Slide 1

EVALUATION AND TREATMENT OF BENIGN AND MALIGNANT EYELID LESIONS

Ana Carolina Victoria, MD
Oculofacial Plastic and Reconstructive Surgery

Slide 2

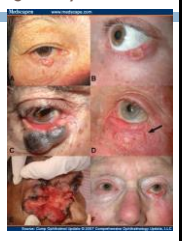
Characteristics suggestive of benign lesions

- Longstanding stability
- Regular, well defined margins
- Soft texture
- Intact surface epidermis
- No telangiectasias
- Preserved underlying architecture

Slide 3

Characteristics suggestive of malignancy

- Irregular border
- Induration
- Ulceration
- Telangiectasia (i.e. basal cell)
- Altered normal architecture
- Madarosis
- Tethering to deeper structures
- Growth or change of a lesion's color, border, or size



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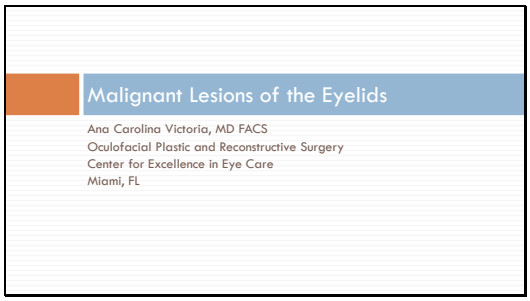
Slide 5



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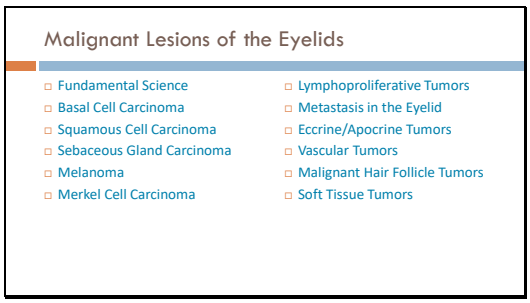


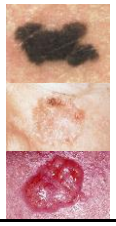
Slide 7

Incidence of Skin Cancers

- Most common malignancies in the human body
- Whites: 80-90% BCC, then SCC (5-8%), SGC (2-3%), melanoma <1%.
- More pigmented patients, lower incidence of BCC and SCC, higher SGC

Slide 8

Characteristics suggestive of malignancy



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Slide 9

Tumor margin control

- Surgical excision with clear margins
  - Intraoperative: Frozen-sections, Moh's micrographic surgery (MMS, assesses 100% of the margin)
  - Post-operative: Paraffin section
  - **Melanoma** is usually examined in paraffin

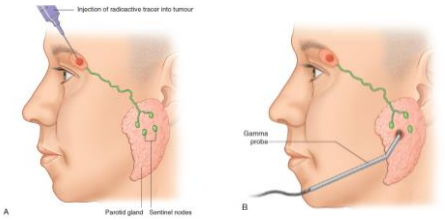
Slide 10

Staging

- TNM System
  - T:Tumor
  - N: Nodes
  - M: Mestastases
- Sentinel Lymph Node Biopsy (SLNB)
  - First Lymph node to which a tumor spreads
  - Eyelid: Parotid gland, salivary gland, cervical chain
  - Identifies subclinical, microscopic nodal spread

Slide 11

SLNB



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BASAL CELL CARCINOMA

Slide 13

Basal Cell Carcinoma

- Epidemiology
  - 80% in white (Australia highest rate)
  - Rare in dark skin
- Pathogenesis: Two mutations causing SMO activation appear to be required.
- Risk Factors
  - UV exposure: sun burns in childhood, M>F, >40yo
  - Fair skin
  - Immunosuppression
  - Cutaneous scars (burns)
  - Ionizing radiation
  - PUVA
  - Genetic conditions
  - Basal Cell Nevus Syndrome (BCNS/Gorlin)
  - Albinism
  - Xeroderma pigmentosum
  - Bazex syndrome (AD syndrome)
  - Rombo syndrome (AD syndrome)

