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Comparison of serum apolipoprotein A-I between Chinese multiple sclerosis and other related autoimmune disease

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Abstract

Background: Serum apolipoprotein (apo) A-I was considered to be an immune regulator and could suppress pro-inflammatory cytokines generated by activated T cell in some autoimmune diseases. However, the change of serum apoA-I levels in multiple sclerosis (MS) patients is unknown.

Methods: In the presentation we performed a study on serum apoA-I levels in the patients with MS. We enrolled some age and gender matched patients with MS, autoimmune demyelinating diseases (Guillain-Barre Syndrome and Clinically Isolated Syndrome), neuroinflammatory diseases (viral encephalitis), autoimmune connective diseases (rheumatoid arthritis and systemic lupus erythematosus) and healthy control groups, and tested their serum lipids levels: total cholesterol (TC), triglyceride (TG), high-density lipoproteins (HDL), apolipoproteinB100 (apoB100), apolipoproteinA-I (apoA-I).

Results: For all patients, age had no effect on serum apoA-I levels ($P > 0.05$). Meanwhile, we proved the highest serum apoA-I levels in MS patients and the lowest serum apoA-I levels in SLE patients. Serum apoA-I levels was significantly elevated in female MS patients ($P = 0.033$; $P < 0.05$).

Conclusion: In short we believed that patients with MS and other autoimmune demyelination had significantly decreased serum levels of apo A-I.

Background

Some previous study suggested that apoA-I was the major structural protein to promote lipid transfer in human plasma, which modulated several cellular functions and involved in the pathogenesis of some autoimmune diseases [1-9]. Hyka et al. approved that apolipoprotein A-I (apo A-I) interfered interreaction between monocytes and activeted T lymphocyte, repressed activation and production of some important pro-inflammatory cytokines in the pathogenesis of some inflammatory and autoimmune diseases (including multiple sclerosis) [6,7].

Multiple sclerosis (MS) is an autoimmune demyelinating disease in central nervous system (CNS) [10,11], and some cytokines secreted by T-help cell (TH1/TH2) play the critical role in initiation and progression of MS [12-14]. Nowadays, more and more study focused on the relationship between apoA-I and autoimmune diseases including rheumatoid arthritis (RA), experimental colitis, thyroiditis and systemic lupus erythematosus (SLE) [15-18]. Although previous studies confirmed elevated serum cholestero, low-density lipoproteins (LDL) and high-density lipoproteins(HDL) during the clinical active phase of experimental allergic encephalomyelitis (EAE) (animal model of MS) [18], few studies explored the effect of apoA-I on MS. Therefore, this is the first study to investigate the relationship between serum apoA-I levels and MS patients.

Methods

In this clinic-based study, we retrospectively learned 298 hospitalized Chinese patients who had been identified consecutively, examined, treated by our medical staff

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from January 2002 to July 2008. These patients comprised of 60 Relapsing-Remitting MS patients (mean age, 35.9 ± 14.8 years; female-male, 32:28), 38 patients with Clinically Isolated Syndrome (CIS) including optic neuritis and myelitis (mean age, 36.0 ± 18.3 years; female-male, 19:19; myelitis: optic neuritis, 23:15), 28 patients with Guillain-Barre Syndrome (GBS) (mean age, 36.2 ± 20.0 years; female-male, 10:18), 51 patients with viral encephalitis (mean age, 30.0 ± 13.7 years; female-male, 25:26), 25 patients with rheumatoid arthritis (RA) (mean age, 36.3 ± 9.8 years; female-male, 20:16), 36 patients with systemic lupus erythematosus (SLE) (mean age, 31.6 ± 10.7 years; female-male, 22:14), 60 healthy subjects (mean age, 35.7 ± 10.2 years; female-male, 27:23).

