Malignant Cutaneous Neoplasms

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Objectives:

1. Identify common cutaneous malignant neoplasms.

2. Identify the etiology, pathophysiology and treatment of common malignant cutaneous neoplasms.

3. Synthesize the information to identify common malignant cutaneous neoplasms.

4. Discuss pharmacologic management of common malignant cutaneous neoplasms.
Disclosures

- The speaker has no conflict of interest relevant to this topic
Actinic Keratosis

Thick crusty, erythematous scale

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Pigmented Actinic Keratosis

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Etiology/Pathophysiology

More likely

- Over 40, more common in men.
- Have a history of frequent or intense sun exposure or sunburn.
- Have pale skin, red or blond hair, and blue or light-colored eyes.
- Progressively lower incidence in Fitzpatrick 1-5, almost nonexistent in 6.
- Personal history of an actinic keratosis or skin cancer.
Etiology/Pathophysiology

- Have a weak immune system as a result of chemotherapy, chronic leukemia, AIDS or organ transplant medications.

- If not treated they have a 20% chance of conversion to SCC.

- A skin biopsy to confirm the diagnosis and rule out invasive squamous cell carcinoma for suspicious or more advanced lesions (hyperkeratosis, increased erythema, or induration or nodularity) or if they are recurrent that did not respond to therapy.

- UV-induced mutations in key genes, including TP53 and deletion of the gene coding for p16 tumor suppressor protein.
Etiology/Pathophysiology

- Dysplasia and architectural disorder of the epidermis.
- Abnormal keratinocytes of the basal layer, variable in size and shape
- Altered cellular polarity
- Nuclear atypia
- Hyperkeratosis/parakeratosis of the epidermis
- Irregular acanthosis may be present
- In general, hair follicles, sebaceous glands, and apocrine and eccrine ducts are not involved
Appearance

- Small red, brown, flesh colored, pink, or white scaly patches that do not go away.
- Distribution pattern on face, ears, scalp, hands, but may occur anywhere.
- May be numerous and coalesce.
- If they itch, are tender, or bleed they need to be biopsied.
Treatment

- Cryotherapy.

- Photodynamic Therapy after treatment with ALA (Levulon) that activates the light in the AK to destroy the lesion.

- Electrodesiccation and curettage.

- Shave biopsy to treat and assess that it has not converted.

- Chemical peels
Treatment

- Laser resurfacing.

- Topical agents such as 5-FU (Effudex, Carac both may increase nodular BCC), Imiquimod (Aldara, Zyclara), Picato, and diclofenac (Solaraze, Voltaren) gel,

- AK is a field disease and needs to be treated as such:
  - Cryo
  - PDT the area
  - Cryo
  - Topical agents.
Discuss Prevention

- UVA, UVB, UVC waves.
- Ultraviolet rays are electromagnetic radiation with a wave length less than that of visible light.
- UVA penetrate deeply into the dermis and subcutaneous tissue (may easily penetrate glass).
- UVB penetrate into the epidermis and upper dermis.
- UVC usually bounce off the ozone layer.
- UVA waves are associated with autoimmune disease.
Prevention

- UVA, UVB, and UVC can all damage collagen and accelerate aging.

- UVA and UVB destroy Vitamin A in the skin further damaging collagen and accelerating photo-aging.

- UVA was not recognized to be as harmful but it creates many oxygen free radicals which cause further DNA damage.

- UVA is immunosuppressive. Because UVA is not measured by most SPF testing there is no good measurement for UVA protection. According to the Skin Cancer Foundation, even if it’s not hot outside, 50 to 80 percent of UV rays still burn right through the clouds.
Prevention

- UVA is immunosuppressive. Because UVA is not measured by most SPF testing there is no good measurement for UVA protection.

- According to the Skin Cancer Foundation Even if it’s not hot outside, 50 to 80 percent of UV rays still burn right through the clouds.
Prevention

- UVA waves give a quicker tan but a non-protective tan.
- UVB waves take about two days to begin to create a tan but the tan is protective.
- However, the production of melanin by UVB is called melanogenesis and direct DNA damage occurs.
- Melanogenesis increases the risk of cutaneous carcinoma.
Prevention

- Broad rimmed UV hat.
- Broad rimmed sunglasses.
- Avoid mid day sun.
- Wear sunscreen daily.
- Avobenzone, Zinc and Titanium Oxide reduce UVA.
- Think of the bucket theory with sunscreen.
Clinical Pearl

- If it is itchy or tender biopsy it.
- Remember that actinic keratosis is a field disease and needs to be treated as such.
- Nothing like MOHS on a patient with BCC or SCC that has been recurrently treated you may be there for 6 or more stages.
- Recent study shows if severe hypertension post PDT, likely to have BCC.
Basal Cell Cancer

