Case Study

Giant Seborrheic Keratosis- A Rare Entity

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Abstract

Background: Seborrheic Keratosis (SK) is most common benign epidermal tumor with distinctive "stuck on" appearance. It appears on the sun-exposed sites with equal sex distribution in middle- and old-aged individuals. Head, neck and trunk are the common sites. They often range in size from a few millimetres to a few centimetres. They are usually slow growing, however, clinical changes such as sudden increase in size, color change and ulceration may indicate malignant change.

Case Presentation: The purpose of this review is to report a case of an unusually large, seborrheic keratosis on the face, highlighting its clinical and histopathological features. The manifestations pointed towards malignancy and the patient underwent biopsy and complete excision with a favorable outcome.

Investigation: Even though they are clinically diagnosed, SK mimickers are well known, with melanoma being the most worrisome. Dermoscopy is used to help with diagnosis when uncertainty arises, as various benign and malignant conditions must be considered in the differential diagnosis due to their morphological diversity.

Management: Treatment options include surgical therapy, laser therapy, electrocautery, cryosurgery, shave excisions and laser-assisted removal.

Conclusion: Giant SK, an atypical SK is uncommon and can manifest in an unusual way, and histopathological analysis is warranted to look for concomitant malignancy.

Keywords: face; giant seborrheic keratosis; benign epidermal tumor, histopathology

Introduction

Seborrheic keratosis(SK) is the most common benign epidermal tumor that affects both sexes equally with predominance in individuals older than 50 years.[1] Although SK lesions can appear anywhere, they are most common on the face and upper body. Clinically the lesions appear as slightly elevated, well-defined brownish patches or plaques, usually on sun-exposed surfaces of the skin. As the lesion enlarges, it may become papular, verrucous, waxy and attain characteristic "stuck on" appearance.[2]

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Seborrheic keratosis can occur in many variations and include clinical variants, such as stucco keratosis and dermatosis papulosa nigra. Histopathologically, it is distinguished into six major subtypes: acanthotic, hyperkeratotic, adenoid, irritated, clonal, and melanoacanthoma with three features in common in all of the subtypes including (ortho-) hyperkeratosis, acanthosis and papillomatosis[1].

They are usually slow growing, however, clinical changes such as sudden increase in size, color change and ulceration may indicate malignant change accompanied by basal cell carcinoma(BCC), squamous cell carcinoma (SCC), keratoacanthoma, malignant melanoma, and hamartomas.[3]

Although large sized lesions of Seborrheic Keratosis are less documented in literature, Baer et al have reported the occurrence of giant lesion of Seborrheic keratosis in inguinal region with dimensions of 5.5cm x 3.5cm[4]. Large lesions of size ranging from 15cm x 10cm have also been reported in perigenital area and vulval area. Other sites include breast, flank, axilla[5,6,3,7] with size ranging from 5cm x 9cm. In our case, rapidly enlarging lesion of large seborrheic keratosis of size 6cm x 5cm was noted on right temporal region. Large seborrheic keratosis on face are rare. However, to the best of our knowledge single study by Luen KK et al[11] have reported lesion of 20cm x 15cm on temporal region in similar age group with a long history of three decades.

Materials and Methods

The patient was a 75-year-old male who came in surgery OPD with slowly progressive verrucous growth on face for last eight years. Rapid two fold enlargement was noted in last two months. The history of skin exposure to sunlight was elicited. The lesion began as a little, pigmented papule and grew larger over time to form a substantial mass, without any secretion or discharge at first.The lesion was painless, non-pruritic and non-hemorrhagic. There was no history of trauma. He had no history of smoking, alcohol consumption, skin burn, allergy or any history of prior medication. The patient never had any surgery or systemic or local treatment for the aforementioned condition. The patient revealed no family history of skin conditions or cancers. Physical examination revealed tan, black oval-shaped pedunculated mass which was 6cm x 5cm, firm, fixed, non-tender fungated growth with uneven surface and margins. On dermoscopy, the presence of comedo-like (CL) openings was the most common finding followed by fissures and ridges, and sharp demarcation of the lesion were consistent findings observed in our case. Hairpin (HP) blood vessels were not seen in our patient. We also noticed a parched paddy field-like appearance on dermoscopy in our patient. Subsequently, excision of the mass was done under local anaesthesia and the excised mass was sent in 10% formalin to the Department of Pathology, Muzaffarnagar Medical College for histopathological examination. A written informed consent from patient regarding procedure, histopathology and publication was subsequently taken.

