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Complementary Article

The Seboblastic Propagation: Sebaceous Hyperplasia

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Preface

Sebaceous hyperplasia is a frequently encountered, benign proliferation of enlarged sebaceous glands occurring in middle aged and elderly population. Although sebaceous hyperplasia can be discerned in middle aged or younger individuals, a nomenclature of "senile sebaceous hyperplasia" can be applied to the condition on account of disease emergence in the elderly. Nodules appear on face, nose or cheeks and induce a prominent cosmetic disability. The condition is devoid of spontaneous resolution.

Disease characteristics

Emergence of sebaceous hyperplasia is contingent to genetic predilection along with occurrence of seborrhoea and immune suppression. Particular predilection to develop sebaceous hyperplasia in specific instances remains obscure although diverse aetiologies and pathogenic factors such as natural aging, excessive exposure to ultra-violet radiation and genetic factors are incriminated. Pre-senile or premature diffuse familial sebaceous hyperplasia is a disorder exceptionally cogitated in adolescents with a familial incidence. Sporadic variant of sebaceous hyperplasia can be contemplated as a benign neoplasm rather than hyperplasia of sebaceous glands.

Molecular considerations exemplify the aforesaid hypothesis such as a pathogenic manifestation of the EGFR-RAS-MAPK pathway in sporadic instances of sebaceous gland hyperplasia [1,2].

Additionally, sebaceous hyperplasia can be induced by administration of cyclosporine in renal or organ transplant recipients. Aforesaid agent engenders multiple lesions of sebaceous hyperplasia responsive to therapy with oral isotretinoin.

Sebaceous hyperplasia also occurs in immune suppressed organ transplant recipients in an estimated 16% subjects [1,2].

Clinical elucidation

Aesthetic implications and clinical countenance of the disorder is distinctive. Majority of instances display disease evolution with the appearance of innumerable, recent lesions or occurrence of enlarged, umbilicated and senescent lesions with centric effluence of sebum. Soft to spongy, miniature, yellowish papules displaying a centric umbilication and orifice arise on facial regions, particularly forehead, cheeks and nose besides appearing on the genitalia, aerola and chest.

Sebaceous hyperplasia can emerge at diverse sites and demonstrate a morphological variability with configurations such as diffuse, unique large form, linear or zosteriform variant or lesions disseminated along lines of Blaschko's [2,3].

Diagnosis of sebaceous hyperplasia is possible to attain on clinical grounds.

Histological elucidation

On gross examination yellow coloured, umbilicated papules are discerned at various sites. Microscopic evaluation elucidates regular sebaceous glands with an expansive lobular architecture, glands which are primarily devoid of inspissated peripheral germinative layer of the seboblasts.

Although examination of a cutaneous specimen is not mandatory for arriving at a cogent diagnosis, a comprehensive histology is beneficial. It exemplifies a lobular pattern of well differentiated, mature, sebaceous gland components. Adherence of a minimum of four sebaceous lobules to the infundibulum of pilosebaceous unit is mandatory for designating a lesion as sebaceous hyperplasia.

Additionally, sebocytes demonstrating lipid accumulation are distinctive of sebaceous hyperplasia. Tissue specimens are often obtained to exclude the presence of basal cell carcinoma [3,4].

Differential diagnosis

Sebaceous hyperplasia necessitates a segregation from adjunctive disorders with sebaceous differentiation such as sebaceous adenoma, nevus sebaceous of Jadasshon, sebaceous epithelioma, steatocystoma simplex or steatocystoma multiplex [1,2].

Therapeutic options

Sebaceous hyperplasia is commonly treated with surgical methodologies such as electro-cauterization, cryotherapy, curettage, surgical excision and chemical ablation. Application of aforesaid techniques depict significant adverse effects such as pain, scarring, dys-pigmentation, delayed recovery and amplified recurrence rate. Innumerable lesions and cutaneous cicatrix necessitates the circumvention of surgical interventions [4,5].

