# ABNORMAL HEMOGLOBIN IN A PSORIATIC FAMILY (A case report)

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## Summary

A Sindhi family with psoriasis and abnormal hemoglobin HbD in some of the members of the family is reported. The significance of this finding as an added genetic marker of psoriasis is discussed.

Familial aggregation of cases of psoriasis is well documented<sup>1</sup>. Among various factors that are blamed in the production of this disease, predisposition might be an important one4. It has been postulated that the disease may be transmitted as an autoirregularly dominant trait2. somal. In spite of widespread research, the etiological factors for the production of the disease, by and large, remain unknown. With this background we started exploring the role of genetic factors in the etiology of psoriasis. During the study of various biochemical genetic markers in psoriatic families we came across 2 members of a family suffering from psoriasis who showed the presence of abnormal hemoglobin on Hb electrophoresis.

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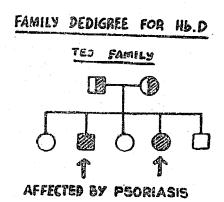
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# Case Report

Tej, a Sindhi family residing in Poona was referred to the genetic clinic, department of Medicine, B. J. Medical College, Poona for psoriasis and studies of various genetic markers. The diagnosis of psoriasis was based on history and clinical examination. Two members in the family were affected by psoriasis. Details of the family pedigree are shown in the accompanying diagram.



All the family members were subjected to the following.

1 ml of blood was drawn and Hb electrophoresis performed on cellulose

Electrophoresis on acetate paper<sup>2</sup>. cellulose acetate membrane at pH 8.4 revealed a conspicuous band of Hb occupying a position similar to HbS in some members. Some members showed homozygosity for the Hb while some others were heterozygous. A sickle cell preparation was mader undereduced oxygen tension by dithinite method which failed to reveal sickling. The Hb thus was HbD and not HbS. This was also proved by solubility test and electrophoresis at acid pH (6.1). The patients who were homozygous for HbD had psoriasis. They had minimal hepatosplenomegaly but not a very significant anaemia. They had no jaundice and were asymptomatic. Out of 7 members studied 2 were homozygous HbD. The parents were heterozygous for HbD (family pedigree).

#### Discussion

A report on a family with psoriasis, which showed the presence of an abnormal Hb-HbD in the blood of its members is presented. Reports of association of psoriasis and alterations in the Hb electrophoretic pattern have

been documented1. The present report points out the association of HbD with psoriasis. Those members who were homozygous for HbD had psoriasis while the others who were heterozygous did not have psoriasis. This may point to an association of the two. The genetic background for psoriasis still remains to be established, besides the various environmental factors that play a role in it. The finding reported here may be useful as a new genetic marker in psoriasis. Further studies on a large number of families may prove beneficial in throwing more light on the problem.

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