some cases (Figure 3, B). Both TE and TB display peripheral palisading, as well as a follicular stroma characterized by orderly, concentric collagen and fine-spindled fibroblasts, arranged in parallel to the periphery of tumor nodules. Papillary mesenchymal bodies (oval condensations of stroma capped by tumor epithelium) (Figure 3, C) are highly specific for benign, hair-germ tumors but may not be identified in some cases. Although mucin may be present within tumor nodules, stromal mucin is lacking. Unlike BCC, TE/TB lack prominent cleft retraction, myxoinflammatory stroma, significant mitotic activity, cytologic atypia, or tumor necrosis (Table).

Desmoplastic trichoepithelioma (DTE) is a histologic variant of TE that merits additional discussion. Typically, DTE presents as a firm plaque or nodule with a central dell on the face of young to middle-aged individuals. Histologically, DTE is characterized by thin cords of small basaloid cells in a densely sclerotic stroma (Figure 3, D). Morphologically, DTE mimics morpheaform BCC (mBCC), which can be challenging to differentiate, especially in superficial biopsies. Histologic features that favor DTE are architectural symmetry, horn cysts with calcifications, narrow cellular aggregates, giant cell reactions, and follicular/sebaceous/infundibular differentiation (Table). Additionally, the lack of features such as mitotic figures, cytologic atypia, and tumor-to-stroma clefting also favors the diagnosis of DTE, rather than mBCC.

In challenging cases, immunohistochemical stains can be helpful in discriminating between TE/TB and BCC (Table). Benign, hair-germ tumors are frequently colonized by benign Merkel cells, which can be highlighted by CK20 (Figure 3, E). CK20 can be used in combination with androgen receptor (AR), with positive tumor AR staining and absent CK20 staining supporting BCC, whereas CK20+ colonizing Merkel cells and lack of AR support a benign diagnosis. Several limitations of the AR/CK20 panel should be kept in mind. The AR expression is often focal and sometimes completely absent in BCC. Some TE/TBs have a low density of colonizing Merkel cells, which may require serial sections for detection. In addition, Merkel cells in a background of vellus follicles may simulate tumor colonizing.
4, C and D). Unlike BFH, infundibulocystic BCC typically displays at least focal mitotic activity, cytologic atypia, myxoinflammatory stroma, and/or cleft retraction (Table). CD10, CD34, and BCL2 have been proposed to be helpful in this distinction, with staining patterns in BFH similar to those described above for TE/TB. One report described colonizing CK20+ Merkel cells in infundibulocystic BCC as well as in BFH; therefore, there is uncertainty regarding the diagnostic utility of CK20 in this specific context.75

**Figure 2.** Follicular induction (FI) in dermatofibroma (DF). A, Basaloid bud with peripheral, palisading and clefting, adjacent to epidermal hyperplasia. Fibrohistiocytic cells are visible in the underlying dermis. B, Larger nodules may form in FI. C, Follicular differentiation in FI may take the form of a papillary mesenchymal body. D, Scanning appearance of a rare DF–basal cell carcinoma (BCC) collision tumor. E, The DF in this collision tumor displays FI. F, Adjacent BCC in the collision tumor (hematoxylin-eosin, original magnifications ×200 [A through C, E, and F] and ×10 [D]).

**Tumor of the Follicular Infundibulum**

Tumor of the follicular infundibulum (TFI) most commonly presents as a solitary, keratotic papule or plaque on the face of middle-aged to elderly patients48,76 or as an eruptive variant in younger patients with nevus sebaceous or certain tumor syndromes.48,77,78 Histologically, TFI has a distinctive pattern that consists of interconnecting plates of monomorphic keratinocytes arising as an extension from the basal
Small, abortive follicular structures may connect to the plate from below. Neoplastic cells are pale in comparison to the surrounding normal keratinocytes. Nuclear palisading is common. Although TFI may be misinterpreted as superficial BCC, TFI lacks cytologic atypia, myxoinflammatory stroma, peripheral clefting, and significant mitotic activity (Table). Unlike BCC, TFI displays colonizing CK20+ Merkel cells, and lacks Ber-EP4 staining. Elastin stains may demonstrate an elastin fiber network at the base of the lesion.

**Squamous Cell Carcinoma**

Squamous cell carcinoma may be challenging to distinguish from BCC in limited biopsies. Ber-EP4 is commonly employed to assist in this distinction, with strong expression in BCC and absent expression in squamous cell carcinoma.
Of note, Ber-EP4 may not be expressed in squamatized areas of BCC, and focal staining with Ber-EP4 has been reported in squamous cell carcinoma.

