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Relations of blood serum apolipoprotein A-I with rheumatic heart valve fibrosis

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Objective. With the aim to check whether the atherogenetic factors are involved in the mechanisms of valve fibrosis, we have studied blood serum concentrations of apolipoproteins A-I and B in patients suffering from rheumatic heart valve fibrosis.

Methods. Blood serum apolipoprotein A-I and B levels were measured in 176 subjects (83 females and 93 males): 11 women (mean age 43.2 ± 3.6 years) underwent aortic valve replacement, 29 women (mean age 41.3 ± 4.1 years) underwent mitral valve surgery, 17 male patients had operated on aortic valves (mean age 49.5 ± 11 years) and 23 men were after mitral valve surgery (mean age 47.3 ± 10.4 years). The results obtained from the patients were compared with the data on age-matched healthy females and males (n = 43; 39.5 ± 5.2 years and n = 53; 44.6 ± 7.8 years). Apolipoprotein concentrations were measured in a fasting serum sample using the ELISA method.

Results. A significantly lower blood serum apolipoprotein A-I level in all patient groups (males and females) suffering from rheumatic heart disease was determined as compared with control ($P < 0.01$). The apolipoprotein B level in rheumatic patients didn't significantly differ from that of control. The apolipoprotein B/A-I ratio for patients with heart valve fibrosis was significantly higher as compared to control in the investigated female groups – the pooled aortic and mitral valve group, aortic valve group and mitral valve group ($p < 0.05$) and in male aortic valve group ($p < 0.05$).

Conclusion. The obtained results indicate that decreased blood serum apolipoprotein A-I levels can indicate an increased risk of developing heart valve fibrosis in rheumatic patients.

Key words: apolipoprotein A-I, gender, rheumatic heart disease, valves

INTRODUCTION

Clinical and experimental studies have shown that atherosclerotic coronary artery disease, mitral and aortic valvular sclerosis could have a similar etiology (1–3). Roberts in an editorial claimed that calcific deposits in the mitral annular area are a form of atherosclerosis (4). Patients with a severe degenerative process in the mitral valves had a more severe atherosclerotic disease in the carotid artery (5). Mitral annulus calcification is associated with known atherosclerotic risk factors such as diabetes mellitus, hypertension and hypercholesterolemia (6). It is associated with an increased prevalence of severe obstructive coronary artery disease in patients under 65 years (7). Atherosclerosis risk factors and proximal aortic atherosclerosis are independently associated with aortic valve sclerosis (8, 9).

Among the most significant inherited atherogenic factors that determine the development of fibrotic atherosclerotic plaques are blood serum apolipoproteins (apo) A-I and B. Increased apoB and low blood serum apoA-I concentrations elicit cholesterol accumulation in arterial wall cells and the early formation of atherosclerotic plaques (10–12). Our earlier studies showed that apoB and apoA-I blood serum concentrations are important inherited markers in the development of coronary arteries atherosclerosis in male and female, and an increased apoB / apoA-I ratio (above 1.0) is an informative atherogenetic index (13, 14).

Derangements of apoB metabolism related to hypercholesterolemia are associated with aortic valve stenosis (15). Accumulation of apolipoproteins in the aortic valve lesions contributes to the pathogenesis of aortic stenosis (16). Early atherosclerosis, indu-

ced by a high cholesterol diet in rabbits, is characterized by significant changes in the elastic lamina of the aortic valve (17). Recently we have reported on a decreased apoA-I level in the blood serum of female patients suffering from rheumatic valve fibrosis (18).

Data reported in the present paper imply that lower serum apoA-I levels can be significant for the development of rheumatic heart valve fibrosis both in female and male.

METHODS

To study the blood serum concentration of apoA-I and apoB, 176 subjects (83 female and 93 male) have been examined. The study protocol was approved by the Lithuanian Bioethics Committee. The apoA-I and apoB levels were determined in 40 rheumatic females (pooled aortic and mitral valve group (PAMVG); mean age 41.9 ± 4.0 years) after heart valve surgery: 11 women (mean age 43.2 ± 3.6 years) had operated on aortic valve (aortic valve group, AVG) and 29 (mean age 41.3 ± 4.1 years) were after mitral valve surgery (mitral valve group, MVG), and 40 rheumatic males (pooled aortic and mitral valve group, PAMVG; mean age 47.4 ± 11 years) had been operated on with replacement of damaged valves (see Table): 17 patients had operated on aortic valves (aortic valve group, AVG; mean age 49.5 ± 11 years) and 23 patients were after mitral valve operation (mitral valve group, MVG; mean age 47.3 ± 10.4 years).