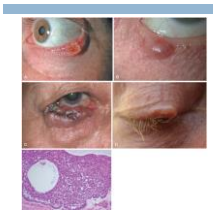
Slide 14

BCC-Clinical features

- Nodular
- Superficial
- Morpheic (sclerosing)
- Pigmented
- Cystic
- Linear

Slide 15

BCC-Nodular

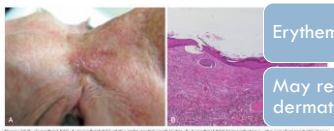


- Pearly papule
- Surface telangiectasia
- Dome shaped
- Central ulceration

Slide 16

BCC-Superficial

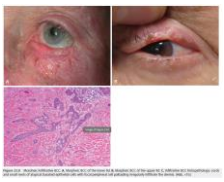
- Slow-growing
- Erythematous patch
- May resemble dermatitis



Slide 17

BCC-Morpheic

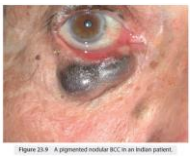
- Indurated
- Pooly defined
- White/pink/scarlike



Slide 18

BCC-Pigmented

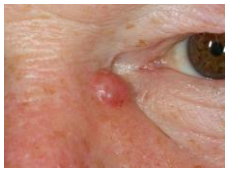
- Uniform or variegated brown, gray-blue, or black pigmentation
- Mimic nevi or melanoma
- Most common among pigmented races



Slide 19

BCC-Cystic

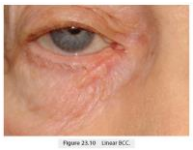
- Mimic benign cysts
- Associated mass or induration



Slide 20

BCC-Linear

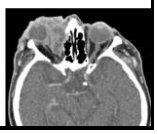
- Follows rhytids or the lid margin



Slide 21

BCC-Orbital Invasion

- 1.6-2.5% Cases
- Neglected/recurrent
- Canthus
- Infiltrative
- PNI
- Fixation to bone
- Restriction of EOM
- Ptosis
- Globe displacement



Slide 22

BCC-Metastasis

- Rare
- Median 9 yrs
- Regional LNs, bone, lungs, liver
- Poor prognosis

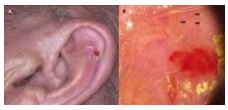
Slide 23

BCC-Pathologic classification

Multiple subtypes, no clinical significance except…

Basosquamous: feats BCC and SCC

Higher recurrence/mets



Slide 24

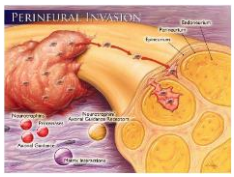
BCC-Perineural Invasion

GROWTH IN OR AROUND THE NERVE

HIGH RISK OF RECURRENCE

GREATER SUBCLINICAL SPREAD

DYSESTHESIA/PAIN



Slide 25

BCC-Prognosis

Treatment History

- Recurrent tumor
- Previous incomplete excision
- Previous nonsurgical treatment (e.g., radiotherapy, cryotherapy)

Tumor Size, Site, and Appearance

- Size
- H-zone
- Medial canthal location
- Poorly defined clinical margins

Histologic Features

- Infiltrative/morpheic/micronodular subtypes
- Perineural invasion

Patient Factors

- Immunosuppression

Slide 26

BCC-Prognosis

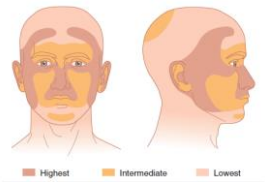


Figure 23.12 The high risk "H-zone" of the face. (From Vidimos A, Ammirati C, Poblete-Lopez C. Dermatologic surgery. London, UK: Saunders; 2008.)

Slide 27

BCC-Management

- Excision with clear margins
  - Post-op margins: 3-4mm (smaller margin with higher recurrence rate 15%)→Re-excision or close observation for narrow margins 0.5mm
  - Intra-op: MMS/frozen 1-2% recurrence rate, 5-7% for recurrent tumors.
- Recurrent
  - 3-4x rate of recurrence
  - Surgical excision with intra-op control
  - Adjunctive radiotherapy

Slide 28

BCC-Destructive Treatments

- Electrodesiccation and curettage→Useful for small (>6 mm) nodular BCCs (usually off the face)
- Cryosurgery with liquid nitrogen: Useful in BCCs of the superficial and nodular types with clearly definable margins; no clear advantages over the other forms of therapy; generally reserved for uncomplicated tumors (off the face)

Slide 29

BCC-Pharmacologic Management

- Topical, intralesional, and systemic chemotherapeutic agents—including topical 5% imiquimod, 5-fluorouracil, cisplatinum, doxorubicin, bleomycin, and interferon
- Vismodegib and sonidegib are orally active hedgehog pathway inhibitors FDA approved for **metastatic BCC**, **recurrent BCC** postsurgery, and **locally advanced BCC** in patients who are not candidates for surgery or radiation.

