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BRIEF COMMUNICATION

Serum levels of dehydroepiandrosterone sulfate (DHEAS) in ocular toxoplasmosis

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There are no previous study about ocular toxoplasmosis and serum levels of dehydroepiandrosterone sulphated hormone (DHEAS). We use the chemoluminiscence automatized Immulite assay to determine the levels of DHEAS. Four groups were studied: (1) Individuals with chronic asymptomatic infection with a positive test for IgG anti-*Toxoplasma* and without ocular lesions ($n = 16$); (2) Chronic asymptomatic patients with retinal scars of retinochoroiditis by *Toxoplasma* ($n = 19$); (3) Acute symptomatic patients with active retinochoroiditis by *Toxoplasma* ($n = 26$); (4) Individuals with negative assays for IgG anti-*Toxoplasma* ($n = 21$). Comparison of DHEAS levels between groups were adjusted by age and sex and non-parametric Kruskall Wallis statistical tests were applied. No significant differences in serum levels of DHEAS were found between groups when age and sex were controlled. DHEAS levels were not significantly different in active ocular toxoplasmosis related to non active or non infected persons.

Introduction

Ocular toxoplasmosis is the most common infectious cause of retinochoroidal inflammation in immunocompetent individuals and is one of the most important causes

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of visual damage in some countries.[1] Thus, in Colombia, it is estimated that 6% of the population has retinochoroidal scars after a non congenital infection and 20% of these persons have reduced visual capacity.[2] Recurrent retinochoroiditis is a typical characteristic of ocular toxoplasmosis and the factors related to this remain to be discovered.[3] In a field study in Brazil, a relation between puberty, gender and apparition of new lesions in ocular toxoplasmosis was described.[4] A hormonal factor triggering recurrences has been evocated but the precise hormones linked to these events have not been studied.

In recent years there has been strong speculation that puberty-related hormones, dehydroepiandrosterone (DHEA) in particular, might play some role in inducing resistance during *Schistosoma*, HIV and malaria infections.[5–7] DHEA is one of the steroid hormones produced by the adrenal cortex and is found mostly in its sulfate form, DHEA sulfate (DHEAS); it is converted to DHEA by DHEA sulfatase.[8] The secretion of DHEAS is high just before birth and again at puberty, and the maximum concentration is reached at around 20 to 30 years of age, followed by a progressive decline with age. DHEAS is implicated in age-related changes in the immune system and has been associated with disease susceptibility.[9] To our knowledge, no report has been made of the DHEAS levels during human toxoplasmosis

Materials and methods

Patients

Four groups of patients were studied: (1) acute symptomatic patients with active retinochoroiditis by *Toxoplasma* ($n = 26$); (2) chronic asymptomatic patients with retinal scars of retinochoroiditis by *Toxoplasma* ($n = 19$); (3) individuals with chronic asymptomatic infection with a positive test for anti-*Toxoplasma* IgG and without ocular lesions ($n = 16$); and (4) individuals with negative assays for anti-*Toxoplasma* IgG ($n = 21$). Diagnosis criteria for active retinochoroiditis and chronic inactive retinal scar were as described previously.[2] Patients without ocular lesions but with positive *Toxoplasma* serology and those without ocular lesions but negative for *Toxoplasma* serology were volunteers identified during fundoscopy ocular screening at the University of Quindio.[2] Anti-*Toxoplasma* IgG antibodies were measured by the Enzyme-Linked Immunosorbent Assay (ELISA) Test (Diagnostic Systems Laboratories Inc, Webster, Texas, USA).

Determination of DHEAS

DHEAS was measured with an immunoluminometric assay (DPC, Los Angeles, USA) with an intra-assay coefficient of variation of 9%. Levels were expressed in μg/dL.

