ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 04 | April - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

SEBACEOUS HYPERPLASIA: A MISSED CLINICAL ENTITY.

Dermatology

Dr. Ojas Dipak Kulkarni Junior Resident, Department of Dermatology Venereology and Leprosy, Sree Balaji Medical College, Chennai.

Dr. Sivaramakrishnan S.* Senior Resident, Department of Dermatology Venereology and Leprosy, Sree Balaji Medical College, Chennai. *Corresponding Author

Dr. Vignesh N.R. Assistant Professor, Department of Dermatology Venereology and Leprosy, Sree Balaji Medical College, Chennai.

Dr. Sukanya G. Associate Professor, Department of Dermatology Venereology and Leprosy, Sree Balaji Medical College, Chennai.

ABSTRACT

A 53-year-old male presented with multiple skin-coloured raised lesions on the forehead and cheeks for 1 year. Lesions were asymptomatic and the patient did not give any history suggestive of the likely pathology. All the papules had central umbilication and a few had a yellowish hue. The patient had normal haematological parameters. He was later subjected to a histopathological examination which revealed flattened epidermis, a sub-epidermal free zone and marked proliferation of sebaceous lobules in entire dermis. Histopathology aided in the diagnosis of Sebaceous Hyperplasia. Patient was counselled and the lesions were removed using radiofrequency ablation.

KEYWORDS

INTRODUCTION:

Sebaceous hyperplasia (SH) is a benign proliferative condition of the sebaceous glands with lesions present predominantly on the face and cheeks of patients. Elderly individuals are more commonly affected. Lesions have also been reported to occur on the vulva, penis and areola. [1,2,3] Neonatal and adolescent cases have also been reported. [4] This condition can be managed with ablative and cryotherapeutic procedures.

Case Report:

A 53-year-old male presented to the dermatology OPD with complaints of raised skin-coloured lesions on the face since past 1 year. He had no associated complaints and was concerned about the sudden appearance and evolution of the lesions. He had no history of similar lesions in the past. Family history was not significant. He had no lesions elsewhere on the body. No history of erythema, discharge, tenderness or warmth at affected sites. No oral involvement. Patient was a known case of rheumatoid arthritis and was on 25mg of methotrexate weekly and daily doses of systemic corticosteroids. He had been treated for acne vulgaris for the lesions over the face with no response.

On examination, the lesions appeared as skin-coloured papules of around 0.3-0.5 cm in size. The lesions were predominantly situated on the forehead and cheeks of the individual (Figure 1A and B). All papules showed central umbilication (Figure 1A-marked by white arrow). No evidence of pseudo-koebnerisation. There was no evidence of atrophy, scarring or superficial telangiectasia. Few papules had a yellowish hue on their surface. With these clinical features we kept few possibilities namely molluscum contagiosum, xanthomas, adult-onset acne, lupus miliaris disseminatus faciei, syringomas etc., We performed a needling for a few lesions on the forehead but were unable to see extrusion of any material.

We subjected the patient to a complete blood count, lipid profile and liver function test, which turned out to be within normal limits. We then performed a punch biopsy from a papule on the forehead and subjected the specimen for a histopathological examination. On a scanner view (Figure 2A) we were able to appreciate that the epidermis was intact but the rete ridges were flattened. The superficial dermis showed a subepidermal free zone below which there were marked proliferations of sebaceous glands in the dermis obscuring the dermal architecture. The sebaceous glands were seen throughout the extent of dermis. On further examination under 100x magnification (Figure 2B), the sebaceous gland lobules were well delineated. Fibrous septa were seen separating individual lobules. With these histopathological features we were able to make a diagnosis of Sebaceous Hyperplasia. Figure 3 shows the presence of multiple sebaceous lobules showing central mature sebocytes and peripheral immature germinative cells. The patient was counselled about the benign nature of the condition. A radiofrequency ablation was performed for the lesions and the patient responded well with complete clearance.

DISCUSSION:

Sebaceous hyperplasia (SH) is a fairly common benign proliferation of sebaceous glands seen affecting around 1% of population. [5] Though the elderly is more commonly affected, lesions of SH can be seen in all ages. Patients on cyclosporine have been seen to be more predisposed to the development of these lesions. Chronic immunosuppressive states can also induce the formation of these benign sebaceous enlargements. A genetic background is suspected as patients of Muir-Torre syndrome present with sebaceous hyperplasia along with epidermoid cysts.[6]

Sebaceous gland is highly responsive to androgens. It has been postulated that as patient ages, the androgen levels have a steady decline which in turn causes decreased sebum production. This stimulates a negative feedback loop which induces a sebaceous gland hyperproliferation resulting in SH. Presenile diffuse familial sebaceous hyperplasia (PDFSH) is a distinct entity which has an earlier onset and a possible genetic etiology.[7]

Lesions of SH present as discrete papules. Central umbilication and yellowish hue to the papules is common. The central umbilication denotes the opening of the central sebaceous duct onto the skin surface. Superficial telangiectasias can also be seen in a few instances. A linear variant of SH is seen on the neck and around clavicles with 'beaded lines' representing hyperplastic glands.[8] A dermoscopy often shows presence of crown blood vessels on the surface of SHs.[9]

A histopathological examination of SH reveals a large sebaceous gland with multiple lobules coalescing towards a central sebaceous duct which is seen opening into the epidermis. The central glandular portion presents with mature sebocytes whereas the peripheral cells may still be germinative with less lipid droplets and thereby poor vacuolisation.[10] Lesions show positive staining for androgen receptors, though the levels of circulating androgens are usually normal. The upregulation of EGF-EGFR and Sonic hedgehog pathway seems to be a trigger to the development of SH.[11]

Lesions of sebaceous hyperplasia may mimic numerous dermatoses like acne, syringomas, angiofibromas, mollucum contagiosum,

xanthomas etc., A possibility of SH should always be borne in mind especially in the elderly.

