ISSN 1996-3351

Asian Journal of Biological Sciences

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Asian Journal of Biological Sciences 6 (6): 292-299, 2013
ISSN 1996-3351 / DOI: 10.3923/ajbs.2013.292.299

Study of HLA-typing Class I in Patients Suffering from Vitiligo

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ABSTRACT

The pathogenesis of vitiligo is still not clear. The relationship between human leukocyte antigen (HLA) and vitiligo is controversial. The aim of this study was to evaluate the HLA class I in patients suffering from vitiligo. In this analytical case-control study, venous blood samples of 50 healthy subjects as control and 50 patients with vitiligo are evaluated to determine the type of human leukocyte antigens with microcytotoxicity technique. The control group was selected from healthy kidney donors that were typed for HLA class I antigens. HLA-A2 (P = 0.009, OR = 2.901), B49 (P = 0.031, OR = 9.333), CW2 (P = 0.008, OR = 10.756), CW3 (P = 0.031, OR = 9.333), CW7(P = 0.008, OR = 10.756) were determined as predisposing antigens and HLA-A3 (P = 0.019, OR = 0.316), B8 (P = 0.031, OR = 0.107), BW4 (P = 0.000, OR = 0.008), BW6 (P = 0.001, OR = 0.206) as preventive antigens for the disease. HLA A2, HLA B49, HLA CW2, HLA CW3 and HLA CW7 are regarded as genetic susceptible factors for manifestation of the disease and HLA A3, HLA B8, HLA BW4 and HLA BW6 as genetic susceptible factors for manifestation of vitiligo.

Key words: Vitiligo, human leukocyte antigen, antigen

INTRODUCTION

Vitiligo is a pigment disorder appearing as acquired lack of pigmentation (Fardiazar *et al*., 2012; Nikanfar *et al*., 2012; Sadeghpour *et al*., 2012). Histologically, it is characterized by lack of epidermic melanocyte seen as white depigmented patches with natural margin or hyperpigmentation (Goldust *et al*., 2011; Palermo *et al*., 2005; Rojana-Udomsart *et al*., 2012; Sadeghpour *et al*., 2011). About 8.8-14% of people suffer from the disease at different parts of the world (Ganjpour Sales *et al*., 2012; Shakeri *et al*., 2013; Vahedi *et al*., 2012). The disease pathogenesis is unknown. But it has been made clear that genetic and immunological factors play a significant role in its developing (Golfurushan *et al*., 2011; Jin *et al*., 2012; Kaufman *et al*., 2005; Milan *et al*., 2011). Available studies suggest that some genetic mechanisms rare involved in its etiology and talk about its polygenic nature (Bowcock and Fernandez-Vina, 2012; Goldust *et al*., 2012; Sadighi *et al*., 2011). HLA molecules have an important function in regulating the immunoresponse. The relationship between HLA antigens and most autoimmune diseases has been well identified (Goldust *et al*., 2013a; Vafaee *et al*., 2012; Zhu *et al*., 2011). A relationship has been proposed between different HLAs classes I and II and vitiligo and it is supposed that those with special HLA subtypes are susceptible to the disease more than others (Goldust *et al*., 2013b, c; Spritz, 2012) According to the suggestions, three locus of two autosomal alleles interacting epistatiscally (concealing effects of one gene due to appearing of another one) are involved in this

disease pathogenesis (Goldust and Rezaee, 2013; Liu *et al*., 2012; Mohebbipour *et al*., 2012). Therefore, the suffered patients are overcome considering all three homozygote locus (Karzar *et al*., 2012; Nourizadeh *et al*., 2013; Seyyednejad *et al*., 2012). Vitiligo may be a self-immune disease in which antibody (vitiligo antibodies) is created in human body against melanocytes (Goldust *et al*., 2013d; Kim *et al*., 2011; Lotti *et al*., 2013). The disease has been transmitted from the patients suffering from vitiligo to those received marrow graft or lymphocyte infusion (Farhoudi *et al*., 2012; Salehi *et al*., 2013a, b; Singh *et al*.,. 2012). Within the last twenty years, inheritance nature of vitiligo as well as probability of being a self-immune disease have provided the possibility of any relationship between vitiligo and polymorphic determinants of human leukocyte antigen (HLA system) (Fardiazar *et al*., 2013; Ganjpour Sales *et al*., 2013; Hu *et al*.,. 2011; Soleimanpour *et al*., 2013). Positive and negative relations between vitiligo and HLA antigens have been described in different population races (Daghigh *et al*., 2013; Nemati *et al*., 2013; Salehi *et al*., 2013c). Depending on race and nationality, the disease and related HLAs distribution varies at different parts of the world (Berti *et al*., 2011; Qadim *et al*., 2013; Razi *et al*., 2013; Salehi *et al*., 2013d). The present study aims at evaluating HLA-Typing Class I in patients suffering from vitiligo in order to determine antigens suscepting or preventing HLA in vitiligo.

