Letters to the Editor

Apocrine chromhidrosis localized to the areola in an Indian female treated with topical capsaicin

Sir,

Apocrine chromhidrosis is characterized by secretion of colored apocrine sweat. The color results from lipofuscin pigment in apocrine secretions. The colors vary from blue, black, yellow to green.[1,2] A 28-year-old North Indian female presented with complaints of black staining of undergarments over the breasts for the last five to six years. Staining was more prominent during summer. There was no history of relevant drug intake during this period including oral contraceptives. Examination revealed a dark black secretion over the areola of breasts, which could be expressed out in multiple droplets on squeezing the surrounding area [Figure 1]. The surrounding skin was normal in appearance. No evidence of colored sweat was found on the face and axillary areas. A skin swab from the involved area was negative for bacterial growth. Urine examination for pregnancy was negative and serum prolactin levels were normal. On the basis of history and clinical examination, a diagnosis of apocrine chromhidrosis was made.

She was started on topical capsaicin (0.1%) gel locally once daily. The secretion decreased in amount after two weeks of application. At eight weeks of treatment, she reported 60% decrease in the secretion. Patient initially complained of an intense burning sensation after each application of capsaicin lasting for two to three hours. A mild erythema had also developed on the application site at one week. However the burning sensation reduced gradually in intensity and became tolerable. At eight weeks she continued to have burning sensation lasting for half an hour after each application. Erythema had also decreased. She is presently continuing the use of topical capsaicin.

Apocrine chromhidrosis is a localized disease predominantly affecting the skin bearing apocrine glands. Two varieties are described—axillar and facial. The pigment lipofuscin is produced inside the apocrine gland in contrast to pseudochromhidrosis in which surface bacteria, fungi and dyes cause color formation.[1] Involvement of the areola has been reported.[3] Colored sweating can be induced by physical activity and emotional factors and patients are usually able to express out the sweat droplets mechanically. Histopathology shows increased number of lipofuscin granules in the secretory cells of apocrine glands. The disease runs a chronic course improving slowly with age.

Capsaicin acts by causing stimulation of afferent unmyelinated nerve fibers causing release of substance P stored in synaptic vesicles. Substance P is found primarily in afferent sensory fibers, dorsal root ganglia and the dorsal horn of the spinal cord. Prolonged use leads to depletion of substance P in the unmyelinated, slow-conducting type C sensory fibers.[2,4,5] However, the exact mechanism of action in apocrine chromhidrosis is not known. Probably it inhibits the acetylcholine mediated release of sweat

Figure 1: Black secretion from the areola in droplets
Sir,

Acanthosis nigricans is a cutaneous disorder of hyperpigmentation and papillomatosis that may precede or coincide with a variety of benign, familial or malignant disorders. Acanthosis nigricans is characterized by hyperkeratosis and pigmentation and the affected skin is covered by papillomatous elevations, which gives it a velvety texture. The etiopathogenesis of acanthosis nigricans is diverse and includes endocrinal as well as metabolic abnormalities. It can also be drug-induced and related to malignancy. HIV / AIDS is associated with many alterations in endocrinal function. A wide range of cutaneous manifestations is reported in AIDS cases but there is a dearth of reports of acanthosis nigricans in AIDS cases.

A 40-year-old nonobese female presented with complaints of fever, diarrhea, weight loss and dysphagia off and on since one year and dark skin lesions over the nape of the neck of similar duration. She had a history of blood transfusion eight years back. She was tested HIV positive by ELISA on two occasions. Due to resource constraints, CD4 count and viral load estimation were not carried out. Presence of oroesophageal candidiasis was suggestive of AIDS. Examination of the nape of the neck showed hyperpigmentation and velvety thickening of the skin, which was suggestive of acanthosis nigricans [Figure 1]. No similar changes were observed in flexural areas. Personal and family history was not suggestive of diabetes mellitus, thyroid dysfunction and tuberculosis. There was no family history of skin changes suggestive of acanthosis nigricans. Patient was not on any medication like nicotinic acid, fusidic acid, oral contraceptives, or triazinate, which are known to cause acanthosis nigricans. Investigations to rule out endocrinopathy and malignancy were carried out. Blood sugar, lipid profile, thyroid profile and occult blood in stool were normal. X-ray chest and USG abdomen also did not show any abnormality. PAP smear for cervical cytology was normal. Histopathological examination revealed at the sympathetic nerve endings.

REFERENCES

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