INDIAN J DERMATOL VENEREOL LEPROL 1985; 51 : 236-237

PSORIASIS IN MONOZYGOTIC TWINS

Pranesh Nigam and R D Mukhija

A pair of monozygotic twins developed a similar pattern of psoriatic lesions in respect of the distribution of lesions and the time of onset of the disease, in spite of living in different living conditions. This suggests a strong influence of genetic factors. Clinically, one had guttate psoriasis and the other, the nummular psoriasis. The possible mode of inheritance is autosomal recessive trait.

Key words : Monozygotic twins, Psoriasis, Inheritance.

Psoriasis sometimes shows a familial occurrence. Many reports[1,2] indicate a hereditary pattern. Various stndies [2,3] suggested that a single dominant gene with incomplete penetration was responsible. Recent advancement in leucocyte alloantigen analysis of psoriatic patients has revealed that certain HLA types are significantly increased in psoriatics,[4-6] suggesting the importance of genetic factors in the pathogenesis of this disorder. We report an instance of monozygotic twins who manifested psoriasis in late childhood in spite of being separated in life and living in different environmental conditions after birth.

Case Report

Twin sisters, aged 13 years had skin trouble at the age of 10 years, which gradually increased in severity. They had well-defined papulovesicular lesions of variable size from 0.5 cm to 3 cm, scattered all over the trunk and limbs. Skin lesions were oval and/or irregular in shape, reddish in colour with bluish tint and covered with silvery white plaques which on scraping gave bleeding points (positive candle grease sign). One of the sisters (IV-7, Fig.1) had her scalp covered with thick scaly papules with intervening normal skin, without any loss or matting of hairs. Pitting of the nails was present. She was diagnosed as a case of guttate psoriasis.

From the Department of Medicine and Dermatovenereology, BRD Medical College, and Nehru Chikitsalaya, Gorakhpur-273 013, India.

Address correspondence to : Dr. Pranesh Nigam, Type-IV Block-8 No.-2, BRD Medical College campus, Gorakhpur-273 013, India.

The other sister (IV-8) had skin lesions similar to nummular psoriasis. Blood group of both the sisters was B Rh(+) and serum uric acid levels were 1.5 and 1.4 mg/dl respectively. Laboratory investigations including haemogram, ESR and electrophoretic pattern of serum proteins were within normal range.

Skin biopsy (IV-7) revealed epidermal thickening (acanthosis) with elongation of the rete ridges forming elongated dermal papillae, over which the epidermis was thinner than normal epidermis. There was infiltration of mononuclear and round cells with oedema of the papillae and upper dermis. Parakeratotic hyperkeratosis was present. The capillaries were dilated and tortuous.

Both of them were alike in size (height 145 cm and 146 cm respectively)and mental and physical developments. There was one placenta for both the sisters at the time of delivery as told by their parents. No other details were known.

Family history revealed that both the patients were born as monozygotic, twin sisters (Fig. 1) but after a year or so, case (IV-8) was adopted by a relative. Her father (III-5) reported that his father's sister (II-5) had the same type of skin lesions. There was no other psoriatic patient either on the paternal or maternal side. Their other brothers and sisters (IV-1 to IV-6) were normal.

Comments

Various studies of large families[1] suggested that a single dominant gene with incomplete

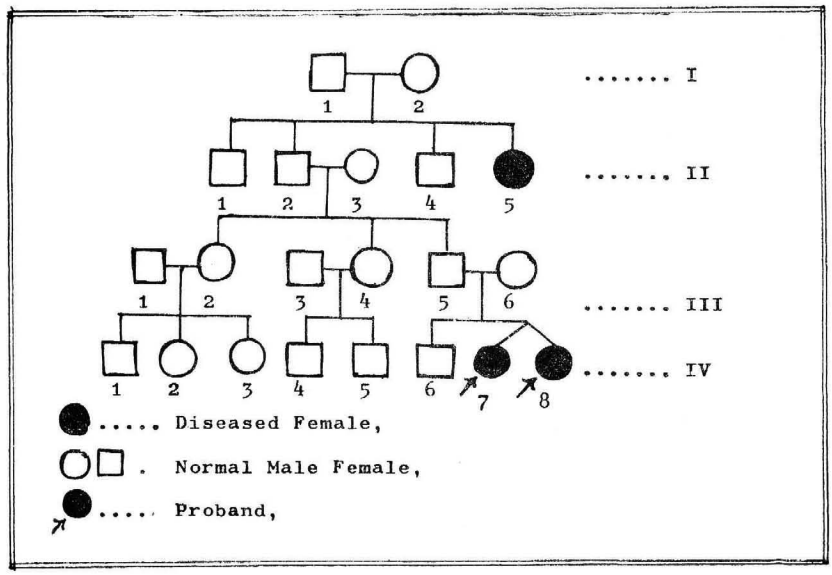

Fig. 1. Family tree of the two twins.

manifestation was responsible for this disease. Sibs were frequently affected specially when one of the parents also had the disease, and in one series this has occurred in 11.9% of the cases.[2-5] Farber[6] recorded psoriasis in 10.6% of brothers, sisters and children, 4.2% of their uncles, aunts, grand fathers and grand mothers, and 1.4% of their cousins, while only 2.6%, 0.4% and 0.9% respectively of the control relatives were psoriatic. Watson et al[3] analysed 33 pairs of identical and 31 pairs of unidentical twins where atleast one twin was psoriatic. Both twins were affected in 21 out of 33 identical pairs and 6 out of 31 unidentical pairs suggesting the importance of the genetic factor as well as the environmental factor in the development of psoriasis. Mori et al[7] have also stressed on the genetic factor in the development of psoriasis in their monozygotic twins. Our patients were monozygotic twins as there was a single placenta (placenta of single ovum), they were alike in size and mental and physical characteristics, their blood group was same and both were females, thereby fulfilling the criteria of monozygotic twins of Paul[8] and Finn Ronald.[9] Development of skin lesions with a similar distribution, in late childhood or early adolescence despite their different living conditions, is a good example of a strong genetic influence. The family tree suggests the possibility of autosomal recessive inheritance.

References

1. Goldstein JL and Brown MS : Genetics and human disease, in : Harrison's Principles of Internal Medicine, 10th edition, Editors, Petersdorf RG, Adams RD, Brawnwald E et al : Mc-Graw-Hill International Book Co, New York, 1983; Part III Section 1, p 311-323.
2. Ananthakrishna R : On the genetics of psoriasis, J Genet, 1974; 61 : 142-146.
3. Watson W, Farber EM, Cavin M et al : The genetics of psoriasis, Arch Dermatol, 1972; 105 : 197-207.
4. Ozawa A : Hereditary and HLA antigens among psoriatics, Jap J Dermatol, 1976; 86 : 205-216.
5. Steinbergh AG : Inheritance in psoriasis, Amer J Hum Genet, 1951; 3 : 267-271.
6. Farber EM : Epidemiology and genetics of psoriasis, Jap J Dermatol, 1971; 81 : 632-639.
7. Mori N, Yoshikawa K and Ohno M : Psoriasis occurring in young monozygotic twins, J Dermatol (Tokyo), 1980; 7 : 71-73.
8. Paul S : About monozygotic twin pregnancy, Canad Med Assoc, 1969; 100 : 254-259.
9. Finn Ronald : Blood groups : twin studies and blood groups, in; Clinical Genetics, 2nd edition, Editor, Sorsby A : Butterworths and Co Ltd, London, 1973; Section II, Ch 17, p 440-441.