Microcystic Adnexal Carcinoma

Microcystic adnexal carcinoma (MAC) is a rare, locally aggressive, malignant neoplasm with follicular and sweat-gland differentiations. Clinically, MAC overlaps with BCC as a light-tan, smooth, firm lesion primarily affecting the central face, head, and neck of adult patients of all ages. Histologically, MAC is an ill-defined, infiltrative lesion that extends deep in the dermis (Figure 6). Deceptively bland squamoid and basaloid cells form tubular and ductal structures that are haphazardly distributed in a desmoplastic stroma (Figure 6, B). Horn cysts, primarily in the superficial portion of the lesion, and eosinophilic secretions within the ductal lumens are frequently identified. Mitotic activity is rare to absent. Perineural invasion is common and often a helpful diagnostic finding.

Differentiating mBCC and MAC poses a diagnostic challenge to dermatologists and pathologists. Furthermore, syringoma and DTE (described earlier) are 2 benign entities that also display a strandlike growth of tumor cells in sclerotic stroma and, hence, may appear highly similar to mBCC and MAC (Table). Adequate sampling to assess the depth of the lesion and the presence of perineural invasion are crucial because syringoma and DTE are superficial lesions that do not invade nerves. Both MAC and mBCC are ill-defined and infiltrative lesions, although mBCC less frequently invades nerves and consists entirely of basaloid cells that have no ductal differentiation. Conversely, peripheral palisading, tumor-to-stroma retraction, and myxoinflammatory stroma are rare in MAC.

Immunohistochemistry is generally not useful to discriminate between MAC and syringoma. Staining with CEA and EMA highlight ductal structures in MAC, which may help differentiate it from DTE and mBCC. In addition, CK15 stains positive in MAC and is negative in mBCC. Reports are mixed regarding whether a subset of MACs may express Ber-Ep4, and MACs may express p75NTR.

Sebaceous Carcinoma

Sebaceous carcinoma (SC) is a rare, aggressive, malignant neoplasm predominately affecting the periorbital region of elderly patients. Other anatomic sites that may be affected include the head and neck, trunk, extremities, and genital regions. The rates of distant metastases and death in periorbital SC are significantly greater than they are in BCC, and thus, accurate diagnosis is critical. The classic histologic appearance of SC is a dermal-based tumor with lobules of basaloid cells. A variable proportion of...
neoplastic cells show evidence of sebaceous differentiation in the form of indented nuclei and microvacuolated cytoplasm (Figure 7, A). Sebaceous differentiation may be diminished or absent in high-grade tumors.89,90,94 Cytologic atypia, nuclear pleomorphism, and mitotic activity are readily identified,93,97,98 and nuclear palisading may be present.74 Although sebocytes and sebaceous ductlike structures have been identified in BCC (Figure 7, B), sebaceous lobular architecture is not observed.90 Intraepidermal pagetoid spread is common in SC and is absent in BCC.89,90 Peripheral clefting is typically absent in SC.89,90 Immunohistochemical stains EMA and low–molecular-weight cytokeratin are strongly expressed in SC, in contrast to the weak-to-absent expression in BCC (Table).57,89,98,100,101 Although AR displays diffuse expression in SC and focal expression in BCC, Ber-EP4 is typically negative in SC and positive in BCC. Adipophilin and perilipin have been shown to stain strongly in SC and are restricted to sebocytes in BCC with sebaceous differentiation; however, nonspecific staining may be a challenge.98,102,103

Merkel Cell Carcinoma

Merkel cell carcinoma (MCC) is a rare, highly aggressive, primary cutaneous neuroendocrine carcinoma affecting older individuals and immunosuppressed patients and often presents as an erythematous or violaceous papule, nodule, or plaque on the skin of the head and neck or extremities. Although less than 1% of malignant skin tumors are MCCs,104 it is a significant cause of death from skin cancer, with a mortality rate of 30% to 75%.105 About one-third of patients will have metastatic disease at the time of diagnosis.106 Because of the increased mortality and high metastatic potential of MCC, it must be distinguished from less-aggressive lesions, such as BCC. Prompt staging is critical for establishing prognosis and guiding proper treatment in MCC.

