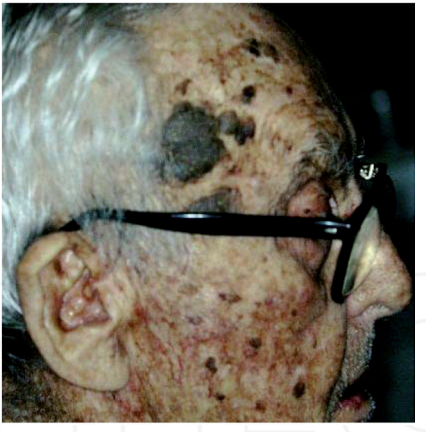
Skin Lesions

Seborrheic keratosis (dermatosis papulosa nigra)
Basal cell carcinoma
Melanotic macule
Melanoma
Actinic lentigo
Perioral dermatitis
Latex contact dermatitis

1. Seborrheic keratosis (dermatosis papulosa nigra)



Description: The most common benign epithelial tumors, seborrheic keratoses begin as small "warty" papules with or without pigmentation, which may evolve to plaques with a "stuck on" appearance. They may present as isolated or generalized lesions on the face, trunk, and upper extremities. They tend to increase in numbers over the individual's lifetime. Physical examination reveals a greasy feel and a flat nodule, which may be brown, gray, black, or skin colored, round, oval or irregular, and have a fine stippled texture.

In African Americans, Black Africans, and deeply pigmented South East Asians the lesions appear as myriad tiny to enlarging raised black lesions.

Epidemiology and Etiology: The lesions are hereditary, occurring more commonly and more extensively in males, and they do not appear until after age 30.

Treatment: Light electrocautery can be used to remove the lesions and to prevent recurrence; but this method precludes histopathological verification. Cryosurgery works only on flat lesions and recurrences are possible. Cryosurgery followed by curettage permits histopathologic examination. Solid black lesions should be excised with a punch biopsy to rule out malignant melanoma. Hypopigmentation can occur in darker skinned individuals where keratoses have been removed.

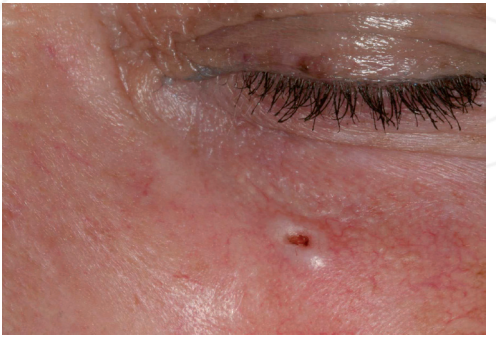
Prognosis: The lesions are seen with increasing age and are benign. They do not become malignant.

Differential diagnosis: The differential diagnosis includes solar lentigo, actinic keratosis, lentigo maligna, lentigo maligna melanoma, and basal cell carcinoma.

2. Basal cell carcinoma

Description: Basal cell carcinoma is the most common type of skin cancer, which can be locally invasive, aggressive, and destructive, but rarely metastasizes. It usually occurs on skin which has the capacity to develop hair follicles. It is rarely seen on the vermilion border of the lips.

The lesions may appear nodular, ulcerating, sclerosing, superficial and multicentric, and pigmented. They may begin as a papule or nodule with a translucent, pearly appearance and be skin colored with superficial telangiectasia. The nodules are usually well defined and firm and may present with focal ulcers with rolled borders (termed rodent ulcers). More than 90 percent of lesions occur on the face and most are isolated single lesions, although multiple lesions can occur. The "danger sites" for development are at the medial and lateral canthi of the eyes, the nasolabial folds, and behind the ears.



2.1. Epidemiology and Etiology

Fair skinned individuals and albinos and persons with a history of extensive sun exposure at young ages are more predisposed to develop basal cell carcinoma later in life. Age of onset is usually over age 40. BCC is seen more frequently in males, and is rare in brown or black skinned individuals. Previous radiotherapy for facial acne greatly increases the risk.

2.2. Treatment

Treatment options include excision with primary closure, cryosurgery or electrosurgery for small lesions, and radiation therapy. Microscopically controlled surgery (Mohs surgery) is the favored approach for excision of lesions in the "danger sites." Cryosurgery and electrocautery can leave scars. Radiation therapy is preferred when there is potential for disfigurement or in old age. Topical treatment with 5-fluorouracil ointment and imiquimod cream is an effective option which will not produce scars, but lengthy treatment is necessary.

2.3. Prognosis

Basal cell carcinoma does not metastasize and lesion sites respond well to surgical treatments. In the "danger sites," the lesions may invade deeper tissues and cause extensive destruction of muscle and bone. Death may result from invasion into the dura mater, hemorrhage of eroded large vessels, or infection.