Slide 30

BCC-Management

- Orbital Invasion and Metastases
  - Surgical exenteration
  - Radiation
  - Vismodegib

Slide 31

Squamous Cell Carcinoma

- Erythematous scaly patch or slightly elevated plaque that often arises within sun-exposed skin of an elderly individual. However, it can develop in younger individuals with significant photodamage or in sun-protected sites.

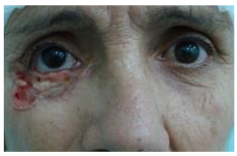
Slide 32

SQUAMOUS CELL CARCINOMA

Slide 33

SCC-Epidemiology

- 5% to 10% of eyelid malignancies in whites
- ore aggressive tumor with a higher risk of perineural, nodal, and distant spread.



Slide 34

SCC –Risk Factors

- M>F
- Avg age >60
- Sun-exposed skin
- Hereditary syndromes:
  - Xeroderma Pigmentosum
  - Oculocutaneous albinism
  - Dyskeratosis congenita
  - Huriez syndrome
  - Epidermodysplasia verruciformis
- White race (Fitz 1-3)
- Precursor lesions
- Immunosuppression
- Chronic inflamm conditions
- HPV
- Env exp
  - Ionizing radiation
  - Arsenic
  - Alkilating agents
  - Others

Slide 35

SCC Pathogenesis

- Mutation in DNA keratinocytes
- AK is initial step to development of SCC

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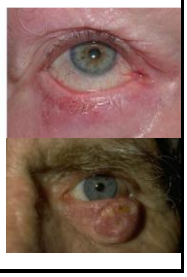
SCC-Clinical Features

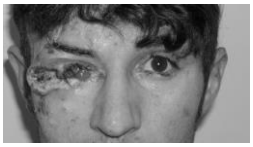
- Raised, wartlike, rough/scaly
- Bleed/crust
- plaque, papule, or nodule, or it may be exophytic
- The lower lid is the most common site for SCC, accounting for approximately 60% of lesions; the upper lid is the least common site.



Slide 37

Invasive SCC clinical features

- Invasive SCC: may manifest as plaques, large ulcerated lesions, or papillomas. Invasive tumor usually occurs in the sun-exposed skin areas of elderly persons with lightly pigmented skin

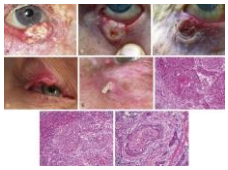


A large eyelid squamous cell carcinoma that invaded the orbit in a patient with **epidermodysplasia verricuformis**.

Slide 38

Invasive SCC

- Most often at lid margin.
- Most common on the lower lid, followed by the medial canthus, the upper lid, and the lateral canthus.



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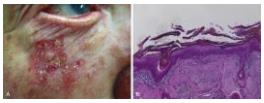
In Situ SCC (Bowen's disease)

- Full thickness of the epidermis is replaced by atypical keratinocytes with mitotic figures at different levels.
- Slowly progressive, well-defined erythematous, scaly plaque
- SCC in situ has a 3% to 8% risk of malignant transformation to invasive SCC.



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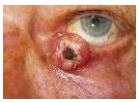
Keratoacanthoma

- Keratoacanthoma (KA) is regarded by some as a type of well-differentiated SCC with a strong propensity for spontaneous regression and by others as a distinct non-metastasizing clinicopathologic entity
- Manage these lesions as SCC with a margin controlled excision
- KA is much less common than SCC in the periocular area.



