Giant pigmented verrucous plaque on face

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CLINICAL FINDINGS

A 67-years old female came to our outpatient department complaining of large dark colored raised skin lesion over left side of the face since four years. Patient had been apparently normal till four years back, she noticed a single small dark colored raised asymptomatic skin lesion over the left side of the face. The lesion gradually enlarged over the last four years to attain the current size. The patient reported that the lesion was getting darker over the past few years. She did not take any treatment. There was no itching or pain in this lesion. On general physical examination her vitals were stable. Routine blood tests like CBC, Kidney function test, Liver function test and thyroid profile were within normal range. Abdominal sonography was done to exclude internal malignancy and the report came back as normal. She was on oral hypoglycemic drugs for her diabetes and diuretics for her hypertension. Local examination showed a large 4.5x 6 cm dark brown velvety plaque over the left side of the face extending to the scalp. Some areas within the plaque showed yellowish to brownish pigmentation, while others showed black coloration (Fig. 1, 2).

Fig. 1 Pigmented plaque in the front of left ear.

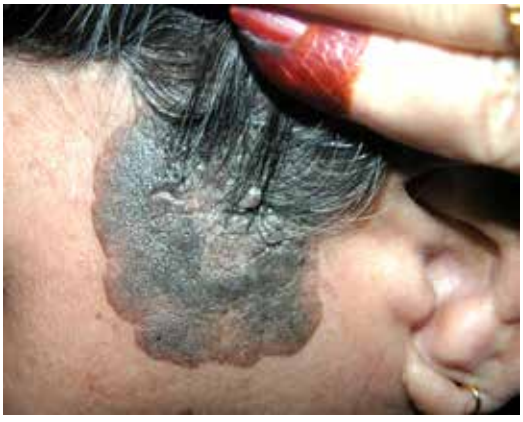

Fig. 2 Close up view showing pigmented plaque with velvety appearance.

What is your clinical differential diagnosis?

Lentigo maligna melanoma
Lentigo simplex
Seborrheic keratosis
Large cell acanthoma

PATHOLOGICAL FINDINGS

A Skin biopsy was taken from the lesion. Histopathological examination of the lesional skin

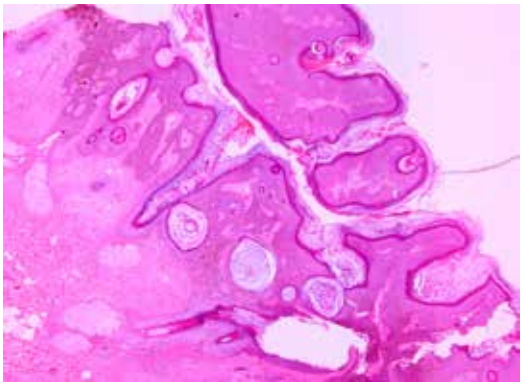

Fig. 3 Hyperkeratosis, acanthosis, papillomatosis, horn cyst and pseudohorn cyst.

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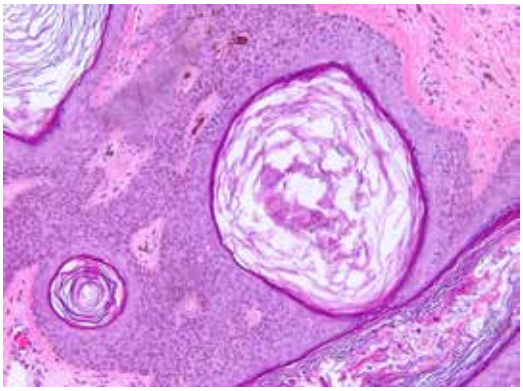

Fig. 4 Horn cysts and increased melanin pigmentation within the epidermis.

showed hyperkeratosis, acanthosis, papillamatosis, horn cyst and pseudo horn cyst (Fig. 3, 4).

DIAGNOSIS

Seborrheic Keratosis

DISCUSSION

Seborrheic keratosis is a benign non-melanocytic epidermal tumour composed of keratinocytes. It is seen more commonly in white skinned people, and usually appears in the fifth decade with equal male and female affection.[1] The disease displays varying morphological features and histopathological types. Family history should be sought if there are multiple seborrheic keratoses, and if associated with an early age of onset.[2]

Both aging and cumulative sunlight exposure were found to be independent contributory factors. Infection, bleeding, oozing and crusting are some of the complications associated with SKs.