In the presentation, MS patients and RA as well as SLE patients were compared, because research had shown that low serum levels of apoA-I in RA and SLE patients [15,17]. We selected the patients with viral encephalitis in order to compare serum apoA-I levels between the those patients and MS patients. To confirmed the difference between MS patients and other patients with central nervous system autoimmune demyelinating diseases, CIS and GBS patients were selected. Meanwhile, a number of age-matched healthy control group were selected.

All selected patients had never received disease-modifying immunosuppressive therapy that had the affect on plasma lipid or lipoprotein levels two months before admission. All patients were not suffering from diabetes mellitus, liver or thyroid dysfunction, hypertensive disease, cardiovascular disease, stroke, excessive alcohol consumption in their active phase. All MS patients had been diagnose with MS according to the criteria of McDonald et al [19], and scored by the Expanded Disability Status Scale (EDSS) [20]. The mean EDSS score was 3.4 ± 1.99, range 1.0-10. The mean disease course was 5 ± 3.9 years, range 0.1-18 years. All MS patients had the relapsing-remitting (RR) type, RA patients were defined by the 1988 revised criteria of the American College of Rheumatology [21], SLE patients met 1997 criteria for SLE [22].

The blood were collected to detect serum apo A-I at 6 o'clock in the morning and no eating all over the patients and healthy people.

Statistical analysis

All statistical analyses were performed using the Statistical Program for Social Sciences (SPSS) statistical software (version 11.0, Chicago, IL, USA). Results were expressed as means ± standard deviation (SD). To analyze the effect of age, gender and different entity on serum apoA-I levels in different groups, comparison of serum apoA-I levels among all male or female patients, comparison of serum apoA-I levels between male and female patients in each group using the Multivarite ANOVA. All comparisons were two-sided, with a P-value of less than 0.05 used to indicate statistical significance.

Results

Table 1 shown age at onset had little effect on serum apoA-I levels, however, different kinds of diseases ($P < 0.001$) and gender ($P < 0.05$) have different levels of serum, therefore, we first compared apoA-I levels in different disease groups not taking into account gender and age factors. We found significantly higher serum apoA-I levels in MS (1.392 ± 0.047 g/L) and other autoimmune demyelinating diseases (GBS, CIS) than healthy subjects (1.179 ± 0.047 g/L), RA (1.035 ± 0.061 g/L) and SLE patients(1.179 ± 0.047 g/L). Serum apoA-I levels in RA and SLE patients ($P = 0.002$) significantly lower than healthy control.

In order to access the impact of gender on apoA-I, we compared with male and female patients respectively (Table 2). For women, healthy control (1.230 ± 0.062 g/L) had significantly higher serum apoA-I levels than SLE patients (0.897 ± 0.068 g/L; $P < 0.001$), but significantly lower than female MS patients (1.516 ± 0.057 g/L; $P = 0.001$). Female patients with viral encephalitis (1.243 ±

Table 1: Analysis of serumapo A-I in the entire patients

Group***	MS	CIS	GBS	Viral encephalitis	SLE	RA	Healthy controls
Gender (female:male)**	32:28	19:19	10:18	26:25	22:14	20:15	27:32
Mean age ± SD*(years)	35.9 ± 14.8	36.0 ± 18.3	36.2 ± 20.0	30.0 ± 13.7	31.6 ± 10.7	36.3 ± 9.8	35.7 ± 10.2
Apo A-I (g/L)	1.392 ± 0.047	1.388 ± 0.058	1.282 ± 0.071	1.151 ± 0.051	0.940 ± 0.061	1.03 ± 0.061	1.179 ± 0.047

MS = multiple sclerosis; SLE = systemic lupus erythematosus; RA = rheumatoid arthritis; CIS = Clinically Isolated Syndrome; GBS = Guillain--Barre Syndrome; apoA-I = apolipoproteinA-I.

Data are means ± SD.* No significant differences, multivarite ANOVA, $P = 0.755$($P > 0.05$). There was no significant effect of age on serum apoA-I levels in different disease groups. ** Significantly different, multivarite ANOVA. $P = 0.04$($P < 0.05$). There was significant effect of sex on serum apoA-I levels. ***Significantly different, multivarite ANOVA. $P < 0.001$. There was significant effect of gender on serum apoA-I levels.