Additionally, sebaceous hyperplasia can be therapeutically subjected to electrodessication, low intensity laser and adoption of acid application and photodynamic therapy. Therapeutic outcomes are variable with incurrence of adverse side such as scars and residual hyperpigmentation. Employment of aforesaid treatment options such as lasers or photodynamic therapy is expensive and difficult to obtain.

Topical application of 70% trichloroacetic acid (TCA) for a few months continuously at the site of sebaceous hyperplasia lesions is a cogent therapeutic tool which is devoid of adverse reactions.

Trichloroacetic acid is an cogent, self-neutralizing, deep-seated chemical peel which induces cutaneous exfoliation and is devoid of systemic absorption and functions at mid- reticular dermis. It induces coagulation of dermal and epidermal cells besides necrosis of collagen in the upper reticular dermis. Necrotic cutaneous tissue is displaced by regular skin on account of re- epithelialization originating from circumscribing islets of keratinocytes and cutaneous appendages. Fresh crop of papules are particularly responsive to aforementioned treatment. Subsequent monitoring demonstrates an absence of lesion reoccurrence [5,6].

Topical administration of an emollient cream with enhanced ultra violet protection index and prevention of sun exposure is recommended in durations betwixt topical applications of trichloroacetic acid for optimal alleviation.

Topical application of 70% trichloroacetic acid is a safe, convenient, inexpensive and efficacious method with appropriate patient compliance, good tolerability and outcomes and can be adapted in treating sebaceous hyperplasia with improbable therapeutic alternatives.

Diverse clinical and cosmetic indications are benefitted with the application of trichloroacetic acid such as striae rubra, plantar callus and warts, scars of cutaneous leishmaniasis chemo-cauterization of miniature trachea-cutaneous fistula, cervical intraepithelial neoplasia (CIN), acne, juvenile pyogenic granuloma and sebaceous hyperplasia [6,7].

Oral isotretinoin is beneficial for alleviating singular instances of sebaceous hyperplasia. Systemic administration of isotretinoin initiates a morphological reduction of magnitude and functional inhibition of sebaceous glands along with decimation of proliferating basal sebocytes. Suppression of sebum production and prohibited differentiation of in vivo sebocytes ensues.

Sebo-suppressive outcomes of isotretinoin includes induction of apoptosis or concurrence of isotretinoin in androgenic metabolism. Molecular incursions of retinoid anti sebotropic manifestations remain obscure or are probably contingent to nuclear receptor adherence and malfunction, isotretinoin is decidedly anti-sebotropic.

Declining dose of isotretinoin or limited duration of administration can demonstrate recurrence in lesions of sebaceous hyperplasia and emergence of minimal adverse affects [7,8].

Duration of isotretinoin administration is decisive factor in treating sebaceous hyperplasia as the therapy can modify the magnitude of sebaceous gland. A significant and preponderant decrease or comprehensive alleviation of quantifiable lesions of sebaceous hyperplasia ensues as a quick response to systemic administration of isotretinoin.

Evaluation of therapeutic outcome is required at commencement of treatment, an interval of two months and termination of therapy and demonstrates a recurrence of sebaceous hyperplasia with an average emergence of four lesions although the median count is reduced by half. Cessation of therapy can induce up to three lesions of sebaceous hyperplasia. Despite reoccurrence of sebaceous hyperplasia, enumerated lesions are fewer at termination of therapy rather than at initiation [1,3].

Systemic administration of isotretinoin is accompanied by adverse effects such as dryness of skin or mucosa and occasionally elevated triglyceride levels.

Isotretinoin administration also enhances cutaneous cosmetic appearance with declining blemishes and softening of fine wrinkles. Isotretinoin is forbidden in pregnancy on account of teratogenic consequences.

Continuance of isotretinoin therapy for around two months demonstrates a recurrence rate at an estimated 19%. Systemic therapy with isotretinoin depicts an outright (100%) efficacy of lesion reduction although standardization of dose and duration is necessitated. Sebaceous hyperplasia can display decimated magnitude and quantity of lesions sooner than a two month period of treatment. Standardization of isotretinoin dose and period of therapy is imperative as it is cost-effective and depicts minimal and dose-dependent adverse effects [7,8].