Sudden appearance of multiple eruptive SKs "Leser - Trelat sign" is associated with gastric adenocarcinoma, due to the growth factors secreted by the tumour. The total number and size of seborrheic keratosis increases with increase in age.[4,5]

Genetic predisposition is seen and the disease is inherited in families as an autosomal dominant trait. Activating mutations in FGFR3 which provide proliferative signals to keratinocytes, increased expression of Bcl2 and Ki-67, increased DNp63a expression and activating PIK3CA mutations has been noted in the pathogenesis of SKs. Increased expression of keratinocyte derived Endothelin I mediated by TNF α and Endothelin Converting Enzyme 1α (ECE 1α) is linked to pigmentation seen in SKs.[1]

Seborrheic keratoses are usually asymptomatic, sometimes pruritic, starts as well defined round to oval slightly hyperpigmented papules or plaques ranging in size from few millimeters, but rarely exceeding 3 cm in size. It is commonly seen over face and trunk but also seen on neck and extremities.[6] Nipple, areola, vulva and conjunctiva are other unusual sites where SKs have been reported. Mucous membranes are spared in SKs.[7]

Lentigo maligna, actinic keratosis, melanocytic naevi, malignant melanoma and pigmented BCC are some of the differential diagnosis for SKs. seborrheic keratoses are usually diagnosed clinically.

Dermoscopy has revealed various characteristic patterns, which are helpful in differentiating SKs from other pigmented tumours. Comedone like openings (CL), Fissures and ridges (FR) and Sharp demarcation (SD) were consistent findings on dermoscopy in common SKs. Other findings were Finger print (FP), Moth eaten borders (ME), milia like cysts (ML) and network like structures (NL).[8]

A Skin biopsy shows hyperkeratosis, acanthosis with immature keratinocytes, papillomatosis with church steeple appearance, melanocytic proliferation, pseudo horn cysts with lower border which lies in a straight line with normal epidermis. Six histopathologic variants are seen: Hyperkeratotic, Acanthotic, Adenoid or Reticulate, Clonal, Irritated and Melanoacanthoma.[6]

Treatment is sought mainly for cosmetic reasons. Dyspigmentation and recurrence may occur following lesional clearance. A variety of techniques may be used to treat seborrheic keratoses. They include cryotherapy with carbon dioxide (dry ice)

The Clinicopathological challenge of Seborrheic keratosis

Diagnosis	Clinical	Pathological
Actinic keratosis	• Tan-brown, red or skin colored, circumscribed lesions, sandpaper texture • May have cutaneous horn (due to excessive production of parakeratotic scale)	• Basal cell and squamous layer atypia and disorderly maturation, hyperkeratosis, parakeratosis.May have atrophy of epidermal surface. Usually no granular layer except at follicular orifices • Elastosis and often chronic inflammation of dermis • Follicular apparatus and intraepidermal sweat duct are spared
Lentigo maligna melanoma	• Flat, tan to black with irregular hyperpigmentation, > 2 cm • 5 - 15% of (invasive) melanoma • Increasing prevalence, particularly among men age 65+ year • Slow growing lesion of sun exposed skin of elderly whites, often cheek • Partial regression is common	• Contiguous growth of atypical melanocytes in basal layer, individually and in nests (theques). • Cells may be spindled, pleomorphic and have cytoplasmic retraction • Dermis shows solar elastosis • Also epidermal atrophy, actinic damage and basilar keratinocyte hyperpigmentation
Lentigo simplex	• Macular hyperpigmentation • Often > 1 cm • "Ink spot lentigo" variant: small, darkly pigmented, stellate	• Elongation of rete ridges and increased pigmentation at tips of retes, pigmentation may be irregular • Solar elastosis, telangiectasia, variable chronic inflammatory infiltrate in dermis
Large cell acanthoma	• Light-tan to dark brown, well circumscribed macule or patch • Few millimeters to centimeters in diameter • Typically on sun exposed skin • Clinically resembles a seborrheic or actinic keratosis	• Atrophic or acanthotic epidermis containing larger than usual keratinocytes (~2x normal epidermal keratinocytes) • Usually well demarcated from adjacent epidermis • Basal layer may appear hyperpigmented, sometimes conspicuous rete ridges, orthokeratosis, may have hypergranulosis • Minimal nuclear pleomorphism, basal mitoses only

or liquid nitrogen, electrodesiccation, electrodesiccation and curettage, curettage alone, shave biopsy or excision using a scalpel, or a laser or dermabrasion surgery. Some of these techniques destroy the lesion without providing a specimen for histopathologic diagnosis. Application of 70% glycolic acid for 3-5 minutes prior to curetting is also effective. Ammonium lactate and alpha-hydroxy acids have been reported to reduce the height of seborrheic keratoses.[9] A concentrated solution (40%) of hydrogen peroxide was approved by the FDA in 2017 for in office application to patients with raised seborrheic keratoses.[10]

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