Blood serum apoA-I and apoB levels obtained for rheumatic female and male patients were compared with the data on healthy females (n = 43; mean age 39.5 ± 5.2 years) and males (n = 53; mean age 44.6 ± 7.8 years).

Apo investigations in rheumatic patients were performed no sooner that 3 months following surgery. Apolipoprotein concentrations were measured in fasting serum samples. We applied the ELISA method, using sheep polyclonal antibodies against human apoA-I and apoB (DACO). The blood serum samples were kept frozen at –40 °C until use.

Our study cohort (both healthy individuals and patients) differed statistically significantly by age ($p < 0.05$), therefore in this paper we could not assess gender-related differences in apoA-I and apoB concentrations. Thus, the results were derived only by comparing the groups of patients and healthy individuals of the same gender ($p < 0.05$).

RESULTS

ApoA-I and apoB levels in healthy and rheumatic females. Compared to the control, serum apoA-I levels were significantly lower in all groups of rheumatic women: in the PAMVG ($p < 0.01$) as well as in AVG and MVG ($p < 0.01$).

In healthy women, a negative correlation between apoA-I level and body weight ($r = –0.27$, $p < 0.05$) was found. A correlation was found between weight and the serum apoB / apoA-I ratio in healthy women ($r = 0.3$; $p < 0.05$). Such correlations were not specific for the examined groups of patients.

There was no significant difference in blood serum apoB concentration ($p > 0.05$) between the control female group and the groups of women suffering from rheumatic heart valve disease (PAMVG, in AVG and MVG). In the PAMVG females, a significant correlation was found between apoB and apoA-I levels ($r = 0.32$; $p < 0.05$).

Compared to the age-matched control group of women, the apoB / apoA-I ratio was significantly higher in all female patient groups: PAMVG ($p < 0.01$), in AVG ($p < 0.05$), and MVG ($p < 0.05$). An increase of the apoB / apoA-I ratio in the serum of the rheumatic women was essentially related to a significantly lowered apoA-I level. There were no differences in the mean values of apoB / apoA-I ratio among different rheumatic female patient groups.

An apoB / apoA-I ratio more than unity was found in 4.5% of healthy females. The frequency of the apoB / apoA-I more than 1.0 was statistically

Table. **Concentration of apolipoproteins B and A-I and apoB / apoA-I ratio in the blood serum of females and males suffering from rheumatic heart valve disease and in healthy controls**

	Females				Males			
	PAMVG n = 40	AVG n = 11	MVG n = 29	Controls n = 43	PAMVG n = 40	AVG n = 17	MVG n = 23	Controls n = 53
Age, years	41.9 ± 4.0	43.2 ± 3.6	41.3 ± 4.1	39.5 ± 5.2	47.4 ± 11.0	49.5 ± 11.0	47.3 ± 10.4	44.6 ± 7.8
ApoA-I, g/l	1.02 ± 0.22*	0.98 ± 0.21*	1.03 ± 0.23*	1.23 ± 0.23	1.13 ± 0.24*	1.14 ± 0.30*	1.12 ± 0.25*	1.40 ± 0.26
ApoB, g/l	0.93 ± 0.30	0.93 ± 0.28	0.92 ± 0.31	0.88 ± 0.20	1.07 ± 0.35	1.14 ± 0.41	1.00 ± 0.32	1.15 ± 0.22
ApoB/apoA-I	0.93 ± 0.31*	0.97 ± 0.29*	0.97 ± 0.29*	0.73 ± 0.15	0.93 ± 0.20**	1.0 ± 0.35*	0.90 ± 0.22	0.85 ± 0.23

Data are means ± standard deviation. Apo, apolipoprotein; AVG, aortic valve group; MVG, mitral valve group; PAMVG, pooled aortic and mitral valve group.
* Compared with control, $p < 0.05$; ** compared with control, $p = 0.05$.

significantly higher in the female PAMVG patients (25.0%, $p < 0.05$) compared to control.

ApoA-I and apoB levels in healthy and rheumatic males. Serum apoA-I concentration has been found to be statistically reliably lower in all groups of patients than in control ($p < 0.01$).

In healthy adult males, blood serum apoB concentration directly correlated with the apoA-I level ($r = 0.17$, $p < 0.03$). In this group of subjects a negative correlation between apoA-I level and age ($r = –0.25$ $p < 0.05$), as well as an inverse dependence of apoA-I levels on body weight ($r = –0,38$, $p < 0.05$) was found. A correlation was found between age or weight and serum apoB/apoA-I ratio in healthy men ($r = 0.38$ and $r = –0.24$ respectively; $p < 0.05$).

