NODULAR COLLOID DEGENERATION OVER HERPES ZOSTER SCARS

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A rare case of nodular colloid degeneration is reported which presented clinically as plaques studded with soft yellow papules simulating adult colloid milia superimposed only on herpes zoster scars of right side of the face.

Key Words : Colloid material, Amyloid, Herpes zoster

Introduction

Out of three types of colloid milium degeneration, nodular colloid degeneration (NCD) is very rare and it may not be related to sun exposure as lesions can be limited to trunk, chin or scalp. NCD can be seen as single large nodule or as multiple soft to rubbery, purple to yellow plaques or nodules with smooth or nodular surface. Telangiectasia and purpura can be seen over early papules or plaques on the face.[1,2] In NCD, colloid is seen as 3-4 nm, short wavy irregularly arranged filaments embedded in amorphous material simulating colloid of adult colloid millium (ACM).[3] In NCD and ACM colloid is derived from elastic fiberes.

Case Report

A 40-year-old female suffered from herpes zoster ophthalmicus of right side of face 4 months back . Three months back she reported as a case of post -herpetic neuralgia associated with post-herpetic scars showing superimposed plaques studded with yellowish, soft, 2-5 mm, globular papules, some having punctum and others showing dilated hair follicular openings closely simulating grouped lesions of adult colloid milium. Few similiar discrete papules were also present. In addition bilateral asymmetrical, skin coloured, firm 1-2 mm papules with hair filled central pits of nodular elastoidosis (NE) were seen. Colloid milium was unilateral and in close relation to herpes zoster scars and plaques of NE papules were also more in number near the colloid milium plaques. General physical, systemic examinations and routine investigations were normal. Histopathologically, flattening of epidermis with underlying big homogeneous pale pink area filling 2/3rd of dermis with dilated blood vessels, clefts, normal hair and mononuclear cells admixed with plasma cells infiltration were seen. Similiar infiltrate was seen around skin adenexae and dilated dermal vesels. At one side 3 dilated hair follicles with follicular keratosis, horn cyst formation, perifollicular mononuclear infiltrate and near by one cyst like space lined by flattened epidermis were seen. Histopathology was like NCD and associated change of NE on one side.

PHN was treated with combilaser therapy and interestingly colloid milium plaques showed improvement along with disappearance of pain. After 10 exposures, 50% relief in pain and 50% decrease in size of colloid milium papules was observed. After 15 exposures, 75% relief in pain was associated with 80% clearing of colloid milium papules. After 20 exposures pain disappeared and papules of colloid milium also disappeared. Even papules of NE also showed

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marked improvement and became bilaterally symmetrical.

Discussion

Plaques of NCD were seen superimposed over HZ scars. Histopathology from colloid milium plaque revealed that colloid material was deposited in upper 2/3rd of dermis rather than being limited to dermal papillae and therefore diagnosis was NCD although clinically individual soft yellowish papule resembled ACM.

As the NCD plaques were only over HZ scars, it was hypothesised that colloid degeneration resulted from degeneration of dermis caused by HZ virus infection when all NCD plaques disappeared alongwith PHN after 20 exposures of combilaser, it was also proved that colloid degeneration was closely related to degeneration of dermis associated with HZ.

References

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