Sebaceous hyperplasia emerging in Fitzpatrick skin type II-III are frequently managed by pulsed dye laser (PDL). Essentially a safe and efficacious technique, pulsed dye laser is associated with PDL associated purpura. Utilization of visible light wavelength of 585 - 595 nm is devoid of significant absorption by sebaceous gland lipids. Vasculature abounding with haemoglobin, which circumscribes the sebaceous gland, is the specific chromophore for applying intense fluence of pulsed dye laser and appropriately absorbs laser energy. Thereby, coagulation necrosis of the vasculature is initiated with consequent decimation of sebaceous gland lobules.

A combined treatment protocol of high and moderate intensity PDL can be adopted for simultaneously managing sebaceous hyperplasia and PDL induced purpura. Majority (85%) of instances of PDL induced purpura can be eradicated by a singular, moderate - fluence PDL therapy [8,9].

High fluence PDL is initially administered with a sequential moderate-fluence PDL after twenty four hours. Aforesaid concurrence of pulsed dye laser therapy demonstrates a probable emergence of tissue necrosis.

Untreated PDL induced purpura spontaneously resolves in one or two weeks. Adverse effects of application of laser therapy are erythema, oedema and purpura. Pulsed dye laser therapy is not recommended in Fitzpatrick skin type IV-VI on account of a definitive hypopigmentation [9,10].

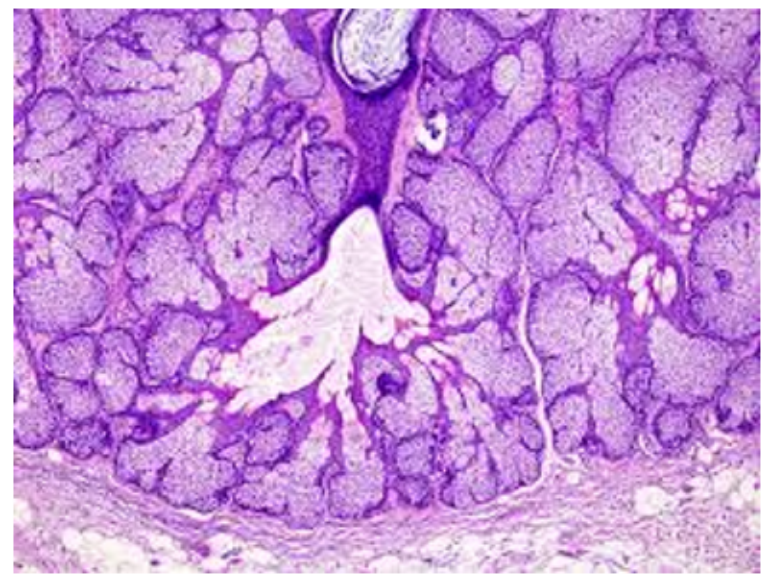

Figure 1: *Sebaceous hyperplasia with lobular arrangement and thin, basal germinative layer [11].*