MATERIALS AND METHODS

This case-control analytical study conducted on 50 patients and 50 healthy subjects from July 2010 to July 2012. Out of 50 patients suffering from vitiligo, 24 (48%) were males and 26 (52%) females at age range of 14-60 years old. Mean age of the patients was 31.50 years (SD = 11.61). The 50 healthy subjects were consisted of 33 (66%) males and 17 (34%) females. Venous blood samples of 50 healthy subjects (as the control group) and 50 patients with vitiligo were evaluated to determine type of leukocyte antigens using microcytotoxicity method at Imam Reza hospital, Tabriz. The case group was selected from among healthy kidney-grafting subjects underwent HLA-Typing Class I. SPSS version 16b was used as analytical software. Prevalence of HLA antigens belonging to the patients and healthy subjects was compared using Chi-square Test. Fischer's Exact Test was used in case of lack of the conditions required to use Chi-square Test. The odds ratio was calculated if the relationship was meaningful ($p<0.05$).

RESULTS

Frequency of HLA antigens belonging to the patients and healthy subjects is seen in Table 1. In comparison with 38% healthy subjects, HLA-A2 was positive in 64% of the patients ($p = 0.009$, OR = 2.901). In comparison with 2% healthy subjects, HLA-B49 was observed in 16% of the patients ($p = 0.31$, OR = 9.333). In comparison with 2% healthy subjects, HLA-CW2 was seen in 18% of the patients ($p = 0.008$, OR = 10.756). Comparing with 2% healthy subjects, HLA-CW3 was observed in 16% of the patients ($p = 0.031$, OR = 9.333). In comparison with 2% healthy subjects, HLA-CW7 was positive in 18% of the patients ($p = 0.008$, OR = 10.756). Similarly, HLA-A2, HLA-B49, HLA-CW2, HLA-CW3 and HLA CW7 were significantly high in the patients and are known as suscepting antigens for vitiligo. In comparison with 14% of the patients, HLA-A3 was positive in 34% of the healthy subjects ($p = 0.019$, OR = 0.316). In comparison with 2% of the patients, HLA-B8 was positive in 16% of the healthy subjects ($p = 0.031$, OR = 0.107). In comparison with 2% of the patients, HLA-BW4 was seen in 72% of the healthy subjects ($p<0.001$, OR = 0.008). Comparing with 84% of the patients, HLA-BW6 was observed in 52% of the healthy subjects ($p = 0.001$, OR = 0.206). Similarly, HLA-A3, HLA-B8, HLA-BW4 and HLA-BW6 were

Table 1: Prevalence of HLA Class I antigens in patients and the control group

Antigen	Case (%)	Control (%)	χ^2	p	Or
A1	10	18	1.329	0.249	
A2	64	38	6.763	0.009	2.901
A3	14	34	5.482	0.019	0.316
A11	6	8	0.154	1.000 (FET)	
A23	6	16	2.554	0.110	
A24	14	26	2.250	0.134	
A25	14	6	1.778	0.182	
A26	0	4	2.041	0.495 (FET)	
A29	8	0	4.167	0.117 (FET)	
A30	2	4	0.344	1.000 (FET)	
A33	0	4	2.041	0.495 (FET)	
A36	4	0	2.041	0.495 (FET)	
B5	46	48	0.40	0.841	
B7	4	8	0.709	0.678 (FET)	
B8	2	16	5.983	0.031 (FET)	0.107
B12	16	8	1.515	0.218	
B13	10	8	0.122	1.000 (FET)	
B14	2	6	1.042	0.617 (FET)	
B16	8	0	4.167	0.117 (FET)	
B17	8	10	0.122	1.000 (FET)	
B18	8	10	0.122	1.000 (FET)	
B21	20	20	0.000	1.000	
B22	2	10	2.873	0.204 (FET)	
B27	6	2	1.042	0.617 (FET)	
B35	18	22	0.250	0.617	
B38	6	8	0.154	1.000 (FET)	
B39	2	0	0.010	1.000 (FET)	
B40	2	8	1.895	362 (FET)	
B42	4	0	2.041	0.495 (FET)	
B45	2	0	1.010	1.000 (FET)	
B49	16	2	5.983	0.031 (FET)	9.333
B51	4	8	0.709	0.678 (FET)	
B53	10	2	2.837	0.204 (FET)	
B57	2	0	1.010	1.000 (FET)	
B63	0	2	1.010	1.000 (FET)	
BW4	2	72	52.553	0.000	0.008
BW6	52	84	11.765	0.001	0.206
CW2	18	2	7.111	0.008	10.765
CW3	16	2	5.983	0.031 (FET)	9.333
CW4	52	38	1.980	0.159	
CW5	6	0	3.093	0.242 (FET)	
CW6	0	2	1.010	1.000 (FET)	
CW7	18	2	7.111	0.008	10.765
CW8	2	0	1.010	1.000 (FET)	