There was no significant difference in blood serum apoB concentration ($p > 0.05$) between the control male group and the groups of men suffering from rheumatic heart disease.

A statistically significantly higher apoB / apoA-I ratio was characteristic only of AVG ($p < 0.05$). In the PAMVG group this difference was found to be at the reliability limit ($p = 0.05$). An increase of apoB / apoA-I ratio in the serum of such patients was related with a lower apoA-I level.

A statistically significant inverse dependence between serum apoA-I concentration and age ($r = –0.26$, $p < 0.05$) was found in PAMVG patients. In this contingent, serum apoB concentration directly correlated with the apoA-I level ($r = 0.42$, $p < 0.001$), and the apoB / apoA-I ratio in blood serum was directly dependent on age ($r = 0.2$, $p < 0.05$). The latter dependence was related with age-dependent changes in serum apoA-I concentration, since the apoB concentration does not depend on age ($r = 0.04$).

An apoB/apoA-I ratio more than unity was found in 7.5% of healthy men and in 33.3% of PAMVG patients ($p < 0.05$).

DISCUSSION

Heart valvulopathies are the objective of a new scientific approach supporting the relationship between cardiac valve fibrosis and cardiovascular atherogenic factors. It has been suggested that aortic and mitral valve sclerosis is a manifestation of the atherosclerotic process (1–3, 19). The presence of aortic valve sclerosis accounts for a higher rate of ischemic events and increases mortality. The presence of aortic valve sclerosis predicts coronary artery disease: in female patients the risk for coronary artery disease in the presence of aortic valve sclerosis was found to be significantly higher than in male (20). Population studies show that the mitral valve degenerative sclerosis more often occurs in women (21).

Investigation data confirm a pathogenetic similarity between lesions of inflammatory origin observed in fibrotic aortic valves and atherosclerotic plaques (22). Studies have proved that foam cells representing early atherosclerotic lesions in affected patients can be found already during adolescence in coronary arteries, in the ventricular surface of the mitral leaflet and in the aortic valve cusps (1, 4). Hypercholesterolemia has been shown to be related to aortic valve stenosis (15). During inflammatory reactions, arterial wall elastin damaged by macrophage proteinases exhibits a stronger tendency to bind to atherogenic lipoproteins (23, 24). There is no doubt that inflammatory damage of arterial wall plays a role in the development of atherosclerotic plaques. There is a well-known relationship between heart valve damage and the pathogenesis of valve fibrosis with immune-inflammatory response (10, 25). The immune-inflammatory mechanisms of connective tissue damage are important in the processes of atherogenesis within arterial wall and aortic valve (17, 26).

Furthermore, morphological studies have shown that in fibrotic aortic valves lipoproteins containing apoB, apo(a), apoE are found along with macrophages, which is not the case in fibrosis-free parts of the valves. All these apolipoproteins emerge in the valve tissue at the early stages of heart valve damage: most apolipoproteins get into the valve tissue from the blood, and only an insignificant part is locally synthesised by the cells (16). As a consequence, the corresponding alterations appear also in the arterial wall with a possible initiation of fibrotic atherosclerosis plaque formation (27).

The data of our study corroborate the hypothesis that atherogenetic factors can modulate the progress of heart valve fibrosis in rheumatic patients. It has been shown that rheumatic female and male patients, compared to the control groups, had significantly lower blood serum apoA-I levels. A statistically reliable age-related decrease of blood serum apoA-I concentrations is characteristic of healthy males and those with rheumatic valve fibrosis. However, this is not characteristic of females suffering from rheumatic heart valve disease. We did not find statistically significant changes in apoB levels of rheumatic women and men operated on for aortic or mitral valve fibrosis, either.

Our earlier data showed that the progression of atherosclerosis of coronary arteries in the population was directly related to increased plasma apoB concentrations, lower apoA-I levels and to an increased frequency of apoB/apoA-I ratio higher than unity (13, 14). The inherited tendency to early development of atherosclerosis is related to a low blood serum apoA-I level (11, 12). Panamonta has found that rheumatic children have statistically reliably lower blood concentrations of high density lipoprotein cholesterol compared to control (28). The course of rheumatic fever is characterized by the expression of immunological and biochemical disorders. ApoA-I is the main protein component of high density lipoprotein and the high density lipoprotein

cholesterol carrier (29). Blood serum levels of high density lipoprotein and apoA-I are decreased during inflammatory states (30). An inverse relationship between the plasma apoA-I level and proteolytic activity caused by neutrophil activation has been found, i.e. the higher proteolytic activity the lower blood serum apoA-I concentration (31, 32). Increased plasma proteolytic activity is a characteristic feature of patients suffering from various inflammatory diseases, atherosclerosis (26, 32) and rheumatism (33) included.