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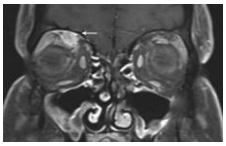
Perineural invasion (PNI)

- Neurotrophic tumors
- May spread into orbit via CN V, VII, and extraocular motor nerves
- asymptomatic, incidental microscopic finding in 4% to 8% of periocular SCC.
- Clinical PNI carries worse prognosis
- Symptoms: dysesthesia, fornication, pain, motor deficit
- MRI imaging



Slide 42

Orbital Invasion

Contiguous invasion or PNI

S/S: EOM restriction, epiphora, dysesthesia

Slide 43

SCC Metastasis

- 2-10%
- Reginal LN to preauricular or submandibular LN

Slide 44

SCC Pathology

- SCCs arise from epidermal keratinocytes and consist of malignant cells with large vesicular nuclei and an abundant eosinophilic cytoplasm. These cells can form nests, sheets, or infiltrating cords

Slide 45

SCC Prognosis

Recurrent tumor	X3 local recurrence risk, X5 metastasis risk
Size	X2 metastasis risk in >2 cm SCC
	1.7% recurrence in tumors <1 cm, 5% recurrence in tumors >5 cm
Poorly defined or rapidly growing tumors	May have a higher risk of recurrence
Thickness	16% risk of metastasis in tumors >6 mm
Depth of invasion	Perhaps less applicable in periocular area because of thin skin and lack of subcutaneous tissue
Location	Auricular, nasal, and periocular tumors have higher rates of recurrence and metastasis
Histologic differentiation: moderately or poorly differentiated	28.6% recurrence for poorly differentiated versus 13.6% for moderately differentiated
	22.8% metastasis for poorly differentiated versus 9.2% for moderately differentiated.
Histologic subtype	Adenoid, adenosquamous, and desmoplastic SCC are high risk
Extratumoral perineural invasion	Up to 47% recurrence rate
	Up to 33% metastasis
	Worse prognosis with nerve size >0.1 mm
Metastasis	55%–75% 5-year mortality rate if regional or distant metastases present
Immunosuppression	Higher risk of local recurrence and metastasis
Chronically injured skin (e.g., previous radiotherapy, thermal injury, and chronic ulcers)	Higher risk of local recurrence and metastasis
Preexisting SCC in situ	Higher risk of metastasis
Age >60 years	Two times recurrence risk
Male	Higher recurrence risk

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SCC Investigation and Staging

- Clinical examination of cranial nerves and regional nodes.
- Nodal assessment with imaging (CT, MRI, positron emission tomography (PET) scan, or ultrasonography) or SLNB should be considered in tumors greater than 20 mm in size, recurrent tumors, tumor greater than 4 mm in thickness, and those with PNI.

Slide 47

SCC In-Situ Management

- Predominantly surgical
- Wide excision, frozen section control, and MMS
- Insitu (non-periocular):
  - CryoTx
  - Curetagge/cautery
  - Imiquimod
  - PDT
  - XRT

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Invasive SCC

- Sx with margin control (MMS, frozen, paraffin)
- Sx exc with 4mm margin
- XRT as adjunctive tx for high risk
- ChemoTx: Palliation or adjunctive for advanced tumors
  - cisplatin, doxorubicin, bleomycin, peplomycin, methotrexate, capecitabine, and 5-fluorouracil

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PNI management

- Margin-controlled excision, exenteration, or skull base surgery followed by adjuvant radiotherapy

Slide 50

Orbital SCC

- Exenteration
- LN dissection
- XRT
- ChemoTx

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SCC Prognosis

- The majority of local recurrences occur in the first 3 years
- Overall disease-specific mortality rates vary between 1.6 and 6.2%
- Regular follow-up, sun protection, and skin checkups are advisable. Clinical or radiologic nodal surveillance should be considered in high-risk tumors.

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Slide 53

SEBACEOUS GLAND CARCINOMA

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Sebaceous Gland/Cell Carcinoma

- Meibomian glands, glands of Zeis, and sebaceous glands in the caruncle or brow

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SGC—Risk factors

- Older
- Female>Male
- South/SE Asians
- h/o facial irradiation
- Muir-Torre Syndrome
- Retinoblastoma

Slide 56

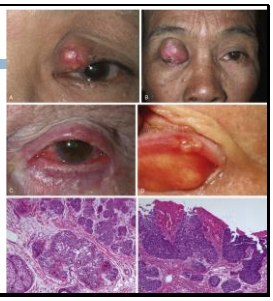
SGC Pathogenesis

- Germline mutation of on allele of DNA mismatch repair gene, followed by a "second hit" allows tumor development