Possible Molecular and Pathophysiological Mechanisms

The aetiology and pathophysiology of SK are still mostly unknown, despite its high prevalence. Age is the primary and most important risk factor for SK. Genetic susceptibility is undoubtedly another danger to SK.UV exposure and viral genesis have also been identified as risk factors for SK, in addition to age and genetic predisposition.[8]

- **Role of amyloid-associated protein (APP)**

Ageing and UV-exposed skin are shown to have higher levels of APP. Senescent keratinocytes have lower levels of presenilin 1 and 2, which prevents beta-amyloid from being released from APP, resulting in its accumulation and this has also been postulated in SK formation in mice.[9]

- **Infectious etiology**

Fluorescent in situ investigations (FISH) and polymerase chain reaction were used to identify HPV DNA in SK. However, compared to non-genital areas, genitalia have a significantly higher incidence of HPV DNA.

Only the superficial area of SK had HPV-DNA, and deeper biopsies showed a lower percentage, suggesting surface contamination rather than the real aetiology.[9]

- **Oncogenic variation**

Oncogenic mutations have been discovered in SK lesions despite the fact that SK is a benign tumour.

Fibroblast Growth Factor Receptor 3 (FGFR3) was the most often occurring somatic mutation. Nevertheless, cancer cannot be caused by this mutation alone.

In inflammatory dermatoses, fibroblast growth factor deficiency results in abnormalities of the skin, and the epidermal growth factor receptor (EGFR) has been connected to abnormal differentiation and proliferation of keratinocytes in SK. The AKT1, HRAS, EGFR, and KRAS oncogenes19 are genetically stable despite alterations in the PI3K-AKT and FGFR3-RAS-MAPK pathways. This stability may be explained by the absence of a p53 mutation in SK. [9]

Diagnostic Challenges

The present case illustrates the diagnostic challenges in distinguishing seborrheic keratosis from malignancy, with initial suspicion raised by both physiological and dermoscopic features overlapping with basal cell carcinoma (BCC) or melanoma. Physiologically, the lesion exhibited uneven, irregular margins and rapid growth in an elderly patient—traits that mimic the asymmetric borders and evolution seen in melanoma, or the infiltrative edges of BCC. Dermoscopically, atypical findings included asymmetry in color and structure, with patchy brown pigmentation and irregular borders, further overlapping with melanoma's asymmetry, border irregularity, and color variegation, or BCC's arborizing vessels and leaf-like structures (though the latter were absent here).To rule out these mimics in a logical sequence:

1. **Dermoscopic exclusion of BCC**: No arborizing telangiectasias, shiny white structures, or leaf-like areas were observed; instead, classic seborrheic keratosis features predominated—multiple milia-like cysts, comedo-like openings, and a sharply demarcated, "stuck-on" appearance with cerebriform surface.
2. **Dermoscopic exclusion of melanoma**: Despite initial asymmetry and uneven margins, the lesion lacked a multicomponent pattern, atypical network, blue-white veil, or peripheral streaks/radial streaming; the uniform keratin-filled crypts and fat fingers further supported benignity.
3. **Histopathological confirmation**: Excisional biopsy revealed acanthosis, papillomatosis, and horn cysts with keratin-filled invaginations, without atypia, mitotic activity, or invasion—definitively excluding BCC (no basaloid nests or peripheral palisading) and melanoma (no melanocytic proliferation or nesting).

Since BCC or squamous cell carcinoma can coexist with seborrheic keratosis, any lesion suggestive of the latter but with atypical features warrants dermoscopic triage followed by histopathological verification.[10] Other synonyms for seborrheic keratosis include basal cell acanthoma, verruca senilis, senile wart, verruca seborrhoica, seborrhoeic wart, benign basal cell papilloma, and benign acanthokeratoma.[7] Despite classic "stuck-on" appearance, all atypical verrucous lesions demand excision and histopathology.[11]

Differential Diagnosis and Diagnosis

Clinical differential diagnoses include pigmented basal cell carcinoma, pigmented Bowen's disease, condyloma, verrucous melanoma, extramammary Paget's disease, common warts, and acanthosis nigricans.[6] On histo-pathological examination, the biopsy revealed an epidermal hyperkeratosis, extensive acanthosis, and papillomatosis of the epidermis, upward growth of basaloid cells with small horn cyst formation along with mild chronic lymphocytic infiltrate in dermis showing seborrheic keratosis without cellular atypia.

A clinical diagnosis of giant seborrheic keratosis was made since the biopsy report showed no indication of concomitant malignancy, despite the clinical signs being suggestive of malignancy.