Histologically, MCC typically appears on scanning magnification as a large, dermal, “blue” tumor nodule with sheetlike or trabecular growth (Figure 8, A and B). Nested or infiltrative growth patterns reminiscent of BCC may be observed.107 Both BCC and MCC may display epidermal connections. However, unlike BCC, MCC may have an intraepidermal component.108–111 Both MCC and BCC may have focal or extensive squamous differentiation.105 Peripheral palisading may be identified in a few MCC tumors but is typically focal.107,112–115 Clefts between aggregates of MCC and adjacent stroma are not infrequently identified in MCC (Figure 8, C).107 Although increased stromal mucin is typical of BCC,9 more than 90% of MCC tumors may also have some degree of stromal mucin (Figure 8, C).107,113 Stromal fibrosis and inflammation may also be present in MCC.105 However, despite that overlap in stromal features, the classic BCC myxoinflammatory stroma is not observed in MCC.

The distinction between BCC and MCC may be more readily distinguished by cell morphology. Although tumor cells of BCC are characteristically elongated with hypochromic nuclei, visible cytoplasm, and scattered mitotic activity and necrosis, tumor cells of MCC have a homogenous salt-and-pepper chromatin pattern, nuclear molding, scant cytoplasm, and abundant mitotic activity and necrosis.105

Merkel cell carcinoma cells share antigenic profiles of both epithelial and neuroendocrine cells. They express epithelial markers, such as low–molecular-weight keratins and EMA, as well as neuroendocrine markers, including chromogranin and synaptophysin.116 Intermediate filamentous, such as cytokeratins and neurofilament, classically demonstrate a paranuclear, dotlike pattern of staining in MCC (Figure 8, D), which is not observed in BCC. In some MCC tumors,
there is membranous or cytoplasmic cytokeratin staining, with or without paranuclear dots. Most MCCs express CK20, whereas that stain is negative in BCC. Merkel cell polyomavirus large T-antigen protein, expressed in approximately 70% to 80% of MCCs, is a highly specific marker for distinguishing MCC from BCC. Chromogranin or synaptophysin are not useful for the differential diagnosis of MCC from BCC because some BCC may express neuroendocrine markers. Ber-EP4 is expressed in both BCC and MCC.

Figure 7. A, Sebaceous carcinoma. Cells displaying microvesiculated clear cytoplasm consistent with sebaceous differentiation, intermingled with undifferentiated, malignant basaloid cells. B, Basal cell carcinoma (BCC) with sebaceous differentiation displaying scattered sebocytes. Mucinous stroma and subtle peripheral palisading is evident. Other areas of this tumor displayed more classic findings of BCC (hematoxylin-eosin, original magnifications ×200 [A] and ×100 [B]).

Figure 8. Merkel cell carcinoma (MCC). A, Scanning magnification typically demonstrates a large, blue tumor in the dermis. B, Round cells with neuroendocrine chromatin, minimal cytoplasm, and numerous mitoses. C, Stromal mucin and cleft retraction may be prominent. Note the absence of prominent fibroblasts and fibrosis mingled with mucin that would be seen in characteristic basal cell carcinoma (BCC) stroma. D, Cytokeratin 20 displaying a dotlike paranuclear staining pattern in MCC (hematoxylin-eosin, original magnifications ×10 [A], ×400 [B and D], and ×200 [C]).
Any large basaloid tumor presenting in the dermis, including poorly differentiated adnexal carcinoma or metatatic carcinoma, has the potential to be mistaken for BCC if attention is not given to the clinical presentation, high-grade cytologic atypia, and absence of hallmark features of BCC.

Rare variants of BCC may be associated with specific diagnostic pitfalls. Adenoid BCC may be confused with adenoid cystic carcinoma, in which setting CD117 and CD43 may be useful. In addition, BCC with extensive clear cell changes may raise consideration for trichilemmal carcinoma, clear cell melanoma, and other clear cell tumors presenting in the skin. Rarely, BCC may display prominent, thick, fibrous stroma mimicking cylindroma. Matrical differentiation may raise consideration for pilomatrical carcinoma.

CONCLUSIONS

We present several benign and malignant mimics of BCC and compare and contrast their clinical and histologic features to BCC. When a tumor is suspected of being a BCC but lacks some hallmark features of BCC or exhibits features not commonly seen in BCC, a differential diagnosis that includes the entities described above should be considered. Immunohistochemistry is helpful in differentiating BCC from its mimics; however, no stain is completely sensitive or specific. Therefore, integration of clinical, morphologic, and immunohistochemical features may be necessary in challenging cases.

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