ACANTHOSIS NIGRICANS

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Acanthosia Nigricans is a rare dermatosis and is usually recognized as 2 distinct types (i) The benign type and (ii) the malignant type. Clinically as well as histologically, the cutaneous manifestations are essentially, the same in both the types. The dermatosis is characterised by the appearance of dark brown to black pigmentation studded with discrete or agglomerated papules, nodules, papillomatous growths or vegetative masses, symmetrically distributed in certain areas of predilection. According to Pollitzer (1890) who reported the first case and coined the term Acanthosis Nigricans, the axillae, neck, external genitalia, groins, face, inner aspect of the things, flexor aspect of the elbows and knees, umbilical region, perianal region, dorsal aspects of the hands, breasts, gluteal region, hypogastrium, forearms, perineum and eye lids, are involved in that order of frequency. The skin markings are usually intensified. Pigmented nevi, pigmented warts and seborrhoeic warts are often seen scattered over these areas. Occasionally, the lesions are seen in the mucous membrane of the mouth, pharynx, eosophagus and vagina. Rarely the involvement may be universal. Hyperkeratosis of the Palms and soles has been noted in some cases. There may be partial or complete alopecia of the scalp. Nails may show varying degrees of dystrophy.

The benign acanthosis nigricans is also known as the juvenile type because it starts early in life at birth, in childhood or at puberty. The hyperpigmentation and papillary hypertrophy occur in a milder degree and after reaching its height during adolescence the disorder remains stationary or even shows partial or complete regression. It is not associated with any internal malignancy. It is suggested that B. A. N. is activated by the steroid hormones at puberty. However, it is not accompanied by obesity or endocrine disturbances.

The malignant acanthosis nigricans is also called the adult type because it appears in middle or late life. But no sharp division between the two types can be made on the basis of age alone, because a few cases of M. A. N. have been reported in infancy and childhood. The dermatosis itself is not malignant but it is so called because 50 to 80% of cases appearing in adult life may be associated with a fatal adenocarcinoma of stomach, intenstines, cervix, ovary, breast, lung, oesophagus or prostate.

Apart from the two main types, there is a third type known as pseudoacanthosis nigricans. This type is regularly associated with obesity or with endorcrine disorders

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TABLE No. I

Name, Age and Sex	Case 1 (Fig. 1)	Case 2 (Fig. 2)	Case 3 (Fig. 3)
	Sattemma, Hindu, Female 35 years.	Raghava, 18 years. Daughter of Case 1.	Satti Babu, 2 years, son of case 1.
Complaints	Increasing pigmentation and small growths in neck and axillae.	Same as case 1.	Same as case 1.
Duration	5 years,	2 years.	About 3 months.
Skin lesions:			
a) Type of lesions:	Deep brown to black pigmentation studded with numerous papules of 1 to 5 mm. dia. and a few pigmented mucules.	Same as case 1.	Same as case 1, but papules smallers in size, measuring 1 to 2 mm.
b) Distribution.	Neck (on all sides) Retro-auricular areas, face, axillae, waist, groins, flexor aspects of elbows and knees. A cafe au lait patch of about $1\frac{1}{2}$" on right arm.	Neck, axillae, waist and groins.	Papules on neck and axillae. Only tiny pin point to pin head sized pigmented spots in groins. A cafe au lait patch of 1" dia. on left side of chest.
Mucous membranes.	Normal	Normal	Normal
Hair	Diffuse thinning of scalp hair.	Normal	Normal
Nails	Normal	Normal	Normal
Systemic association	Nil	Nil	Nil

Biopsy :—(All three cases)—There is evidence of hyperkeratosis and keratotic plugs. There is hyperplasia of the epidermis, especially the basal and prickle cell layers. The number of pigmented cells in the basal layer is increased. Few histiocytes deeper down in the underlying stroma, are seen which are filled with melanin pigment. The appearances are in favour of Acanthosis Nigricans (Fig. 4).

like pituitary adenoma, polycystic ovaries and adrenal insufficiency. Perlman mentions of many instances in which the benign type is found in association with genital hypoplasia, cryptorchism, acromagaly, achondroplasia, dwarfism and diabetes.

To the best of our knowledge, no case of acanthosis nigricans is reported in Indian literature. Hence, 3 cases of benign acanthosis nigricans seen in a mother and her only 2 children are being reported below : (Table No. I).