Table 2: Analysis of serum apoA-I in all male or female patients.

	MS	CIS	GBS	Viral encephalits	SLE	RA	Healthy controls
Female	32	38	28	26	22	20	27
Mean age ± SD*	35.7 ± 14.2	31.2 ± 17.3	29.6 ± 19.9	27.2 ± 13.6	31.4 ± 8.57	35.1 ± 7.75	38.1 ± 14.3
apoA-I (g/L)	1.516 ± 0.057	1.368 ± 0.073	1.321 ± 0.101	1.243 ± 0.064	0.897 ± 0.068	1.107 ± 0.072	1.230 ± 0.062
P**	0	0.114	0.097	0.002	0.000	0.000	0.001
Male	28	19	10	25	14	15	32
Mean age ± SD*	36.0 ± 15.6	40.7 ± 18.5	39.9 ± 19.7	30.1 ± 13.8	31.9 ± 13.7	37.9 ± 12.1	33.8 ± 4.11
apoA-I (g/L)	1.263 ± 0.075	1.422 ± 0.091	1.260 ± 0.093	1.062 ± 0.080	0.979 ± 0.106	0.963 ± 0.102	1.112 ± 0.070
P**	0	0.954	0.194	0.001	0.000	0.000	0.001

Data are means ± SD. * Significant different, multivarite ANOVA, $P = 0.159$ ($P > 0.05$). There was no significant effect of age on female or male serum apoA-I levels in different disease groups. **Compared serum apoA-I levels of between male/female MS patients and male/female patients in other groups, patients with autoimmune demyelinating diseases had higher serum apoA-I levels than other patients and healthy subjects.

0.064 g/L) showed lower serum apoA-I levels than MS patients (P = 0.002). In this study, female SLE patients had the lowest serum apoA-I levels, female MS patients had the highest serum apoA-I levels (Figure 1).

For male patients (Table 2), male MS patients (1.263 ± 0.075 g/L) had significantly higher serum apoA-I levels than male RA patients (0.963 ± 0.102 g/L; P = 0.000), male SLE patients (0.979 ± 0.106 g/L; P = 0.000) and male healthy subjects(1.112 ± 0.070 g/L; P = 0.001) (Figure 2). There was no significant different serum apoA-I levels among patients with CIS (1.422 ± 0.091 g/L; P = 0.177), GBS (1.260 ± 0.093 g/L; P = 0.978), viral encephalitis(1.062 ± 0.080 g/L;P = 0.067) and healthy subjects (1.112 ± 0.070 g/L; P = 0.142).

Finally, we compared serum apoA-I between male and female in each disease group (Table 3). The results showed that serum apoA-I levels was much higher in female MS patients (1.523 ± 0.082 g/L) and female RA patients (1.120 ± 0.042 g/L) than the corresponding male MS patients (1.262 ± 0.087 g/L; P = 120.033) and male RA patients (0.948 ± 0.049 g/L; P = 120.012) (Figure 3).

Discussion

In this study, we found age at onset have a significantly effect on serum apoA-I levels in MS patients relative to other lipid indicators (TG, HDL-C, LDL-C, apoB100), which show that apoA-I is not only associated with serum lipid metabolism, but with the pathogenesis of MS. Shore et al. considered apoA-I was significantly more concen-

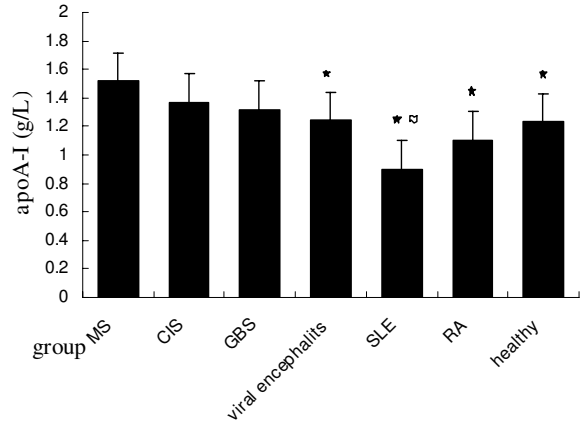

Figure 1 Comparison of serum apoA- I levels in entire female patients (black star symbol) comparison between MS and other groups, $P < 0.01$; (flag symbol) comparison between healthy controls and SLE patients, $P < 0.001$.