Patient has to be counselled and reassured about the benign nature of this condition. Local ablative and destructive procedures like radiofrequency ablation, CO_2 laser ablation, cryotherapy and surgical excision can be considered for cosmetic purposes. Topically bichloracetic acid and trichloracetic acid can be applied for chemical cauterisation.

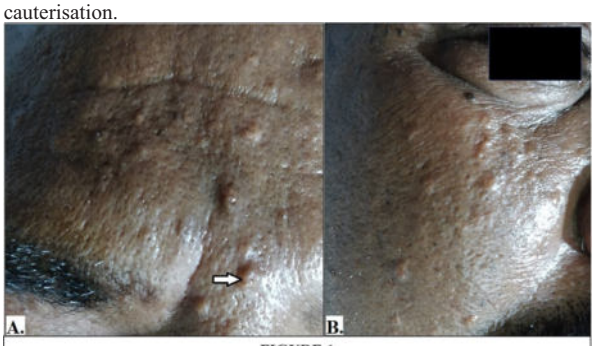

FIGURE 1

Figure 1-A: Presence of multiple skin coloured papules seen over the forehead. Central umbillication over a papule seen (marked with white arrow). ; **1-B:** Presence of numerous skin coloured papules seen over the cheek.

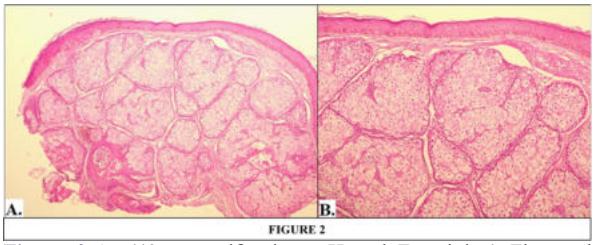

FIGURE 2

Figure 2-A: (40x magnification ; H and E staining) Flattened epidermis with presence of proliferation of sebacous lobules seen in the entire dermis. A sub-epidermal free zone is seen seperating the epidermis and sebaceous proliferations. **2-B:** (100x magnification ; H and E staining) Multiple sebaceous gland lobules seen in the dermis seperated by fibrous septa.

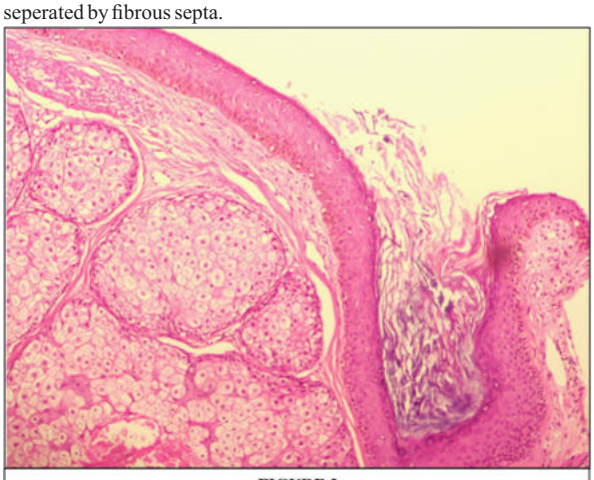

FIGURE 3

Figure 3 (100x magnification; H and E staining) Individual sebaceous lobules show central mature sebocytes and peripheral immature geminative cells. Follicular plugging is also seen.

REFERENCES:

1) Ortiz-Rey JA, Martin-Jimenez A, Alvarez C, et al. Sebaceous gland hyperplasia of the vulva. Obstet Gynecol 2002;99(5, Pt 2):919–921.
2) Carson HJ, Massa M, Reddy V. Sebaceous gland hyperplasia of the penis. J Urol 1996;156(4):1441.
3) Belinchon I, Aguilar A, Tardio J, et al. Areolar sebaceous hyperplasia: a case report. Cutis 1996;58(1):63–64.
4) Kanada KN, Merin MR, Munden A, Friedlander SF. A prospective study of cutaneous findings in newborns in the United States: correlation with race, ethnicity, and gestational status using updated classification and nomenclature. J Pediatr. 2012; 161:240-5.
5) Farci F, Rapini RP. Sebaceous Hyperplasia. StatPearls [Internet]. 2020;10.
6) Lazar AJ, Lyle S, Calonje E. Sebaceous neoplasia and Torre–Muir syndrome. Curr. Diagn. Pathol. 2007; 13:301-19.
7) Liu YC, Cheng YP, Liu CI, Yang CY, Yang CY. Presenile diffuse familial sebaceous hyperplasia successfully treated with low-dose isotretinoin: A report of two cases and review of the published work. J Dermatol. 2016; 43:1205-8.
8) Finan MC, Apgar JT. Juxta-clavicular beaded lines: a subepidermal proliferation of sebaceous gland elements. J. Cutan. Pathol. 1991; 18:464-8.
9) Zaballos P, Ara M, Puig S, Malvehy J. Dermoscopy of sebaceous hyperplasia. Arch Dermatol. 2005 1;141:808
10) Luderschmidt C, Plewig G. Circumscribed sebaceous gland hyperplasia: autoradiographic and histoplanimetric studies. J Invest Dermatol 1978;70(4):207–209.
11) Zouboulis CC. Epidermal growth factor receptor and the sebaceous gland. Exp. Dermatol. 2013; 22:695-6.