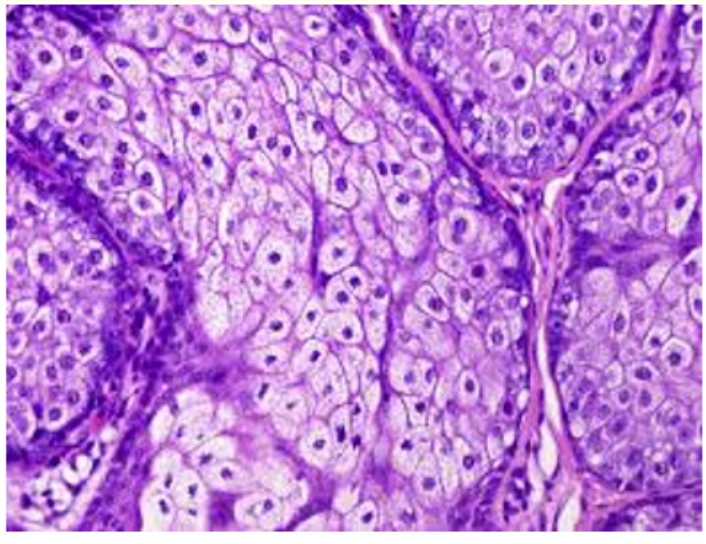

Figure 2: *Sebaceous hyperplasia with sebocytes impacted with lipids and attenuated basal layer [11].*

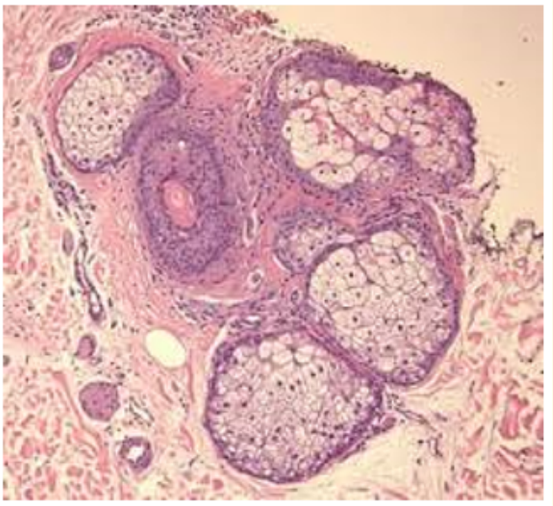

Figure 3: *Sebaceous hyperplasia with aggregated sebocytes and adherence to hair follicular infundibulum [12].*

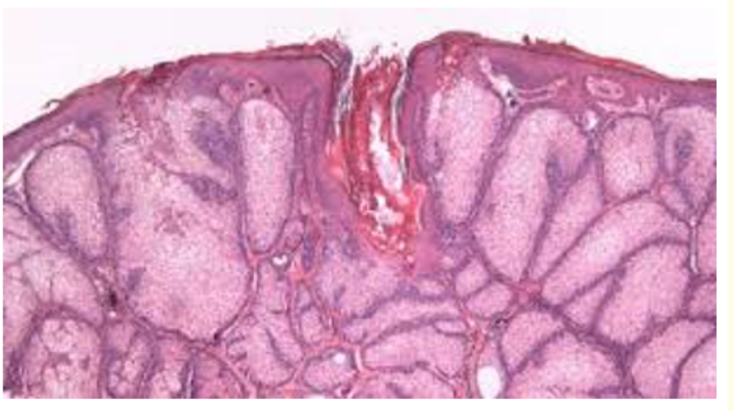

Figure 4: *Sebaceous hyperplasia with lipid included sebocytes, lobular configuration and superimposed stratified epithelium [12].*

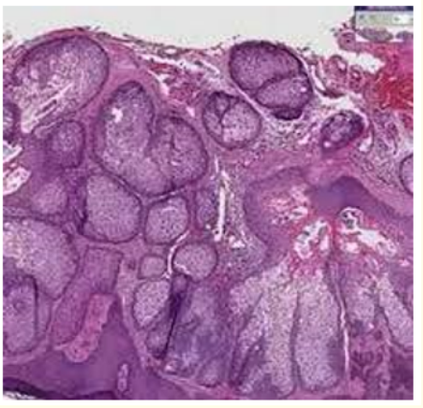

Figure 5: *Sebaceous hyperplasia with lobular architecture, nests of lipid rich sebocytes and attenuated germinative layer [13].*