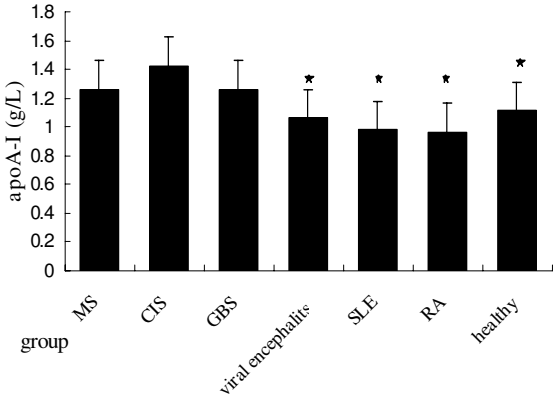

Figure 2 Comparison of serum apoA- I levels in entire male patients (black star symbol) comparison between MS and other groups, $P < 0.01$.

trated during active phase of the EAE (experimental allergic encephalomyelitis, a highly relevant model of MS) than untreated controls [18]. Similar to the Shore ea al, our research showed increased serum apoA-I levels in MS patients and other autoimmune demyelinating disease (CIS, GBS) whether male and female patients. Recently, Gaillard et al conformed that the decreasing CSF (cerebral spinal fluid) apo E (apolipoprotein E) concentrations in MS patients as apoE was postulated to be a major lipid carrier protein [23], therefore, we wonder if the CSF apoA-I concentrations would be increased in MS patients, our next task is to conform the hypothesis.

The imbalance between pro-inflammatory cytokines and anti-inflammatory cytokines would lead to autoimmune diseases such as RA, MS, SLE, atherosclerosis [24-27], these cytokines production were modulaed by contact-mediated induction between monocytes and stimulated T lymphocyte. ApoA-I bound the stimulating factor at the surface of T lymphocytes, hampered the binding of stimulated T lymphocytes with its specific receptor at monocyte surface, thus inhibited the production of pro-inflammatory cytokines including TNF-α and IL-1 [6,28]. Therefore, some researchers believed serum apoA-I concentrations should be declined during active phase of autoimmune diseases, and has played an important role in anti-inflammation, such as RA, SLE [29-31]. Consisted with above findings, in our study, serum apoA-I levels in RA and SLE patients were significantly lower than healthy subjects. It is interesting that MS patients had the highest serum apoA-I levels contrary to the hypothesis of above studies.

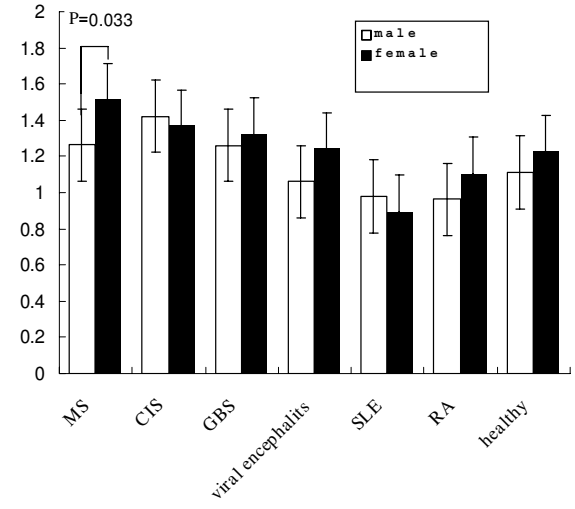

Figure 3 Comparison of serum apoA-I levels between female and male patients.