Slide 57

SGC—Clinical Features

- Masquerader
- 2x more common on upper eyelid



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SGC—Clinical Features

- Uninflamed tarsal nodule or mass, →yellowish color, leads to destruction of lid margin architecture and madarosis
- Misdiagnosed as chronic chalazion recalcitrant to incision and curettage
- Diffuse pseudoinflammatory pattern, in which diffuse eyelid thickening is seen with inflamed conjunctiva
- Tarsal conjunctiva may have a yellowish appearance because of the presence of lipid droplets
- Strong propensity for intraepithelial spread either in the form of Bowenoid in situ disease or pagetoid spread into adjacent conjunctiva and eyelid skin
- spread into orbital soft tissue, the lacrimal gland, the lacrimal drainage system, and the cranial cavity
- Regional lymph nodes and also to the liver, lung, bone, and brain

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SGC Pathology

- Multivacuolated cells with scalloped nuclei, cytologic atypia, increased mitotic activity, variable necrosis, and infiltrative growth
- **Lipid staining** (with oil red O or Sudan black stains) can be performed on frozen sections, and immunochemistry (BER-EP4, EMA, apidophilin, and androgen receptor)

Slide 60

SGC Investigation and Staging

- Multiple preoperative or intraoperative **mapping** conjunctival biopsies from at least all four **bulbar** quadrants and the medial and lateral **tarsal** conjunctiva of both **upper** and **lower** eyelids should be used to look for diffuse **intraepithelial spread**
- In large (>20 mm) tumors, suspected lymphadenopathy, perineural invasion, or poorly differentiated histology, nodal imaging or SLNB should be considered

Slide 61

SGC Management

- Sx excision with 5mm margins and/or margin control (paraffin, MMS, frozen section)
- Orbital involvement usually necessitates exenteration, and nodal involvement is cleared with neck dissection

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Adjunctive Treatment

- *Cryotherapy:* A double freeze–thaw cycle is applied to the bulbar and palpebral conjunctiva
- *Radiotherapy:* This has been used as a primary and as an adjunctive treatment, particularly after regional lymph node dissection
- Chemotherapy:
  - Topical MMC for diffuse intraepithelial invasion
  - Systemic: 5FU, platinum-based agents

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SGC Prognosis

- Recurrence 4-28%
- Mortality 5-10%
- Poorer px: upper and lower eyelid involvement, orbital invasion, lymphovascular invasion, poor differentiation

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Melanoma

- 1% eyelid tumors
- Risk factor: intense intermittent UV exposure, light skin, changing nevus, dysplastic nevus syndrome, >50 nevi >2mm in size, FHx, congenital nevi, immunosuppression, sunburns, tanning beds

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Pathogenesis

- Accumulation of genetic and molecular mutations that damage tumor suppressor factors and promote cell growth factors

Slide 66

MELANOMA

Slide 67

Melanoma—Clinical Presentation

- De novo or pre-existing nevus
- Lower eyelid more common
- ABCDE
  - Asymmetry
  - Border
  - Color
  - Diameter>6mm
  - Elevated surface/enlarging

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Melanoma—clinical presentation

- Three major types
  - Lentigo Maligna Melanoma
  - Superficial Spreding Melanoma
  - Nodular Melanoma

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Lentigo Melanoma

- Irregular, flat, pigmented lesion most commonly found on the temple, cheeks, and nose
- May extend over the lid margin onto the conjunctiva as primary acquired melanosis (PAM)
- 3% to 8% risk of developing invasive melanoma→Lentigo Maligna Melanoma (LMM)
- Tx: Margin-controlled paraffin sections

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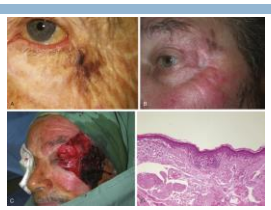
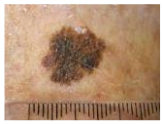
LMM



Slide 71

Superficial Spreading Melanoma

- Fifth decade of life and may arise from a pre-existing melanocytic nevus. These tumors are initially flat, with variegated colors and an irregular border



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Nodular Melanoma

sixth decade and usually arise de novo

elevated, symmetric tumors with regular borders and a single color

relatively rapid changes (over weeks and months rather than days) occurring in size, elevation, or bleeding.