Crusted, ulcerated, bleeds easily

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BCC

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Nodular Basal Cell Cancer

Slowly developed, bleeding, papule that has been there

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Superficial Basal Cell Cancer

Pink patch of skin that has been there

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Pigmented BCC
Etiology/Pathophysiology

- Most common skin cancer.
- Represent 90% of cutaneous carcinoma.
- Almost never metastasize but can cause significant local destruction.
- Risk factors include: low Fitzpatrick score, sun exposure, weakening of the immune system by disease or medication.
- 3 in 10 Caucasians will develop a BCC in their lifetime.
Appearance

- Appears as a small, dome shaped bump covered with telangectasias.
- It may appear pearl like or shiny/translucent.
- They are usually somewhat firm to the touch.
- Appear as a “pimple that comes and goes or comes and stays or a patch that comes and goes or comes and stays”.
Types of BCC

- Superficial BCC
- Nodular BCC
- Pigmented BCC
- Cystic BCC
- Infiltrative BCC
  - Morpheaform
  - Micronodular BCC
Types of BCC

- Neviod Basal Cell Carcinoma Syndrome also known as Gorlin syndrome.
Shave Biopsy to determine diagnosis but all biopsies that involve skin cancer require excision or other treatment beyond the biopsy even if margins are negative.

- EDCT
- Topical
- Excision
Treatment

- MOHS
- Topical Imiquimod, Picato under aluminum disc
- Radiation therapy
- PDT with photosensitizer (ALA)
- Vismodegib, (Erivedge)
Pearls

- If a BCC occurs at a site where you have had recurrent cryo...always MOHS.
- The pimple or patch that comes and goes.
Squamous Cell Cancer

Crusted scale, ulcerated, rapidly developed

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Squamous Cell Cancer

Thick scale, itchy, and slightly painful. Been there 1 year

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Squamous Cell Cancer

Red border with yellow scale

Has had kidney transplant and immune suppressed

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Etiology/Pathophysiology

- Second most common.
- Rarely fatal except in transplant and immune compromised patients where mortality can exceed 20%.
- Linked to alcohol, HPV, and tobacco as well as UV.
- Unlike BCC, SCC more linked to chronic rather than intense intermittent UV exposure.
- PUVA is a risk factor.
Etiology/Pathophysiology

- Histologically unique uncontrolled proliferation of squamous cells (presence of keratin, desmosomes, and/or tonofilament bundles into dermis and surrounding tissues).
- Squamous cell in situ the tumor has not penetrated the basement membrane.
- Bowens is UV induced and is an early form of SCC.
- Keratocanthoma is rapidly growing.
- One method to identify a SCC is by its appearance under the microscope.
Appearance

- An ulcer or plaque with scaling, ulceration, and/or crusting.

- Frequently on sun exposed skin.

- May present as a cutaneous horn (height greater than width).

- Peri-neural involvement is associated with pain, numbness, change in vision, or muscle twitching.
Treatment

- Surgical excision.
- MOHS
- EDCT on low risk lesions, low risk sites, and low risk patients.
- Radiation.
- If patients have invasive SCC, on high risk areas, in high risk patients consider imaging for metastasis first.
Pearls

- If the height of a cutaneous horn exceeds the width it is more apt to be benign.
- If more than 50% of the base of a lesion is erythematous it is more likely to be a BCC.
- Immune compromised patients are at high risk for metastasis and may need imaging prior to treatment.
Melanoma

New lesion, dark and asymmetric

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Melanoma

When you see it with a derm-llite it is so easy

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Nodular Melanoma
Melanoma upper arm referred
Other melanoma on same patient with FSE

© Kathleen Haycraft
Dermlite melanoma
Melanoma Chest

© Kathleen Haycraft
Dermlite melanoma

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Lentigo Maligna Melanoma

Chromically UV exposed skin

Peppering

Changing small structures

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Melanoma is a malignancy arising from the melanocyte.

Less common than basal or squamous but results in 75% of the deaths from cutaneous carcinoma.

Caused by damage to the DNA by UV rays.

Genetic links with BRAF, MEK, CDND2A.

Genetic risk is higher with associated BRCA mutations.

Eumelanin is more protective than pheomelanin.
Appearance

- May appear as
  - Asymmetric
  - Irregular border
  - Black, gray, blue, red, white or multiple colored lesion
  - White veil
  - Greater than 6 mm diameter
  - Evolving

- Nodular elevated, firm, and growing.
Treatment

- Biopsy properly.
- Treatment indicated by:
  - Breslow depth (Clark value with thin areas)
  - Ulceration
  - Mitotic Index
  - Other factors

Surgery, SLNB, chemotherapy, radiation, interferon, and vaccine.