The reason remained unknown, but some emerging evidence that may explain this phenomenon. Some reports considered serum apoA-I was an inhibitory factor as a "negative" acute-phase protein, they suggested that apoA-I might be transported and get into the "leaky" blood-brain barriers by cerebral endothelial cells, and proposed apoA-I could enter the demyelinating nerve to

Table 3: Analysis of serum apoA-I between male and female patients in different diseases.

Group		Mean age ± SD (years)	apoA-I(g/L)	P
MS	**Male (n = 28)**	36.0 ± 15.6	1.2620.087	0.033*
	Female (n = 32)	35.7 ± 14.2	1.5230.082	
CIS	**Male (n = 38)**	40.7 ± 18.5	1.4070.082	0.082
	Female (n = 19)	31.2 ± 17.3	1.3700.082	
GBS	**Male (n = 28)**	39.9 ± 19.7	1.2650.086	0.876
	Female (n = 10)	29.6 ± 19.9	1.2880.116	
viral encephalitis	**Male (n = 25)**	30.1 ± 13.8	1.0760.060	0.080
	Female (n = 26)	27.2 ± 13.6	1.2210.058	
SLE	**Male (n = 14)**	31.9 ± 13.7	0.9840.067	0.296
	Female (n = 22)	31.4 ± 8.57	0.8930.053	
RA	**Male (n = 15)**	37.9 ± 12.1	0.9480.049	0.012*
	Female (n = 20)	35.1 ± 7.75	1.1200.042	
Healthy controls	**Male (n = 32)**	33.8 ± 4.11	1.1170.075	0.277
	Female (n = 27)	38.1 ± 14.3	1.2410.082	

Data are means ± SD.* Significantly different, multivarite ANOVA, $P = 0.033$ ($P < 0.05$), there was significant effect of gender on female and male serum apoA-I levels in MS patients.

regenerate impaired nerve and myelin from plasma when the blood-nerve barrier was disrupted after injury [32,33]. In recent years, some researchers conformed that astrocytes generated apoA-I and apoE in rat, apoA-I facilitated translocation of newly synthesized cholesterol and phospholipid to cytosol to form the lipid-protein complex particles as an initial event in cholesterol trafficking for the assembly of HDL, and found cholesterol efflux from rat astrocytes induced by apoA-I and apoE. In CNS, apoA-I could modulate transport of cholesterol and reduce CNS impairments by activating the brain lecithin cholesterol acyl transferase (LCAT) [34-36]. They found apoA-I had increased 26-fold in rat homogenates of regenerating sciatic nerves within 3 weeks after injury [35]. Therefore, a large number of serum apoA-I synthesized by liver will be albe to meet the remyelination during acute phase of MS.

In the study, our data showed elevated serum apoA-I concentrations in MS patients may be an important feature that is different from other autoimmune diseases which had significantly reduced serum apoA-I levels (such as RA and SLE). In order to clarify the effect of CNS inflammatory response on serum apoA-I levels, we compare neuroinflammation patients with MS patients, This study further confirmed that, compared to other CNS inflammatory diseases, the imbalance between demyelination and regeneration in MS patients may be related to elevated serum apoA-I concentrations.

Finally, the results indicated that female MS patients had significant higher serum apoA-I levels than male MS patients, but this phenomenon have not been found in other demyelinating diseases. The reason remained unknown, but it may be associated with the greater susceptibility and incidence of female MS patients.

Conclusion

MS patients had the highest serum apoA-I levels compared with other disease groups and healthy control, and female MS patients had a significant higher levels than male MS patients. Then the following work should be done to expose the reason for our results, determine the CSF apoA-I levels in MS patients and discuss relationship between serum/CSF apoA-I and anti-inflammatory cytokines.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

BZ and CG, co-designed and coordinated the study as well as prepared; BZ carried out lipids measurements, analytical work; BZ and SP carried out analytical and statistical; JY carried out analytical work. All authors read and approved the final manuscript.

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