Secretory phospholipase A2 (PLA2), an acute-phase protein, may play a key role in the pathophysiology of the phenomenon described as lowered apoA-I in chronic immune-inflammatory conditions (34). PLA2 activation is characteristic of rheumatic heart disease patients (35). PLA2 is an enzyme overexpressed in chronic inflammatory disease (36). In animals, a prolonged PLA2 overexpression may induce accelerated atherosclerosis (37). It has been shown that in chronic inflammatory conditions normal high density lipoprotein undergoes marked alterations, losing a major part of apoA-I and converting it into acute-phase high density lipoprotein, which is proinflammatory and possibly atherogenic (38). High density lipoprotein inhibits activation of monocytes, subsequently decreasing the tumor necrosis factor-alpha and interleukin-1beta production (39). ApoA-I inhibits immunoglobulin G-induced neutrophil activation (40). Low high density lipoprotein levels were found to be associated with increased levels of C-reactive protein. This kind of relationship may imply upregulation of proinflammatory mechanisms (41). ApoA-I is in association with paraxone, an enzyme located on high density lipoprotein, which reduces the oxidative modification of low density lipoproteins and thus may protect against atherosclerosis (42).

Data reported in the present study show that atherogenic changes of blood serum apoA-I concentrations in rheumatic patients could be related with an increased risk of developing fibrosis in heart valves. The anti-inflammatory as well as antiatherogenic function of apoA-I might lead to new therapeutic approaches to patients wih rheumatic fever. Elucidation of the relationship between atherogenesis and heart valve fibrosis in rheumatic patients could contribute to a better understanding of the reasons why in separate cases, even with an active antirheumatic-antiinflammatory treatment applied, heart valve damage develops into valve fibrosis. Therefore it is very important to determine the pathogenetic mechanisms and risk factors causing a progressive fibrosis of valves in rheumatic patients.

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KRAUJO SERUMO APOLIPOPROTEINO A-I SÀSAJOS SU REUMATINE ÐIRDIES VOÞTUVØ FIBROZE

S a n t r a u k a

Darbo tikslas. Išsiaiškinti, ar aterogeniniai kraujo serumo apolipoproteinø A-I ir B pokyèiai yra susijæ su reumatinës ðirdies voþtuvø fibrozës patogeneze.

Tyrimo medþiaga ir metodai. Tirti 176 asmenys, operuoti dël reumatiniø ðirdies voþtuvø ydø (83 moterys, 93 vyrai): 11 moterø (amþiaus vidurkis 43,2 ± 3,6 metai) po aortos voþtuvo protezavimo, 29 moterys (amþiaus vidurkis 41,3 ± 4,1 metai) po mitralinio voþtuvo protezavimo, 17 vyrø (amþiaus vidurkis 49,5 ± 11 metai) po aortos voþtuvo protezavimo ir 23 vyrai (amþiaus vidurkis 47,3 ± 10,4 metai) po mitralinio voþtuvo protezavimo. Ryte nevalgius paimti kraujo serumo mëginiai ir ELISA metodu iðtirta apolipoproteinø A-I ir B koncentracija, gauti duomenys palyginti su panaðaus amþiaus sveikø moterø ir vyrø (n = 43; amþiaus vidurkis 39,5 ± 5,2 metai ir n = 53; amþiaus vidurkis 44,6 ± 7,8 metai, atitinkamai) apolipoproteinø A-I ir B kiekiu kraujo serume.

Rezultatai. Visose serganèiøjø reumatu grupëse nustatytas patikimai maþesnis apolipoproteino A-I kiekis lyginant su kontrole ($p < 0,01$). Apolipoproteino B kiekis serganèiøjø reumatu ir kontrolës grupiø kraujo serume statistiðkai reikðmingai nesiskyrë. Serganèiøjø reumatinëmis ydomis apolipoproteinø B ir A-I santykis buvo didesnis tirtøjø moterø grupëse (bendroje aortos ir mitralinio voþtuvø ydø, aortos voþtuvo ydø ir mitraliniø ydø) lyginant su kontrole ($p < 0,05$) bei vyrø aortos voþtuvø grupëje, lyginant su kontrole ($p < 0,05$).

Išvada. Serganèiøjø reumatu sumaþëjæs kraujo serumo apolipoproteino A-I kiekis gali liudyti besivystanèià ðirdies voþtuvø fibrozæ.