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Melanoma-Pathology

- Melanocyte progenitor cells in the epidermis
- Variably sized and shaped nests of melanocytes with pagetoid spread, and they may ulcerate
- Growth patterns:
  - **Biphasic** pattern, initial radial growth confined to the epidermis (in situ melanoma) is followed by a vertical growth phase, in which there is invasion into the dermis with expansile growth and mitotic activity
  - **Monophasic** pattern, which occurs in **nodular** melanomas, there is only a vertical phase

Slide 74

Pathology

- Transformation to melanoma:
  - Dysplastic nevi (up to 6% lifetime risk),
  - Giant or large congenital nevi (approximately 2% lifetime risk)
  - Oculodermal melanocytosis (approximately 0.25% lifetime risk). Other nevi carry minimal risk

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Melanoma—Investigation and Staging

- Biopsy:
  - Excisional Bx for confirmation and Breslow thickness (Given the significance of the thickness in determining recurrence risk and survival, shave biopsies should be avoided)
  - Regional lymph nodes and distant metastases are present in around 0% to 5% and 0% to 3% at presentation in series of eyelid melanomas
  - Head and neck imaging (MRI or CT) with ultrasound-guided fine-needle aspiration for suspicious nodes should be done for intermediate-risk to high-risk melanomas. SLNB should be considered in tumors 1 mm or greater in thickness

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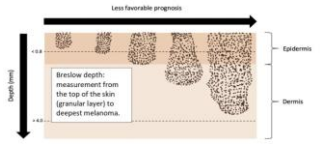
Melanoma--Staging

- Breslow depth (the distance in millimeters between the upper layer of the epidermal stratum granulosum and the deepest point of the tumor penetration), mitotic rate, and ulceration



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Melanoma--Management

- Excision with clear margins
- Palliative sx (exenteration)

- Current WHO guidelines recommend **5-mm margins** for in situ disease, **1-cm margins** for tumors less than 1 mm thick, and 1 to 2 cm margins for those 1.01 to 2 mm thick. However, in the **periocular region**, the literature has described the use of **5-mm margins** for tumors less than 2 mm thick with good local disease control.

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Melanoma--Management

- Known nodal metastases, neck dissection and parotidectomy should be conducted at the time of excision of the primary lesion

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Melanoma—Adjuvant Tx

- XRT in larger lesions as a means of avoiding exenteration and preserving the globe
- Topical therapies such as mitomycin C and interferon can be considered for conjunctival extension of in situ disease

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Melanoma—Pharmacotherapy

Topical imiquimod has been used for large LM in patients who decline or are unfit for surgery.

Targeted chemotherapies are being developed for melanoma treatment. These include BRAF inhibitors (e.g., vemurafenib, dabrafenib, and encorafenib) and the MEK inhibitor trametinib

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Melanoma--Prognosis

- Local recurrence 21%, regional mets 11%, distant mets 6%
- Stage 0 – **annual** skin monitoring.
- Stage IA – skin and lymph node examination every 3 to 12 months for 5 years and then annually.
- Stage IB–IV – examination of skin and lymph nodes every 3 to 6 months for 2 years and then every 6 to 12 months for the next 2 years and then annually. **Nodal imaging surveillance**, chest radiography, and blood tests every 6 to 12 months should be considered for high-risk tumors.

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MERKEL CELL CARCINOMA

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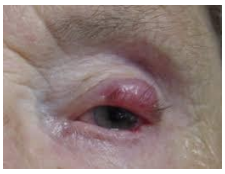
Merkel Cell Carcinoma

- Rare in periocular region
- Risk of local recurrence and regional LN spread



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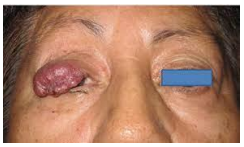
MCC Risk Factors

- White, male
- UV light
- Immunosuppression
- Possible viral contribution



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MCC Pathogenesis

- MCC is generally thought but not proven to derive from Merkel cells in the epidermis.
- It may arise from dermal neuroendocrine cells or pluripotent stem cells.
- **Polyomavirus** is found to be clonally integrated in 80% of cases of MCC and is likely to have an oncogenic role in many cases

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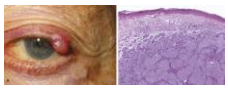
MCC clinical features

- rapidly growing, dome-shaped, firm painless, nodule with a shiny, telangiectatic surface
- red, violaceous, or flesh-colored
- more common on the upper eyelid near the lid margin, where they cause local madarosis.
- all patients should have clinical **regional nodal examination.**



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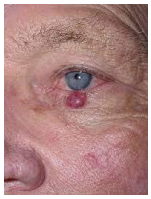
MCC Pathology

