FAMILIAL DISCOID LUPUS ERYTHEMATOSUS
(Discoid Lupus Erythematosus in a brother and sister)

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Summary

A Familial occurrence of discoid lupus erythematosus is reported in a brother and sister.

The incidence of lupus erythematosus in families is quite rare although its occurrence is known since 1901 when Rona[1] described the condition in a brother and sister. The familial discoid lupus is still rarer than the systemic variety. In 1903 Sequeira[2] reported two families each with two sisters of the discoid variety. Von Gruenhagen[3] reported discoid lupus erythematosus in identical twins. McCuistion and Schoch[4] reported discoid lesions in a newborn infant whose mother subsequently developed systemic lupus erythematosus. Steagall et al[5] reported the discoid lupus erythematosus in identical twins which is considered to be still a rarer phenomenon.

Although there have been a few reports in western literature regarding the familial incidence of discoid lupus erythematosus, yet to the best of our knowledge there has been no such report from India. Main purpose of this report is to present the occurrence of discoid lupus erythematosus in a brother and sister.

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Received for Publication on 19—2—1973

Case No. 1

In December, 1972, one male patient aged 25 years was admitted in Dermatology Unit, Rajendra Hospital, Patiala, with the complaints of scaly erythematous eruption on the cheeks, nose, ears and areas behind the ears for the last 2 years. Similar lesions were present on the mucous membrane of both lips and more marked on the lower lip. In addition to this, patient had macular erythematous patches on both palms and chilblain like lesions were also present. No history of arthritis, fever or any other complaint. History of photosensitivity was present. It was diagnosed as Discoid Lupus Erythematous.

Past History

No significant illness in the past.

Family History

Similar disease in younger sister for the last 3 years. Parents living and healthy. Other siblings were normal.

Skin Biopsy

Discoid Lupus erythematosus.

Laboratory data —

L. E. cell, ESR, Hb, TLC, DLC, Platelet Count, R.B.C. Count, P.C.V., Peripheral blood film, Blood urea, Total

serum proteins, VDRL, M.C.H, M.C. H.C, M.C.V., X-ray hands, X-ray Chest, are within normal limits, E.C.G. shows Sinus tachycardia.

Fig. 1

Chronic Discoid Lupus Erythematosus. Showing white, depigmented, atrophic scar on the nose with peripheral hyperpigmentation and erythema of the surrounding skin.

Case No. 2

Younger sister of case No. 1 aged 20 years, also attended the hospital same day with her brother. She suffered from scaly erythematous lesions on the nose, cheeks, ears and lips for the last 3 years. Lesions were most prominent on the nose. She was diagnosed as Discoid Lupus Erythematosus later confirmed on biopsy. History of photosensitivity was positive. There was no other complaint.

Past History

No significant illness in the past.

Family History

Elder brother i.e. case No. 1 has similar disease for the last 2 years. Parents and other siblings were normal.

Skin biopsy

Discoid Lupus Erythematosus.

Laboratory data —

L. E. cell, ESR, Hb, TLC, DLC-Platelet count, R.B.C., P.C.V., Peripheral blood film, X-ray Chest NAD Blood urea, Total serum proteins, VDRL, MCH, MCHC, MCV, E. C. G., and X-ray chest are within normal limits.

Fig. 2

Chronic Discoid Lupus Erythematosus. Showing white, depigmented, atrophic scar on the nose with peripheral hyperpigmentation and erythema of the surrounding skin.

Discussion

Because of increasing number of reports in recent years of the familial incidence of lupus erythematosus, there is a strong argument that hereditary

factors play an important role in the etiology of the condition. The transplacental passage of LE factor has been demonstrated by some of the authors from the mother to the new born infant (Bridge & Foley[6]. Berman and Oliver[7] McCuistion and Schoch[4]). It is quite understandable from the fact that gammaglobulin associated with immunity against infections can pass the placental barrier and LE factor is also a type of gamma-globulin.

The LE factor is also known to pass the breast milk because the intensity and duration of LE cell phenomenon is far greater in a child who is breastfed as observed by Berlyne[8] et al in 1957. It may, however, be pointed out that transplacental passage and transbreast passage of LE factor cannot explain the occurrence of LE in father and child and such cases have been reported in literature Griffin[9] et al. Besides, it cannot explain the fact that the disease may not manifest itself in the child in a number of cases until adult age. Moreover all the children in the family are not afflicted with this disease.

Leonhardt[10] has reported hyper gamma-globulinaemia in patients suffering from SLE and in their asymptomatic relatives and this appears to be an inherited phenomenon.

Common environmental factors in a family are also blamed in the etiology of the familial basis of the condition but as all the members do not suffer from the disease, it may not be a consistent factor. Another possible explanation for the familial occurrence of the condition appears to be its operation through the genes. This can explain the occurrence in a father and his child and involvement of some of the members only. Tuffanelli[11] et al brought forward another evidence in support of genetic predisposition and found the association of other collagen disease in some members with SLE in other members of the family for example poikiloderma atrophicans vasculare and SLE in one family and dermatomyositis with SLE in another family.

The surmise from the above discussion is that all the above combined factors may have a role to play in the occurrence of familial cases of Discoid Lupus Erythematosus.

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