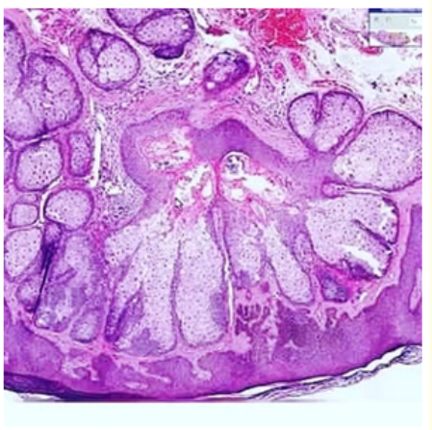

Figure 6: *Sebaceous hyperplasia with lobular articulations, mature sebocytes and attenuated superimposed epithelium [13].*

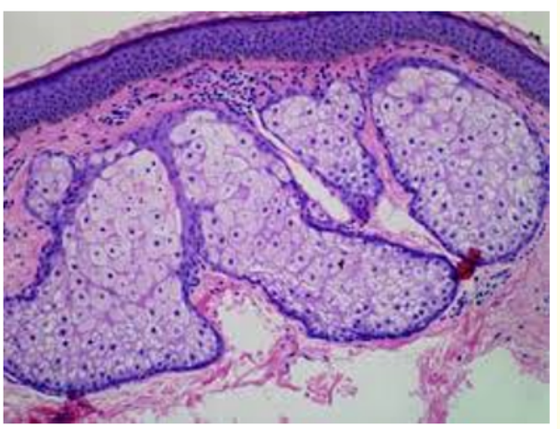

Figure 7: *Sebaceous hyperplasia with lipid rich adipocytes and lobular arrangement with acanthotic stratified epidermal layer [14].*

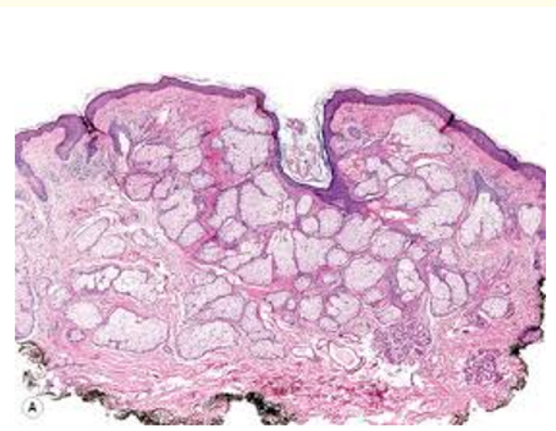

Figure 8: *Sebaceous hyperplasia with multiple lobules of sebocytes and distinctive arrangements of adipocytes [15].*

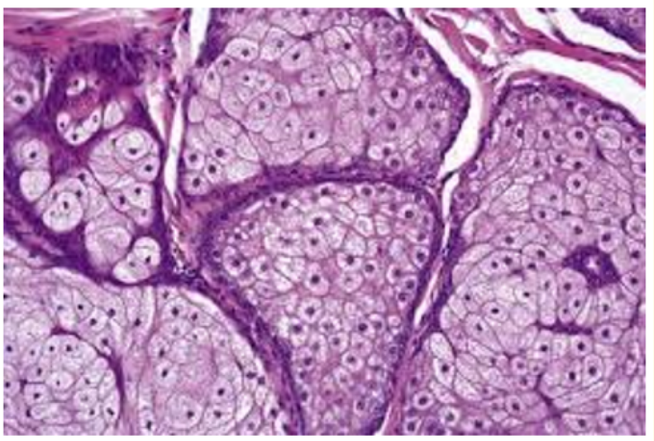

Figure 9: *Sebaceous hyperplasia with vacuolated, lipid rich adipocytes and attenuated, basal germinative layer [16].*

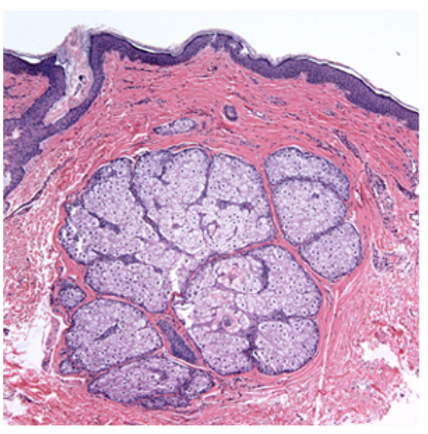

Figure 10: *Sebaceous hyperplasia with lobular architecture, circumscribing fibro-connective tissue and superimposed epidermal layer [17].*