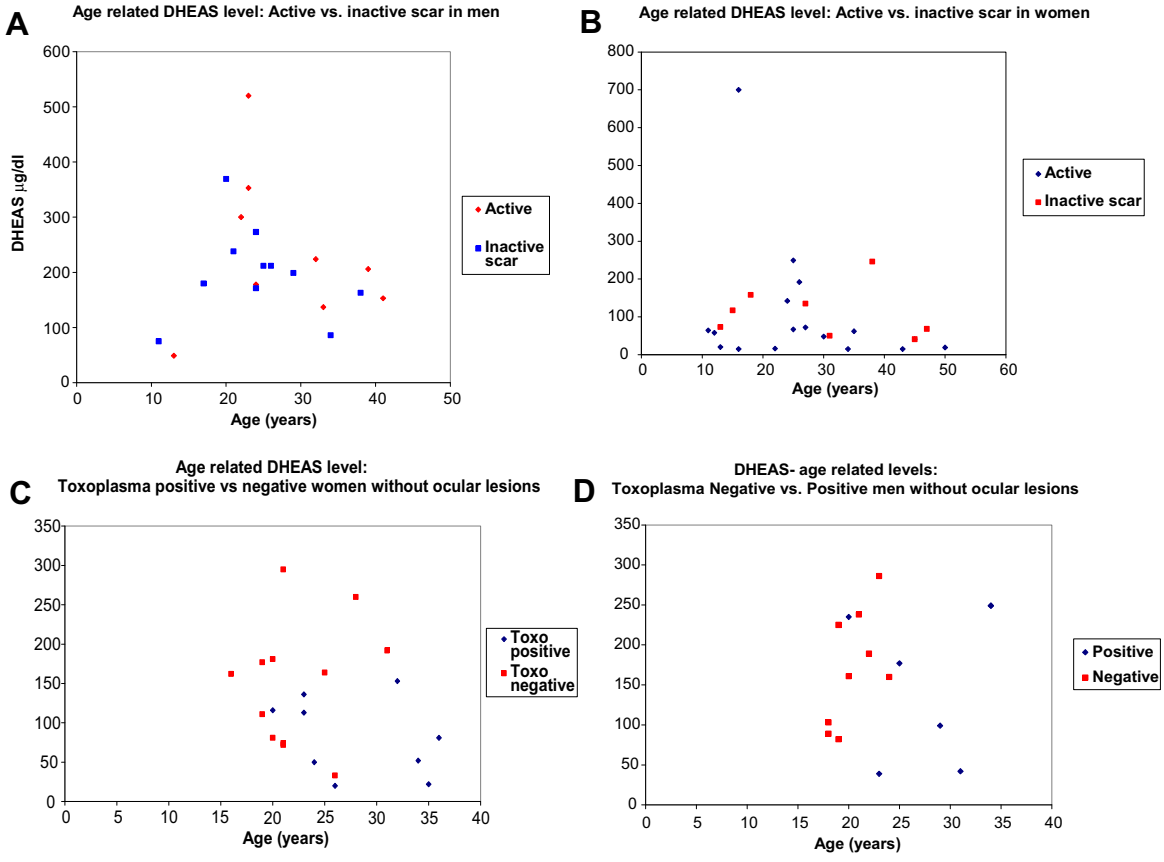

Figure 1. Age-related DHEAS levels in: (A) women with active ocular lesions versus inactive ocular lesions; (B) women without ocular lesions with positive or negative *Toxoplasma* serology; (C) men with active ocular lesions versus inactive ocular lesions; and (D) men without ocular lesions with positive or negative *Toxoplasma* serology. Each point represents one measure by patient. Values are expressed in μg/dL of DHEAS.

Table 1 Distribution of median levels of DHEAS in women by group

Women	*n*	Mean age (y) ± standard deviation	Median DHEAS levels (μg/dL)	Range	*p**
Active toxoplasmic retinochoroidal lesion	17	26 ± 11	58	15—700	—
Inactive toxoplasmic chorioretinal lesion	8	29 ± 13	95	41—246	0.12
Toxoplasma positive control without retinal lesion — age 20 to 30 y	5	28 ± 6	113	20—136	—
Toxoplasma negative control without retinal lesion — age 20 to 30 y	8	22 ± 4	122	33—295	0.30

* Kruskall Wallis test for nonparametric data, significant differences if $p < 0.05$.

Table 2 Distribution of median levels of DHEAS in men by group

Men	*n*	Age mean ± Standard deviation	Median DHEAS levels μg/dl	Range	*p**
Active toxoplasmic retinochoroidal lesion	9	27 ± 9	206	49—520	—
Inactive toxoplasmic chorioretinal lesion	11	24 ± 7	199	75—369	0.79
Toxoplasma positive control without retinal lesion	7	28 ± 5	177	39—249	—
Toxoplasma negative control without retinal lesion	9	20 ± 2	161	82—268	0.87

* Kruskall Wallis test for nonparametric data, significant differences if $p < 0.05$.