2.4. Differential diagnosis

Diagnosis is usually made clinically and confirmed histologically. Lesions to consider in the differential diagnosis include dermatofibroma, superficial spreading and nodular melanoma, squamous cell carcinoma, syphilitic chancre, and nevomelanocytic nevi.

3. Melanoma

Description: Cutaneous melanoma is the most malignant tumor of skin structures, and its incidence is on the rise. It is responsible for 80% of deaths from skin cancer in the United States, with an estimated 8,650 deaths annually. The lifetime risk of developing melanoma in the United States in 2010 was 1 in 50. Melanoma represents 5% of newly diagnosed cancers in men and 6% of newly diagnosed cancers in women annually. Melanoma is among the most common cancer types in younger aged individuals. Deaths from melanoma also occur at younger ages than most other cancers. The most common type of melanoma is superficial spreading melanoma. Other types include nodular melanoma, lentigo maligna melanoma, and acral lentiginous melanoma. The TNM classification system is used to stage cutaneous melanomas.

Melanoma recognition is categorized into six signs:

Asymmetry in shape

A **border** which has irregular edges, which can be scalloped, notched, and clearly defined

Color which is not uniform but rather displaying mixed colors of brown, black, gray, blue, red, and white

Diameter which is greater than 6 mm

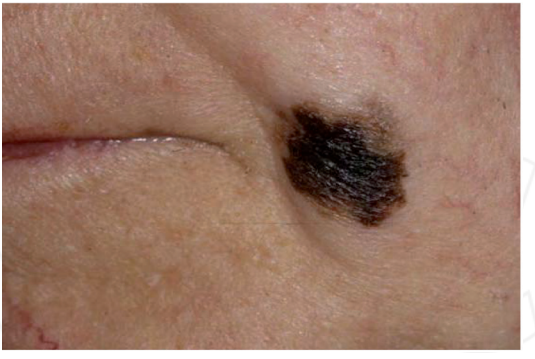
Elevation or surface distortion which can be assessed by side lighting, and

Evolving or increasing in size

Epidemiology and Etiology: The etiology of melanoma is unknown. However genetic predisposition and sun exposure are believed to be factors. Light skinned individuals and those who experienced sunburns during childhood or intermittent burns throughout their youth have a higher incidence. The presence of dysplastic melanocytic nevi, congenital nevomelanocytic nevus, and a family history of melanoma also increase the risk.

There is equal prevalence in males and females and the median age of diagnosis is 65. Rarely seen in brown or black skinned individuals, the highest incidence is in fair or light skinned white individuals, especially those with outdoor occupations and recreational habits. Oral melanoma accounts for less than 1% of all melanomas.

Treatment: Complete surgical excision with a 1 cm margin for lesions which are less than 2mm in thickness is the treatment of choice. Wide surgical excision is indicated for larger, more deeply invasive tumors. Clinically evident regional metastasis in the absence of distant metastasis warrants lymph node dissection. Hypofractionation and neutron beam radiation therapy may be used as adjunctive therapy in some patients. Chemotherapy and immunotherapy also show some promise for treatment.



Prognosis: Early detection and treatment of cutaneous melanoma before metastasis has developed is associated with a high 5-year survival rate. Thicker melanomas at discovery or those with regional lymph node metastasis have a poorer prognosis; and patients with

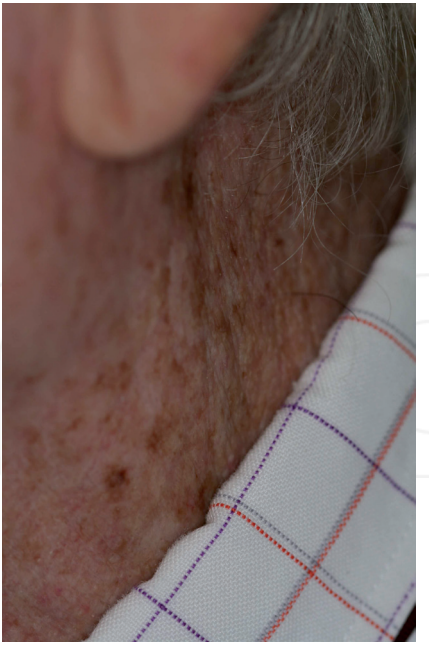
disseminated disease at time of diagnosis have a bleak prognosis. Cutaneous melanomas appearing between the scapulae on the back, the posterior upper arms, posterior and lateral neck, and scalp have worse prognoses. Prognosis is better for women and for patients younger than age 50. Patients with oral mucosal melanomas which are deeper than 0.5 mm and patients with nonpigmented lesions have a poorer prognosis. Continuous follow-up and monitoring are necessary.

Differential diagnosis: Seborrheic keratoses, solar lentigo, melanocytic nevus.