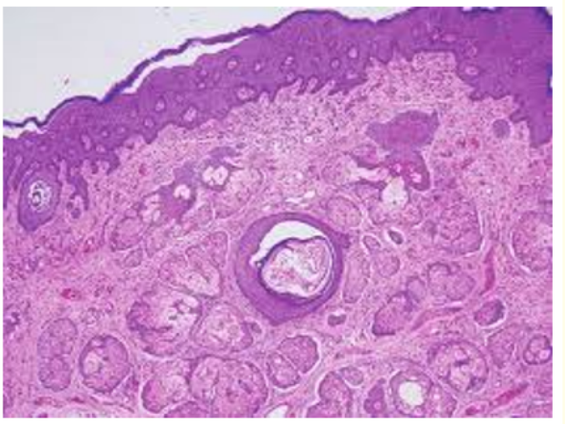

Figure 11: *Sebaceous hyperplasia with sebocytes accumulated in lobular array and acanthotic, hyperkeratotic superimposed squamous epithelium [18].*

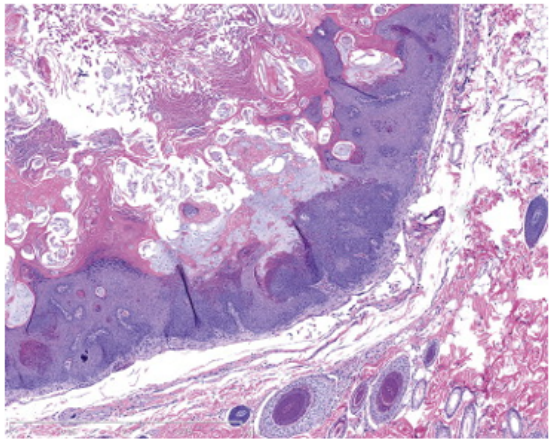

Figure 12: *Sebaceous hyperplasia with infundibular origin of adipocytes, lobules of sebocytes and stratified squamous epithelium [19].*

Conclusion

Sebaceous hyperplasia is a frequent benign proliferation of enlarged sebaceous glands occurring in middle aged and elderly population. Although obscure, diverse aetiologies and pathogenic factors such as natural aging, excessive exposure to ultra-violet radiation and genetic factors are incriminated in the genesis of sebaceous hyperplasia along with pathogenic manifestation of EGFR-RAS-MAPK pathway in sporadic instances. On clinical examination, soft to spongy, miniature, yellowish papules with centrically umbilicated orifice arise on face, forehead, cheeks, nose, genitalia, aerola and chest. Normal sebaceous glands with an expansive lobular architecture and sebaceous glands which are primarily devoid of inspissated peripheral germinative layer of seboblasts are cogitated on microscopy. Disorders with sebaceous differentiation such as sebaceous adenoma, nevus sebaceous of Jadasshon, sebaceous epithelioma, steatocystoma simplex or steatocystoma multiplex are conditions requiring a segregation from sebaceous hyperplasia. Cogent therapeutic options include surgical methodologies such as electro-cauterization, cryotherapy, curettage, surgical excision, chemical ablation, electrodessication, topical application of 70% trichloroacetic acid and photodynamic therapy. Oral or systemic isotretinoin alleviates singular lesions sebaceous hyperplasia as it initiates a morphological reduction of magnitude and functional inhibition of sebaceous glands along with decimation of proliferating basal sebocytes. Sebaceous hyperplasia of Fitzpatrick skin type II-III are managed by pulsed dye laser (PDL). Combination treatment protocol of high and moderate intensity PDL is adopted for simultaneously managing sebaceous hyperplasia and PDL induced purpura.

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