Statistical analysis

Comparisons of DHEAS levels between groups were stratified by age and sex and non-parametric Kruskall Wallis statistical tests were applied. The Epi-info computer programme (CDC, Atlanta, Georgia, USA) was used for analysis of data.

Results and discussion

Levels of DHEAS were significantly different between women (median = 73 μg/dL, range 15—70 μg/dL) and men (184 μg/dL, range 39—520 μg/dL). Subsequent analyses were made separately for men and women. We compared patients with ocular lesions with active inflammatory retinochoroidal lesion by *Toxoplasma* versus patients with an inactive toxoplasmic retinochoroidal scar and patients without ocular lesion but positive *Toxoplasma* serology versus patients without ocular lesion and negative *Toxoplasma* serology. Age means were not different between the groups with the exception of women without ocular lesions. Therefore, for these two groups of women (positive versus negative *Toxoplasma* serology), comparisons of DHEAS levels were made only between women with an age range of 20 years to 30 years. As shown in Fig. 1, DHEAS levels were higher between the ages of 20 years and 40 years. No significant differences of DHEAS levels were found by comparing the groups of active versus inactive chorioretinal lesions or in people without ocular lesions between positive or negative *Toxoplasma* serology (Tables 1 and 2).

There were no significant differences between the active or inactive lesions in ocular toxoplasmosis or in infected versus non infected patients with *Toxoplasma*. DHEAS levels were significantly affected by age and sex; when we controlled the groups for comparison, no differences were found with regards to the infection status. Interestingly, the two higher values for DHEAS in all groups were found in two patients (one male, one female) with active chorioretinal *Toxoplasma* lesions.

Although the intrinsic correlation of DHEAS and the immune system merits further exploration[10] regarding the role of this hormone in human toxoplasmosis, our present results indicate that the levels of DHEAS are not useful to indicate activity and that they are not altered significantly in ocular toxoplasmosis.

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References

1. Holland GN. Ocular toxoplasmosis: a global reassessment. Part I: epidemiology and course of disease. *Am J Ophthalmol* 2003; **136**:973—88.
2. de-la-Torre A, Gonzalez G, Diaz-Ramirez J, Gomez-Marin JE. Screening by ophthalmoscopy for toxoplasma retinochoroiditis in Colombia. *Am J Ophthalmol* 2007;**143**:354—6.
3. de-la-Torre A, Rios-Cadavid AC, Cardozo-García CM, Gomez-Marín JE. Frequency and factors associated with recurrences of ocular toxoplasmosis in a referral centre in Colombia. *Br J Ophthalmol* 2009;**93**:1001—4.
4. Glasner PD, Silveira C, Kruszon-Moran D, Martins MC, Burnier Júnior M, Silveira S, et al. An unusually high prevalence of ocular toxoplasmosis in southern Brazil. *Am J Ophthalmol* 1992;**114**:136—44.
5. Fallon PG, Richardson EJ, Jones FM, Dunne DW. Dehydroepiandrosterone sulfate treatment of mice modulates infection with *Schistosoma mansoni*. *Clin Diag Lab Immunol* 1998;**5**:251—3.
6. Abebe F, Birkeland KI, Gaarder PI, Petros B. The relationship between dehydroepiandrosterone sulphate (DHEAS), the intensity of *Schistosoma mansoni* infection and parasite-specific antibody responses. *APMIS* 2003;**111**:319—28.

7. Kandathil AJ, Kannangai R, David S, Selvakumar R, Job V, Abraham OC, et al. Human immunodeficiency virus infection and levels of dehydroepiandrosterone sulfate in plasma among Indians. *Clin Diag Lab Immunol* 2005;**12**:1117—8.
8. Kurtis JD, Mtalib R, Onyango FK, Duffy PE. Human resistence to *Plasmodium falciparum* increases during puberty and is predicted by dehydroepiandrosterone sulfate levels. *Infect Immun* 2001;**69**:123—8.
9. Parker LN. Control of DHEA secretion. *Semin Reprod Endocrinol* 1995;**13**:275—381.
10. Chen CC, Parker CR. Adrenal androgens and the immune system. *Semin Reprod Med* 2004;**22**:369—77.