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HAEMATOLOGY

Hereditary Ovalocytosis and Haemoglobin E-Ovalocytosis in Malayan Aborigines

THE red blood cells of mammalian vertebrates, including man, are normally round, except those of the camel, which are characteristically oval in shape. Less than 15 per cent of the red blood corpuscles in normal healthy men are oval, but in severe anaemias the number of oval cells in the peripheral blood may become much higher. This symptomatic ovalocytosis in severe anaemias is to be distinguished from hereditary ovalocytosis, or elliptocytosis, first described by Dresbach[1] in 1904. In the hereditary condition, more than 25 per cent of the red blood cells are oval or elliptic, and elliptic rod-shaped cells are more common than in the symptomatic condition. The two conditions can usually be distinguished, with experience, but the diagnosis can only be confirmed by demonstration of a family occurrence. Incidence of the hereditary condition in the general population has previously been estimated at approximately 0·04 per cent[2,3], although more prevalent in Dutch, German and Italian families[4,5]. A focus of high frequency of hereditary ovalocytosis (elliptocytosis) has been reported from central Celebes[6]. I now report another focus of high frequency of hereditary ovalocytosis (elliptocytosis) in Malayan aborigines.

Blood smears of 440 persons comprising two groups of aborigines, mostly from the Senoi tribe, were examined. A group of 152 healthy males was made up of 110 jungle fighters of the Senoi Praak regiment and 42 personnel of the Ulu Gombak Aboriginal Hospital. The other group consisted of 288 patients from the same Aboriginal Hospital. In the healthy group, 15 soldiers and 6 Aboriginal Hospital personnel were found to have ovalocytosis with many elliptic rod-shaped/red cells, while 33 of the 288 hospital patients had ovalocytosis. The total frequency was 12·3 per cent among the 440 persons.

Although, except in 2 cases, no confirmation of the family trait could be obtained, as all relatives lived in the deep jungle, this condition was believed to be hereditary, as none of the healthy group was anaemic, while many elliptic cells, rather than oval cells, were seen in the blood. Frequency of this abnormality in the aborigines was in marked contrast to findings among 150 healthy individuals of different non-aboriginal races in Malaya, none of whom had ovalocytosis of the blood. Among patients from the General Hospital at Kuala Lumpur referred to the Haematology Division of the Institute, mostly for anaemia, 990 were examined for their peripheral blood picture and only three (two Malays and one Chinese) were found to have hereditary ovalocytosis, confirmed by study of the family trait. This hospital serves Chinese, Indians and Malays. Although many of the patients suffering from anaemia were found to have numerous oval cells in the blood, these were readily recognized as symptomatic and could be differentiated from the aboriginal ovalocytosis.

Since the aborigine has a high frequency of the gene for abnormal haemoglobin E, ranging between 8 and 50 per cent in different groups[7], it is to be expected that the new genetic combination of the gene for ovalocytosis with the gene for haemoglobin E, not reported until now, is not rare in this population. Indeed, of 18 cases of ovalocytosis among healthy aborigines, whose haemoglobins could be analysed, I found 2 associated with haemoglobin E in the blood, and of 22 cases of ovalocytosis among Aboriginal Hospital patients whose blood was examined for abnormal haemoglobin, 4 carried haemoglobin E. Attempts are being made to examine the families, and results of full haematological studies will be reported elsewhere in collaboration with Dr. J. M. Bolton of the Aboriginal Hospital, Ulu Gombak.

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I Antigen in Leukaemic Patients

P. J. SCHMIDT *et al.* have observed 20 diminutions or disappearances of I antigen in 20 patients out of 116 investigated cases[1]. Among these, 15 were suffering from various forms of chronic or acute leukaemia.

We for our part have observed, following many authors, ABO modifications in various leukaemic patients[2,3]. We have also observed a decrease of inhibiting power of Gm factors in these diseases. Table 1 shows our results. We should point out, however, that these modifications are almost always observed in patients suffering from acute leukaemia.

Table 1. BLOOD GROUP MODIFICATIONS IN LEUKAEMIAS

System	No. of cases	No. of modifications
Gm	79	6
ABO	66	1 (A) and 3 (H)
Rh	62	0

We have also investigated I antigen in healthy subjects and leukaemic patients. Our results (Table 2) indicate that we have not observed a decrease of I antigen in the