Therapeutic Intervention

The therapeutic techniques available for the treatment of SKs is primarily procedural. As described in the case report, surgical excision was performed without distorting the surrounding structure. Other minor surgical modalities includefollowing low-intensity procedures: curettage, electrodessication, cryosurgery, chemical and laser destruction. Unfortunately, these methods frequently result in pigmentation changes, scarring, and recurrence.[12] Lesion removal with an erbium-based ablative laser treatment: Another option for treating SK is YAG (Er:YAG) or CO_2 laser, which has been shown to completely heal 100% of lesions when compared to

cryotherapy.[7] Compared to the cryotherapy group, hyperpigmentation was considerably less severe while erythema increased more in the Er:YAG laser-treated group.[6]

Two topical treatment formulations have recently been assessed: an aqueous nitric–zinc solution and a product containing 40% hydrogen peroxide (HP40). Clinical research suggests that HP40 is considered to be less toxic to melanocytes, indicating a viable substitute for surgery, especially for face lesions.[6]

Discussion

This study presented a rare case of giant Seborrheic keratosis located on sun-exposed area with rapid enlargement.[3] Seborrhoeic keratosis is a benign skin tumour that primarily affects individuals over the age of 50.[7]Both sexes are equally impacted. Although it can happen anywhere on the body, but is more common on the head, neck, chest, and back. Mucous membranes, palms, and soles are typically unaffected. SK is typically asymptomatic. However, discomfort and itching may be caused by trauma or inflammation. Lesions usually appear as stuck on verrucous plaques of varying thickness and are either black, gray-brown, or yellowish in colour.[11]They often range in size from a few millimetres to a few centimetres.(7)In our case, SK reached 6cm in greatest dimension necessitating histopathological analysis. [13]Clinical variants of SK include stucco keratosis, dermatosis papulosis nigra, lichen planus-like SK, inflammatory SK, large cell acanthoma, flat SK and Leser Trelat syndrome.[11] On routine hematoxylin-eosin (H&E)staining of the slides prepared from paraffin wax sections, two keratinocytic components—basaloid cells and monomorphous squamous epithelial cells were identified. Additional features include marked epidermal acanthosis, horn cysts/pseudocysts, hyperkeratosis, and papillomatosis are observed.[13] Acanthotic, hyperkeratotic, adenoid, irritated, clonal, and melanoacanthoma are its six main histopathological variations.[1] On dermoscopy, it was seen that comedo-like openings, fissures and ridges, and sharp demarcation of the lesion were consistent findings in common seborrheic keratosis and less frequently observed findings were moth-eaten borders, milia-like cysts and network-like structures. The exact etiology and pathogenesis of seborrheic keratoses is currently unclear. Infection, UV exposure, and genetics have all been suggested as potential contributing factors.[6] The pathophysiology of SK appears to be significantly influenced by mutations, specifically in the AKT signalling pathway and in the epidermis' fibroblast growth factor receptor 3 (FGFR3).[14] It is emphasized that seborrheic keratosis at sun-exposed areas is at a higher risk of concurrent malignancy, and histological diagnosis is important despite its benign nature, even though the exact cause of SK is still unknown. The lesion in our case originated in the right temple area, but aside from its remarkable size, it did not exhibit any typical malignant alterations.[11]

Surgical excision of the lesion was performed and is a preferred method in order to preserve aesthetic and functional outcomes.[11] The patient was discharged on antibiotic course, faropenem 300 mg, twice a day, for one week for preventing the infection and better wound healing. The defected area was closed with a split skin graft from the surrounding skin laxity. At 2 months of follow-up, the excision area was examined and no primary complication like infection was observed. There was no evidence of a recurrence and the surgical site had healed adequately as observed in routine follow up visit of patient. Cryotherapy, curettage or laser treatment can also be considered as other treatment modalities as discussed.[3]

Below is the table summarizing previously reported cases of giant seborrheic keratosis particularly highlighting their size, location/site of lesion and whether malignancy was present or not. [Table 1]

Author	Age/Gender	Size(in cm)	Location of lesion	Whether malignancy was detected or not
*Koh KL et al[11]	75/M	20 x15 cm	Face	Not present
Livaoglu et al[15]	42/M	20 x25 cm	Pubic	Not present

Cont.....

Nath et al[16]	50/F	15 x10 cm	Vulva	Not present
Salah et al[17]	66/M	15 x10 cm	Perianal	Not present
Okazaki and Ueda[18]	63/M	9 x7.5 x3 cm	Scalp	Not present
Part et al[19]	33/M	8 x10 cm	Penis	No malignany transformation noted due to presence of HPV 6 low-risk virus.
Tsuji and Morita[20]	44/M	8 x 6 cm	Frontal scalp	Not present
Pepper[21]	71/M	8 x 6 cm	Hip	Not present
Alapatt et al[6]	70/F	7 x 6 cm	Breast	Not present

*Large seborrheic keratosis on the face are rare. However, to the best of our knowledge single study by Koh KL et al have reported lesion of 20 x 15cm on temporal region in similar age group with a long history of three decades.