- poorly differentiated neuroendocrine carcinoma composed of small to medium-sized atypical cells with large, hyperchromatic nuclei
- cohesive nests, trabecular structures, or more diffusely within the dermis and may extend into the subcutis



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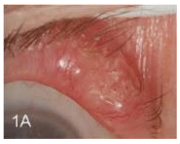
MCC Investigations and Staging

- All patients should be staged with head and neck imaging (preferably MRI and possibly PET scans) because of the risk of nodal spread
- SLNB can also be considered



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MCC Management

- Surgery: 5-mm margins with margin control are often used. Paraffin sections are often utilized, although frozen sections or MMS can also be used, depending on the expertise of the pathologist or Mohs surgeon
- XRT: adjunctive tx may improve regional control and survival
- Chemo: carboplatin, etoposide, cyclophosphamide, doxorubicin, and vincristine

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MCC Prognosis

- Local recurrence occurs in 14%, nodal spread in 20%, and distant metastases in 5% of periocular MCC
- disease specific mortality rate is 6%

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OTHER TUMORS

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Lymphoproliferative Tumors

- Painless eyelid mass, diffuse eyelid swelling, and skin nodules or plaques and may be bilateral
- Peripheral T-cell lymphoma; natural killer or T-cell lymphoma; mucosa-associated lymphoid tissue (extranodal marginal zone) B-cell lymphoma; diffuse large B-cell lymphoma; CD30-positive lymphoproliferative disorders, including lymphomatoid papulosis, primary cutaneous anaplastic large cell lymphoma, and systemic anaplastic large cell lymphoma; mantle cell B-cell lymphoma; and small cell lymphocytic B-cell lymphoma or chronic lymphocytic leukemia (leukemia cutis)
- Mycosis fungoides and Sezary syndrome most common cutaneous t-cell lymphoma
- Erythematous plaques that may be misdiagnosed as eczema, blepharitis, or discoid lupus.
- Cicatricial ectropion may develop, followed by necrosis and ulceration

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Metastatic lesions

- Melanoma (skin and uveal) and breast, kidney, thyroid, prostate, and lung cancers
- Nontender nodule but can be as a flat pigmented lesion, diffuse eyelid edema, or multiple nodules

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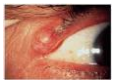
Eccrine/Apocrine Tumors

- Primary Cutaneous Mucinous Carcinoma (Mucinous Sweat Gland Adenocarcinoma)
- Endocrine Mucin-Producing Sweat Gland Carcinoma
- Signet Ring Carcinoma (Histiocytoid Carcinoma/Adenocarcinoma of Eccrine Sweat Glands)
- Apocrine adenocarcinoma
- Microcystic Adnexal Carcinoma (Sclerosing Sweat Duct Carcinoma/Syringomatous Carcinoma)
- Porocarcinoma



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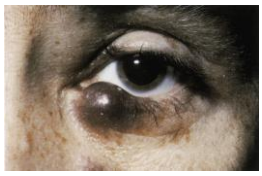
Vascular tumors

Kaposi's sarcoma

- HHV-8 infection
- Vascular proliferation
- HIV+pts grow rapidly
- Ashkenazi jewish males, mediterranean
- African/endemic
- Immunocompromised



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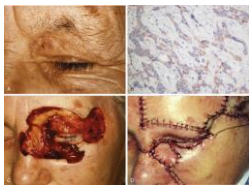
Vascular tumors

Angiosarcoma

- Arises from endothelial cells
- Rapidly expanding red or violet lesion followed by a solitary blue or violet nodule but can be multifocal, causing diffuse eyelid swelling
- Patterns: superficial spreading, nodularity, and ulceration
- Px: poor, 75-90% mortality rate



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Hair Follicle Tumors

- Hair matrix tumor (pilomatrix carcinoma), external hair sheath tumors (trichilemmal carcinoma), malignant proliferating trichilemmal tumor, and hair germ tumors (trichoblastic carcinoma)

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Soft Tissue Tumors

- Superficial Undifferentiated Pleomorphic Sarcoma (UPS)
- Dermatofibrosarcoma Protuberans
- liposarcoma,
- myxofibrosarcoma,
- rhabdomyosarcoma,
- hemangioendothelioma
- malignant peripheral nerve sheath tumor.

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Thank you!

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