Lentigo maligna is a field disease.
Dysplasia, mild, moderate, severe, Clark’s etc.

Asymmetric

Irregular Border

Dark Black

6mm

It is getting bigger

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Severe Dysplasia

- This was a severe
What do you do with this?

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Moderate Dysplasia

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Etiology/Pathophysiology

- Atypical nevi (Dysplastic nevi). Atypical to the eye and dysplastic to the microscope.

- Some atypical nevi become melanoma but the majority do not.

- Roughly half of melanomas arise in these dysplastic nevi whereas the other half arise de novo.

- Melanomas can occur on sun or non-sun exposed sites.
Etiology/Pathophysiology

- Cytological atypia is of more significance than architectural atypia.
- Individuals with dysplastic nevus syndrome have an associated CDKN2A gene defect.
- If 5 or more, 10 times increased risk of melanoma.
ABCDE are helpful with these nevi.

Ugly duckling...the pretty duckling...what doesn’t belong with the rest.

Cutaneous melanoma.

Acral melanoma.

Uveal melanoma.
Treatment

- Individuals with multiple dysplastic nevi should be monitored in a dermatology setting annually.

- Some dermatologists excise all, some monitor, our system is:
  - Mild….Monitor
  - Moderate….Excise with 2 mm margin
  - Severe…Excise with 9 mm margin
Merkel Cell Cancer

Asymptomatic

Pink violaceous plaques

Associated telangectasias

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Merkel Cell Cancer

He was covered with these before death

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Merkel cell carcinoma (cutaneous neuroendocrine tumor) is a malignant solid tumor derived from the Merkel cell.

The Merkel cell is a mechanoreceptor that is present in the basal layer of the epidermis and surrounds the hair follicles.

The tumor usually presents as a solitary lesion on the head or neck but may present as multiple lesions and on any area. The incidence of Merkel cell carcinoma (MCC) in the United States is approximately 1500 cases per year\(^1\). As of 2005, the disease incidence had more than tripled over the previous 20 years\(^1\).
Etiology/Pathophysicsology

- There is an association of MCC with the polyomavirus (MCPyV).
- Eighty percent of MCC are infected with the virus versus 20% skin not involved with MCC.
- Studies confirm that MCC patients who harbor the MCPyV have a better prognosis than those who do not.
The tumor usually presents as a solitary lesion on the head or neck but may present as multiple lesions and on any area.

- A = Asymptomatic.
- E = Expanding rapidly.
- I = Immune suppressed.
- O = Older than 50 years.
- U = UV-exposed skin.
Appearance MCC

- Occurs in the elderly (greater than 65 years of age), male, immunosuppression and fair-skinned patients with chronic UV exposure.

- Generally presents as a rapidly growing, non-tender, erythematous to violaceous nodule that has a shiny appearance.

- It is generally associated with overlying telangetasias.
Treatment

- Contrasted to melanoma with an average 5 year 15% mortality rate, MCC has a 49%.
- With proper staging this can increase to 79% survival rate with proper staging and treatment.
- There is a 4 tiered system for treatment selection that ranges from excision, SLNB, chemotherapy, radiation, or supportive care.
Mycosis Fungoides

Patch

Slightly pruritic

Has been treated and never gets better

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Etiology/Pathophysiology

- Cutaneous T-cell Lymphoma.
- Etiology is unclear.
- Over 20 years of age, and it is more common in men than women.
- Common in sites covered with “underwear”.
- Unusual expression of CD4 T cells.
- May progress to Sezary syndrome 3:100,000.
Presentation

- Rashes, patches, or tumors that are pruritic in 20% of the cases.
- Frequently resembles eczema.
- May take many years and many biopsies to diagnose.
Treatment

- Depends on stage:
  - Many never progress and rely on UVB and topical steroids or topical nitrogen mustard
  - Sezary vorinostat (Zolinza) and other chemotherapies.


Let’s Have Some Fun

Tired of ideas of laser and filler

His face lift

© Kathleen Haycraft
Asymptomatic, Evolving

© Kathleen Haycraft
Getting darker

© Kathleen Haycraft
Slowly getting bigger

© Kathleen Haycraft
Got bigger quickly

© Kathleen Haycraft
Slowly evolving pink patch
Crusty and itchy...been there awhile

© Kathleen Haycraft
Little pimple that won’t go away

© Kathleen Haycraft
Crusty area keeps bleeding
Favorite Sites

- Websites for patients and providers:
  - [http://www.mayoclinic.com/health/DiseasesIndex/DiseasesIndex](http://www.mayoclinic.com/health/DiseasesIndex/DiseasesIndex)
  - [http://www.dermnetnz.org/sitemap.html](http://www.dermnetnz.org/sitemap.html)