Correspondence

Accepted: December 18, 1990

Acta Haematol 1991;86:51-52

Frequency of HLA-A, B and DR Antigens in Patients with Polycythemia vera

C.		Stavropoulos ^a
F.		Petropoulou ^a
M.		Christophoraki ^a
E.		Theodosoudi ^a
E.		Kandidaki ^b
M.		Papanickolaou ^a
M.		Mavrikakis ^b
H.		Gyftaki ^b

^aGeneral Hospital of Athens, Immunology Department and National Tissue Typing Laboratory; ^bAlexandra Hospital of Athens, Nuclear Medicine Department, Athens, Greece

Dr. C. Stavropoulos, Immunology Department, National Tissue Typing Laboratory, General Hospital of Athens, Athens (Greece)

Polycythemia vera (PV) is a chronic disease of insidious onsets and unknown etiology, developing a myeloproliferative disorder with numerical and structural abnormalities that occur in the bone marrow. PV has a genetic base, since an abnormality in the chromosome 1, 8, 9 and 20 was detected, especially in terms of a partial elimination of the chromosome 20 long arm [1]. The association between human lymphocyte antigens (HLA) and various hematological malignancies has been studied extensively. However, relatively scarce attention has been paid to the investigation of HLA in patients with PV, while few, incomplete studies have been published [2–4] to elucidate this matter. As far as HLA-DR are concerned, the authors failed to spot any other such study in literature.

This study aims at the investigation of the possible associations existing between HLA and PV. Thirty-six unrelated Greek patients (22 males and 14 females) with PV were typed for the major histo-compatibility antigens class I (A, B) and class II (DR). The diagnosis of the disease was made according to Berlin's criteria [5]. Patient's age ranged from 22 to 80 years, averaging 56.1 years. The control population comprised 500 healthy volunteers for HLA-A, B, as well as 83 for HLA-DR.

HLA typing was performed by use of an 80-anti-serum panel according to Terasaki's microlymphocyte-toxicity technique, which allowed the detection of 14, 23, and 6 antigens at loci A, B and DR, respectively.

The results were analyzed by the χ^2 test after Yate's correction had been applied. The p values were then multiplied by the number of antigens tested – p corrected – and the relative risk was defined.

The HLA-B52 in PV patients was found to be increased (11.1% of 36 patients compared with 2.8% of 500 controls) ($\chi^2 = 4.82$, $p = 0.028$), which, however, makes a difference of little importance when corrected for the number of antigens tested (p corrected = 1.04). On the other hand, no statistically significant difference was found as concerns the remaining HLA class I antigens.

The HLA-DR frequencies are shown in table 1. In

52

Stravropoulos/Petropoulou/Christophoraki/Theodosoudi/Kandidaki/Papanickolaou/Mavrikakis/Gyftaki

Table 1. HLA-DR frequency rates in 36 patients with PV, in comparison with 83 healthy controls

HLA-DR

PV (n = 36)

Controls (n = 83)

2

X

P

RR

P

corrected**

n

%

n

%

DR1
12

33.3
17
20.5
2.25
NS
1.94
NS
DR2

13

36.1
29
34.9
0.02
NS
1.05
NS
DR3
8

22.2
19
22.9
0.01
NS
0.96
NS
DR4
3

8.3
18
21.7
3.08
NS
0.33
NS
DR5
11

30.6

30
 36.1
 0.35
 NS
 0.78
 NS
 DR7
 15

 41.7
 14
 16.9
 8.38 7.09*
 0.0078
 3.52
 0.047
 * Yates corrected χ^2 .

** p values
 multiplied
 by
 no tested
 antigens.

this study, an HLA-DR7 increased frequency of statistically high significance was found in PV patients (41.7%), in comparison with the control group (16.9%) ($\chi^2 = 7.09$, $p = 0.0078$, p corrected = 0.047 and $RR = 3.52$). HLA-DR4 was found to be decreased (8.3% in comparison with 21.7%) albeit it has not reached statistical significance ($p = 0.079$).

Our data have not been supported as yet by other researchers' findings. However, Miller [3] found the HLA-B5 in 5 out of 9 PV patients; Nagy et al. [4] showed an increased frequency rate of HLA-B5 and B7; Benjamin et al. [2] also found an increased frequency rate of HLA-BW21. In this study, despite the increase in HLA-B52 in PV, the whole antigen HLA-B5 (B51 + B52) does not differ considerably from that focused in the controls (33.3% compared with 27.4%). On the other hand, HLA-B7 and B21 (B49 + B50) have similar frequencies in patients (8.3% and 5.6%) and controls (11.2% and 7.6%).

In view of the fact that the HLA play a primary biological role in susceptibility to various diseases [7], our findings support the hypothesis of a possible genetic predisposition to PV in correlation with the class II HLA. Further family studies could provide more support to this hypothesis. Data bearing family predisposition to PV have been published [8].

References

Kosek M: Medical genetics; in Krupp MA, Chatton MJ, Tierney LM (eds): Current Medical Diagnosis and Treatment. Los Altos, Lange Medical Publications, 1986, pp 1039–1064.

Benjamin D, Zamir R, Gazit E, et al: HLA antigens in polycythemia vera. Tissue Antigens 1978; 11:65–67.

Miller YM: HLA-A antigens and hematologic malignancy. Arch Intern Med 1974;133:397–399.

Nagy G, Stenszky V, Murvay K: Tissue antigens and cytotoxic antibodies in polycythemia rubra vera. Haematologia (Budap) 1975;9:279–282.

Berlin NI: Diagnosis and classification of the polycythemias. Semin Hematol 1975;12:339–353.

Terasaki PI, McClelland JD: Microdroplet assay of human serum cytotoxins. Nature 1964;204:998–1000.

Svejgaard A, Platz P, Ryder LP: HLA and disease 1982 – A survey. Immunol Rev 1983; 70:193–218.

Stamatoyannopoulos G, Motulsky AG: Genetics of polycythemia and erythrocytosis; in Klein H (ed): Polycythemia: Theory and Management. American Lecture Series, 1973, pp 25–41.