OR = Odd Ration

meaningfully low in the patients and are known as preventing antigens for vitiligo. About 70% of the patients suffered from the disease when they were younger than 20 years. The lesions were mostly appeared on organs and face. About 30% of the patients had positive family records, 8% suffered from diabetes Type I and 2% suffered from hypothyroidism.

DISCUSSION

In this study, some HLAs (including A2, BW6, B5 and CW4) were more prevalent than the other ones in both control and patients groups considering evaluation of HLA-Typing. Accordingly, A2 was positive in 64% of the patients and 38% of the healthy subjects, BW6 was positive in 52% of the patients and 84% of the healthy ones, B5 was positive in 46% of the patients and 48% of the healthy subjects and CW4 was positive in 52% of the patients and 38% of the healthy subjects. The comparative study conducted between the control and patients group considering HLA-Tying, statistical evaluations made it clear that HLA A2, HLA B49, HLA CW2, HLA CW3 and HLA CW7 was meaningfully high in the patients of our region and are regarded as susceptible factors for manifestation of the disease in patients with positive values in these HLAs. Additionally, the studies demonstrated that HLA A3, HLA B8, HLA BW4 and HLA BW6 was meaningfully low in the patients and are regarded as preventive factors of vitiligo in patients with positive values in these HLAs (Goforoushan *et al*., 2013; Ha *et al*., 2010; Quan *et al*., 2010; Yousefi *et al*., 2013). These results are different from those results obtained at different parts of the world. It confirms the importance of race and nationality in differences between those HLAs suscepting the disease (Adams *et al*., 2008; Azimi *et al*., 2013; Nejad *et al*., 2013; Jalel *et al*., 2010). In their study in Kuwait, Al-Fouzan *et al*., introduced HLA B21 and HLA CW6 as suscepting gens and A19 as preventive gene of vitiligo (Al-Fouzan *et al*., 1995). In the previous studies different HLAs were introduced suspecting vitiligo (Fain *et al*., 2006; Yang *et al*., 2005). In our study, positive relation was observed between vitiligo and HLAs A2, B49, CW2, CW3 and CW7 and negative one between vitiligo and HLAs A3, B8, BW4 and BW6. In this study, positive relation was not observed between vitiligo manifestation and HLAs BW60, CW6, A3, A30, B13, B21, B27, BW6 and BW35. Also, the negative relationship found between vitiligo and HLA A19 in other studies was not observed in our study. Evaluating HLA-Typing Class I in patients and control groups and comparing these two groups demonstrated that HLA A2, HLA B49, HLA CW2, HLA CW3 and HLA CW7 are regarded as genetic susceptible factors for manifestation of the disease and HLA A3, HLA B8, HLA BW4 and HLA BW6 as genetic susceptible factors for manifestation of vitiligo. Considering inheritance nature of the disease, role of genetic in its manifestation as well as accompanying of the disease with some autoimmune diseases, conducting genetic evaluations will certainly be useful in this regard especially studies conducted on human leukocyte antigens (Lucchese *et al*., 2005). So, it is recommended to identify all HLAs suscepting and preventing from the disease in every region and race. The patients and healthy members of patients' family were screened considering suscepting HLAs and abstention from risk factors such as sun burn, trauma and stress were emphasized if any positive result was observed considering these HLAs.

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