DISCUSSION

The etiology of acanthosis nigricans is completely unknown. It has been thought to be due to toxins, metabolic and and endocrinal disurbances and other factors having a direct or indirect effect on the chromaffin and sypmpathetic system. Curth (quoted by Perlman) states that many inherited anomalies have been noted among patients with benign acanthosis nigricans. eg., Von Reckling hausen's disease, mental retardation (one case), degenerative disorder of the spinal cord involving the pyramidal tracts, epilepsy and mental retardation (one case), chondrodystrophy, dwarfism and mental retardation in association with diabetes mellitus. One case was associated with rudimentary kidney, ureter, and a supernumerary nipple. The benign acanthosis nigricans is a genodermatosis with either a regular or irregular dominance and some times occurs in several members of a family. Baer and Kopf have mentioned that the relationship of benign acanthosis nigricans to nevus verru-

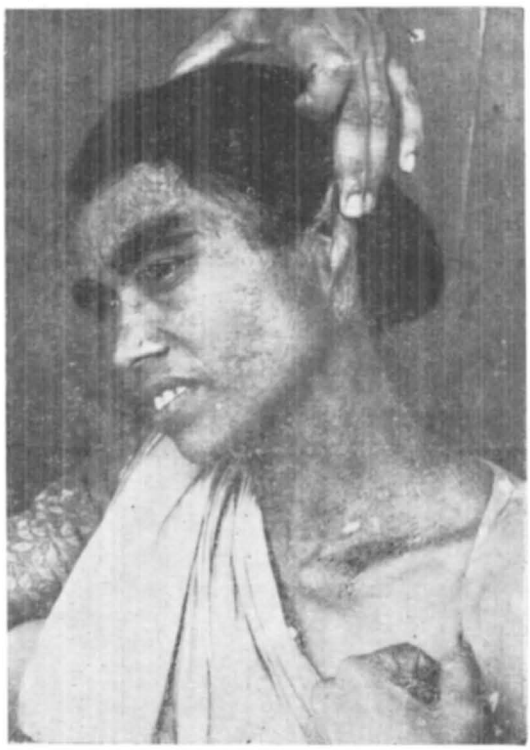

Fig. I

Fig. 2

Fig. 3

Fig. 4

Fig. 5

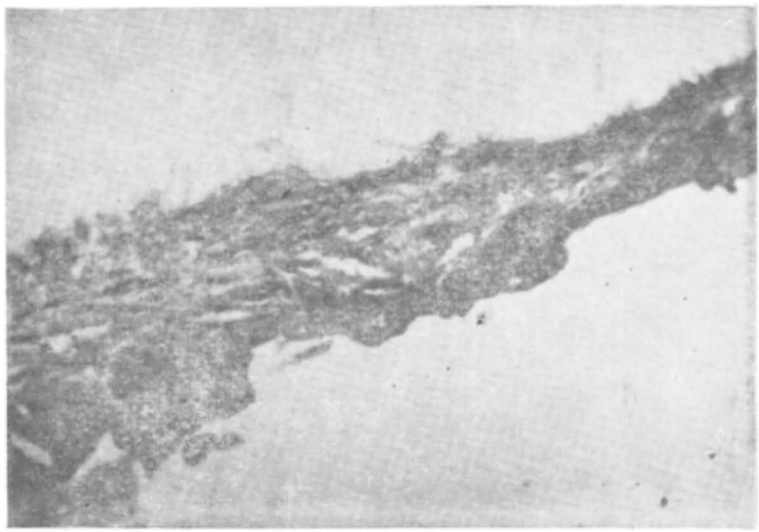

Fig. 6

Fig. 7

cosus and Icthyosis hystrix of a localised type is yet to be determined. According to Andrews, in many respects it resembles a systematised verrucous pigmented nevus. Pillsbury et al believe that abortive forms are probably not rare and are usually diagnosed as nevoid anomalies. The same authors are of the opinion that 2 separate genes may be responsible for Acanthosis Nigricans. The presence of "Skin Gene" alone results in benign acanthosis nigricans. The presence of "Cancer Gene" alone results in cancer and the presence of both the genes together results in the full picture of malignant acanthosis nigricans.

Chronologically acanthosis nigricans and cancer are independent; the skin lesions may appear either before or after the tumour. The dermatosis spreads along with the growth of the associated cancer. Resection of the Cancer is some times followed by temporary mild regression of the cutaneous lesions. The malignant type is not familial.

COMMENT

In the cases reported above, the mother, her daughter and son had cutaneous lesions of Acanthosis Nigricans. In the mother, the dermatosis had a late onset at the age of 30 years. In her daughter, it appeared at the age of 16 years and in her son at the age of 1 year and nine months. It was interesting to note that the mother was not having the disorder when her daughter was born; but still her daughter developed the lesions at puberty. Because of the absence of any symptoms or signs pertaining to internal cancer, all the 3 cases were diagnosed as cases of Benign Acanthosis Nigricans. These cases support the view about the genetic transmission and familial nature of the dermatosis.

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REFERENCES

1. Andrews, G. C.: Diseases of the skin Ed. 4. W. B. Sounder Co., Philadelphia (1954).
2. Baer R. L., akd Kopf. A. W.; The year book of Dermatology (1962–63) Year Book Medical Publishers, Chicago.
3. Curth (quoted by) Perlman, H. H.: Pediatric Dermatology. Year Book Publishers, Chicago (1960).
4. Lever, W. F.: Histopathology of skin (1961).
5. Pillsburry D. M., Shelley, W. B. and Kaligman, A. L.: Dermatology W. B Sanders Company, Philadelphia (1956).
6. Pollitzer (quoted by) Ormsby O. L. and Montgomery H.: Diseases of the Skin Lea and Febiger, Philadelphia (1954).