4. Actinic lentigo (lentigo solaris; solar lentigo; age spot; liver spot; senile lentigo)

Description: Actinic lentigo are benign, flat, brown to tan, evenly pigmented macules with well-demarcated irregular borders, commonly found on the dorsa of hands, on the face and arms of elderly Caucasian individuals. They are rarely seen before age 40 and are very common in individuals older than age 70.

Etiology: Chronic exposure to and damage caused by ultraviolet light



Treatment: No treatment is required, however the color intensity of the lesions can be reduced with the use of topical retinoic acid. Additionally, the lesion can be eliminated completely with a Q-switched ruby laser. Cryotherapy, topical hydroquinone, tazarotene, adapalene, and a combination of mequinol and tretinoin can also be used. Preventive therapy includes use of sunscreen.

Prognosis: Actinic lentigo do not undergo malignant transformation. New lesions can develop at other sites or adjacent to the original site. After removal, they do not reoccur in the same site.

Differential diagnosis: Ephelis (freckle), lentigo simplex, meiasma, seborrheic keratosis.

5. Perioral dermatitis

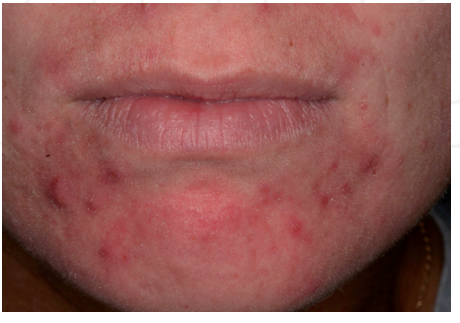
Description: Perioral dermatitis characteristically presents as multiple tiny erythematous papules, microvesicles, and papulopustules which are symmetrically grouped periorally. The nasolabial folds are often involved and a rim of spared skin is seen around the vermilion border of the lips. The lesions often coalesce and confluent plaques may appear eczematous with tiny scales.

Epidemiology and Etiology: Unknown. Seen predominantly in females between ages 16-45. May be aggravated by topical fluorinated glucocorticoids.

Treatment: Oral minocycline, doxycycline, or tetracycline. Topical metronidazole or erythromycin gel. Do not use topical fluorinated glucocorticoids. Caution patients about sun exposure.

Prognosis: The lesions typically show dramatic improvement over several weeks and resolve without recurrence in a few months.

Differential diagnosis: Allergic contact dermatitis, atopic dermatitis, seborrheic dermatitis, rosacea, acne vulgaris, steroid acne, sarcoidosis.



6. Latex (allergic) contact dermatitis

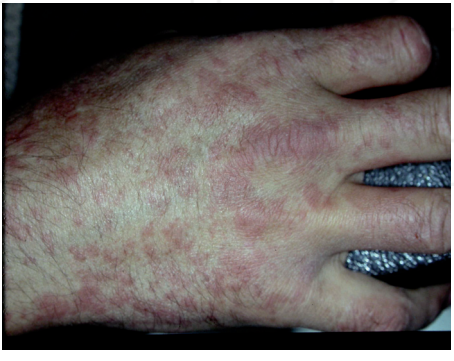
Description: Allergic contact dermatitis is a delayed cell-mediated hypersensitivity reaction which can occur at any age and accounts for 10-50 percent of occupational related illnesses in the United States. Sensitization can occur over weeks to years depending on the strength of the sensitizer. After exposure, the sensitized individual will develop skin eruptions confined to the site of exposure hours to days following the exposure. Repeated exposures will cause the eruptions to worsen. Symptoms include intense itching, stinging and pain, and in severe allergic contact dermatitis fever. Acute skin lesions present as well demarcated erythema and edema. Small papules and vesicles, bullae, and confluent erosions may also develop. Other, non-exposed sites may develop similar signs after several weeks. Chronic skin lesions may show thickening of the epidermis with plaques and scales, lichenification, papules, excoriations, and pigmentation. Chronic contact dermatitis exhibits spreading margins.

Etiology: Allergic contact dermatitis is caused by an allergen that elicits a cell-mediated delayed hypersensitivity reaction. It is an immunologic response, which only occurs in sensitized individuals.

Treatment: Management of the dermatitis involves removing the allergen. Topical glucocorticoids are indicated for mild to moderate cases. Systemic administration of glucocorticoids may be necessary for severe cases. Immunosuppression with oral cyclosporine may be necessary if the allergen cannot be completely avoided.

Prognosis: The dermatitis will reoccur upon re-exposure to the allergen and the eruptions will worsen with repeated exposures.

Differential diagnosis: Irritant contact dermatitis, atopic dermatitis, seborrheic dermatitis, psoriasis, epidermal dermatophytosis, fixed drug eruption, erysipelas phytophotodermatitis.



Additional reading

Actinic lentigo

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Perioral dermatitis

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Seborrheic keratosis

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Latex (allergic) contact dermatitis

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