REVISION CORNER
PIGMENT FORMATION

By
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Colour of the skin though important medically and cosmetically, it has created political, racial and social problems. However here we are concerned with medical aspect only. Colour of skin is largely due to the pigment melanin which is normally present in basal layer cells of epidermis. It performs a protective role against physical and chemical trauma and sensitization. It forms supra nuclear caps in basal cells. The amount of melenin determines the tanning potential of skin. It is actually a melenoprotein and it is firmly attached to protein. Melanin is manufactured by special cells called melanocytes which are found as syncyial sheet in between the cells of basal layer. Melanocytes are dopa positive. Melanin is found in skin, hair matrix and choroid of eyes. Its quantity and distribution depend on various factors. viz.

(1) Basic precursors and enzyeme tyrosinase (2) Hormonal (3) sulfhydryal and other inhibitors (4) Inflammations and irritation and (5) racial.

(1) Melanin is derived by a complex biochemic reaction from a precursor essential amino acid called tyrosine through the catalytic action of the enzyme tyrosinase which is a copper containing protein. It was Bloch who first observed this reaction which he called dopa reaction as the tyrosine is first oxidized to dopa or dihydroxy phyenyl alanine which was further oxidized in the presence of tyrosinase to melanin after going through several intermidiate products. The degree of activity of tyrosinase determines the degree of pigmentation and vice versa. Same is true for Melanocyte stimulating hormone. The increase of melanin in epidermis causes brown colour while that in nulanphagel in papilae and dermis causes greysh as seen in past inflammatory melanosis.

(2) HORMONAL FACTORS

Melanocyte stimulating hormone (MSH) is secreted by anterior pitutary gland. It strongly affects skin pigmentation by its effect on the intracellular distribution of melanin granules that is by clumping or dispersion of the granules. The intensity of pigmentation depends not only on the quantity but also on its distribution. M.S.H. is the principal hormone which controls behaviour of melanocytes. Adrenocorticoids counteract MSH and thus balanced synthesis of melanin is regulated. The following are the clinical examples of the dispersal effect of MSH (i) dark circles round eyes when under stress, strain or anxiety. (ii) Hyper pigmentation in Addison's disease (iii) Increased MSH in pregnancy gives rise to darkening of nipples and chloasma. Progesterone also has a direct MSH like action. Sex hormones also produce hyperpigmentation in a number of ways.

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(3) *Inhibitors* : Sulf-hydryl containing substances retard melanin formation (4) Inflammation and Irritation.

Mild ones excite hyperpigmentation and strong ones lead to hypo or depigmentation by stimulating or destroying melanocytes respectively. Mechanism is different in different conditions e.g. in vitiligo, achromia parasitica etc.

(5) *Racial* : The number of melanocytes is the same in all skins from the black of negroes to white of Caucasions. It is the amount and dispersion of melanin which causes colour changes. Even in vitiligenous and albin skin melanocytes are present but are not functioning probably due to partial or total absence of tyrosinase. Off-springs inherit the median colour of the parents.

Melanosis : Melanotic hyperpigmentation is known as melanosis which is readily recognised in patients with a high tanning potential. The colour of the hair and/or irises does not always give a clue to the tanning power of the skin.

CLASSIFICATION OF MELANOSIS

(1) *Prenatal* : It may be generalized or localized. The localized variety includes (i) familial melanosis of terminal phalanges (ii) lentigo profusa and (iii) freckles which are found on exposed surfaces like face etc. 5% Hydrar ammoniatum ointment, co_2 snow and ethyl chloride spray are recommended treatment for freckles. Generalized type is less common.

(3) ACQUIRED MELANOSIS.

(i) *Lentigo* : It is a dark brown macule which may form flat nevi. Lentigenes are common on vermilion border and on the dorsal surface of hands. Senile lentigenes occur after the midage. According to Miescher they are primitive seborrhoeic keratoses.

(ii) *Hormonal melanosis* : (a) *Pregnancy* : melanosis is the commonest. It involves such areas as genitalia axillae nipples, areola and linea nigra. It is due to oestrogens. It decreases after delivery.

(iii) *Physical Melenosis* : (a) Solar melanosis may occur by direct, indirect and photosensitization mechanism. The common example of genetic photosensitization is xeroderma pigmentosum. Treatment of the latter consists in eugenic prevention of marriage of cousins, if possible. Acquired type of photosensitization melanosis may occur due to various substances in occupation, industry, and plants. This type of melanosis may persist for months even after the cause is removed. (b) *X-ray* and *radium* rays. (c) Melanosis calorica (d) *Tattoo* : the pigment is located in papille and therefore the removal of tatoo pigment is difficult.

(6) Systemic Melanosis (i) Localized (a) Perioral lentigo profusa (b) Kala-azar (c) Acanthosis nigricans (d) chloasma or melasma (e) porphyria cutanea tarda.

(ii) *Generalized* : (a) addison's disease (b) anorexia nervosa (c) Blood dyscrasias (d) Pellagra (e) drugs like ACTH and Cortisone.

(7) Disorders of central nervous system e.g. hepato lenticular degeneration.

(8) Post-inflammatory melanosis. This is a common occurrence. Rothman presented the hypothesis that in inflammatory skin disease, the sulfhydryl compounds in the melanocytes are oxidized or otherwise destroyed, thus permitting tyrosinase to act on tyrosine and produce melanin. Examples are (i) Syphilis (ii Lichen Planus (iii) Dermatitis Herpetiformis (iv) Urticaria pigmentosa (v) Sensitization dermatitis and fixed eruption (vi) palmar melanosis (vii) vagabondism (viii) Trichophyton sulfureum dermatitis and T Versicolour (ix) Incontinentia pigmenti (x) Poikeloderma (xi) Arsenic and lichenoid drug eruptions (xii) Reticuloses and malignancies.

(9) Mucosal Melanosis.

This occurs in mucocutaneous junctions and adjacent mucosae like that of mouth. The patches are brown to black and occur so commonly in dark complexioned persons and races, that they may be considered normal:—

(i) Conjunctival melanosis is seen in avitaminosis A, lentigo, and premalignant melanosis (ii) oral melanosis is seen in Addison's disease, Penty Jeher's syndrome, porphyria, after ACTH, Atabrine glossitis, in tobacco chewers, malaria and Kala-azar. (iii) Vaginal and cervical melanosis : frequent in our women and in others with uterine prolapse and melanoplakia. Besides this melanotic pigmentation, various other pigmentation due to metals, ochronosis etc. may occur.