Conclusion

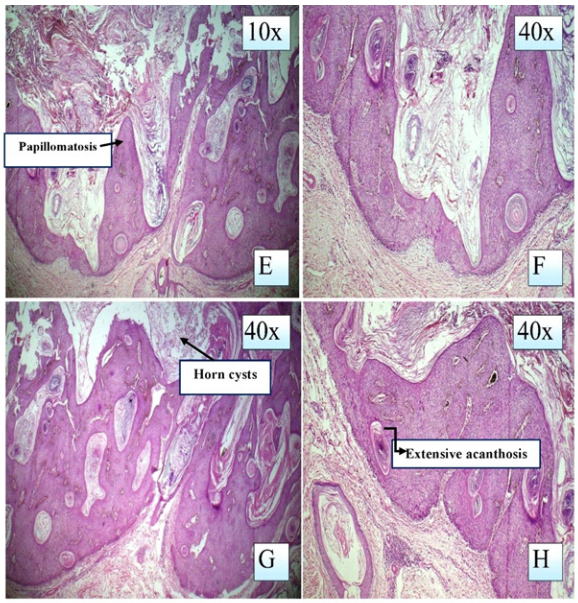
Seborrheic keratosis (SK) is a benign skin tumor. Although, accurate diagnosis is typically simple due to clearly identifiable clinical characteristics, it can be confused with other potentially malignant skin tumors resulting in diagnostic difficulties. Dermoscopy and histopathology may facilitate accurate diagnosis. To the best ofour knowledge, rarely similar type of giant lesion have been reported on face before. In this context, histopathological examination is necessary to confirm the diagnosis, exclude malignancy, and direct the proper course of treatment. Surgical resection, cryotherapy, curettage or laser treatment can also be considered as other treatment options in order to preserve aesthetic and functional outcomes.

Summary/ Take home message

Giant lesions of Seborrheic keratosis are quiet uncommon. It is the most common benign epidermal tumor of skin that affects equal number of men and women. The exact cause and pathogenesis is still not yet clear. Genetic predisposition, advancing age and ultraviolet light exposure are considered to be contributing risk factors in development of SKs. These lesions basically begin as circumscribed plaques or patches, which later gets more papular giving verrucous "stuck on" appearance. Although diagnosis is made clinically but both premalignant and malignant entities should be taken into consideration to exclude the mimickers of SKs.

Dermoscopy should be carried out to differentiate it from melanocytic neoplasms. But in some cases, dermatoscopic findings lacks some features making it difficult to reach a specific diagnosis. When diagnosis is not clear, lesion should be surgically removed and thorough histopathological examination of entire specimen should be carried out to rule concomitant malignancy and to confirm diagnosis which will guide appropriate management. There are several conservative treatment modalities available besides curettage and shave excision are electrodessication, cryosurgery, chemical and laser destruction, YAG (Er:YAG) or CO_2 laser. In recent studies, few clinical trials for topical therapy underwent for managing facial lesions which seems to be less toxic and with no or minimal adverse effects. These are HP40 (40% Hydrogen peroxide) and an aqueous nitric-zinc complex solution. Based on clinical trials, it is reported that it provides more promising results than surgery, particularly for facial lesions. However, histopathological examination is necessary to confirm the diagnosis, to rule out malignancy, and direct the proper course of treatment. The gold standard for treatment is still surgical excision.

- Fig A-Clinical picture.
- Fig B-Gross appearance of the lesion.
- Fig C-Intraoperative image taken showing the tumour arising from right temple region.
- Fid D-Incision made along the base of the lesion leaving a defect size of 6cm x 5cm.



- Fig E,F,G & H- Hematoxylin and Eosin-stained histopathology sections showing marked epidermal hyperkeratosis, extensive acanthosis, horn cysts, and papillomatosis.

Abbreviations

SK: Seborrheic keratosis

BCC: Basal cell carcinoma

SCC: Squamous cell carcinoma

OPD: Outpatient department

CL: Comedo-like openings

HP: Hairpin blood vessels

UV exposure: Ultraviolet radiation exposure

APP: Amyloid Precursor Protein

FISH: Fluorescent in situ hybridization

HPV DNA: Human papillomavirus DNA

FGFR3: Fibroblast Growth Factor Receptor 3

EGFR: Epidermal growth factor receptor

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