Acta clin Croat 2000; 39:139-146 Original Scientific Paper

PROGNOSTIC IMPORTANCE OF MONITORING SERUM AMYLOID A PROTEIN (SAA) IN PATIENTS WITH CEREBRAL INFARCTION

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SUMMARY – Serum amyloid A protein (SAA) is an acute-phase plasma protein, which increases in response to tissue damage. The aim of the study was to investigate SAA in the blood of patients with cerebral infarction. Sixty subjects were studied, including 45 patients with cerebral infarction and 15 controls. SAA was determined using ELISA method on days 1, 3, 7 and 14 after ischemic stroke onset. SAA was found to increase by day 3, which was followed by a decrease if no infectious complication occurred. The concentration of SAA was statistically significantly increased on days 1, 3, 7 and 14 ($p < 0.05$), however, the increase was greater in patients with severe clinical manifestations. A statistically significant SAA increase by day 14 was recorded in patients with an accompanying infectious process. The study showed the concentration of SAA to depend on the clinical severity of ischemic stroke. SAA was found to be a sensitive early indicator of possible infectious complications.

Key words: *Cerebral infarction, complications; Cerebral infarction, blood; Amyloid protein SAA, analysis; Infection, complications*

Introduction

Acute phase proteins are synthesized by the liver as a nonspecific response to tissue damage or infection[1-3]. Serum amyloid A protein (SAA) is an acute phase protein complexed to high-density lipoproteins (HDL) as an apoprotein (apo SAA). It is mainly found in HDL3 fraction, but small amounts can be found in other HDL fractions as well as in low-density lipoproteins (LDL) and very low-density lipoproteins (VLDL). SAA is the most impressive of acute-phase proteins, increasing within 24 h by up to 1000-fold, and resulting in displacement of apo A-I, apo A-II and apo A-III proteins from the HDL complex. Six major isoforms of SAA can be extracted from the plasma HDL fraction of patients with inflammatory diseases[4-9]. The molecular weight of SAA is 11.4 – 12.5 kDa and the protein has from 104 to 112 amino acids. In man, the protein is coded on chromosome 11 p. Interleukin-1(IL-1), interleukin-6 (IL-6) and tumor necrosis factor (TNF) stimulate hepatic SAA production[10,11]. The function of SAA remains unclear[12]. SAA can influence lymphocytic responses to antigens, and plays a role in cholesterol metabolism during the course of acute inflammation[13]. Steinmetz found a significant decrease of esterified cholesterol in plasma during acute phase response due to a decreased plasma lecithin – cholesterol acyltransferase (LCAT) activity[14]. SAA induces the synthesis of collagenase and can inhibit fever induced by IL-1 or TNF [15,16]. It can suppress thromboxin synthesis and platelet release of serotonin, and inhibit platelet aggregation[17]. The acute-phase proteins, apo SAA[1] and apo SAA[2], can be precursors of amyloid AA peptides. These deposits are a complication of chronic inflammatory disorders, but the role of SAA in amyloidogenesis is still unclear[4,12]. SAA has been used in diagnosing and monitoring of different diseases, among others myocardial infarction, bacterial and viral infections, arthritis, and malignant disease[18-20]. The aim

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Received August 18, 1998, accepted in revised form July 17, 2000

of this study was to evaluate SAA concentration in the blood of patients with cerebral infarction in dependence of the disease severity, and the effect of infectious – inflammatory process on SAA concentration in patients with cerebral infarction.

Patients and Methods

Sixty patients hospitalized at the Department of Neurology were included in the study: 25 patients with cerebral infarction not complicated by infectious – inflammatory process; 20 patients with cerebral infarction complicated by infectious – inflammatory process; and 15 control group subjects. Control group (C) included 15 subjects, eight women and seven men, mean age 57 years. Patients with radicular syndrome in remission were included in this group. In the control group, SAA concentration was measured once.

The patients were divided into two groups according to disease severity:

1. patients with cerebral infarction with less severe clinical course (group E-1); and
2. patients with cerebral infarction with more severe clinical course (group E-2).

Disease severity was assessed by use of 8-point scale proposed by Fisher[21]: – up to 2 points – patients with no consciousness disturbances; 1 point – patients without feeling disturbances; up to 2 points – patients without speech disturbances; and up to 3 points – patients without motoneuron function damages. Patients with a score ≤5 were allocated to the group with a more severe course of cerebral infarction.

The group of patients with a less severe clinical course of cerebral infarction not complicated by infectious – inflammatory process (E1) comprised 13 subjects, eight women and five men, mean age 63. The group of patients with cerebral infarction not complicated by infectious – inflammatory process with a more severe clinical course (E2) included 12 subjects, six men and women each, mean age 65. The group of patients with cerebral infarction complicated by infectious – inflammatory process with a less severe clinical course (E3) had 11 subjects, six women and five men, mean age 65, while the group of patients with cerebral infarction complicated by infectious – inflammatory process with a more severe clinical course (E4) had nine subjects, five women and four men, mean age 68. Urinary tract infection was found in 11, pneumonia in seven, and inflammatory thrombotic process in limbs in two patients. In the group of patients with cerebral infarction, SAA was determined on days 1, 3, 7 and 14 after the onset of disease.

Cerebral infarction was recognized on the basis of an interview, neurologic state of patients, and finding of brain computer tomography (CT). Cerebral infarction complicated by infectious – inflammatory process was recognized on the basis of supplementary examinations (urine analysis and inoculation, chest x-ray).

SAA was determined by ELISA (enzyme-linked immunosorbent assay)[22] using a Cytoscreen TM Immunoassay kit from Biosource International. Results were statistically analyzed by Student's t-test and analysis of variance was done by use of Duncan test. Statistical significance was considered at a level of $p < 0.05$.

Results

In the control group patients, SAA concentration was determined only once in the remission period of radicular syndrome, showing a mean concentration of 2.82 ± 5.4 μg/ml. Literature reports indicate the mean concentration of this protein in serum of healthy individuals as determined by the ELISA method to be 1-2 mg/ml, with a value dispersion of 8-10 mg/l[5].

The study showed the SAA concentration in the group of patients with a less severe clinical course of cerebral infarction to increase by day 3 from the onset of disease, whereafter it was found to decline. The highest increase of SAA was recorded between days 1 and 3. The mean SAA concentration in these patients according to day of determination was as follows: 158.9 ± 5.2 μg/ml on day 1; 540.9 ± 83.3 μg/ml on day 3; 404.9 ± 65.1 μg/ml on day 7; and 236.4 ± 65.5 μg/ml on day 14. The concentration of SAA was statistically significantly higher in this group of patients than in the control group of patients on all days of determination ($p < 0.05$; diff.: 158.9 ± 10.3, $t=54.2$ on day 1; 537.7 ± 72.5, $t = 23.4$ on day 3; 401.7 ± 63.9, $t = 22.6$ on day 7; and 233.2 ± 62.9, $t = 13.3$ on day 14). In patients with a less severe clinical course of cerebral infarction, differences in SAA concentration between days 1 and 3 (381.9 ± 84.9, $t = 16.2$), days 3 and 7 (135.9 ± 133.0, $t = 3.6$), and days 7 and 14 (168.5 ± 82.1, $t = 7.3$) were statistically significant ($p < 0.05$) (Fig. 1).

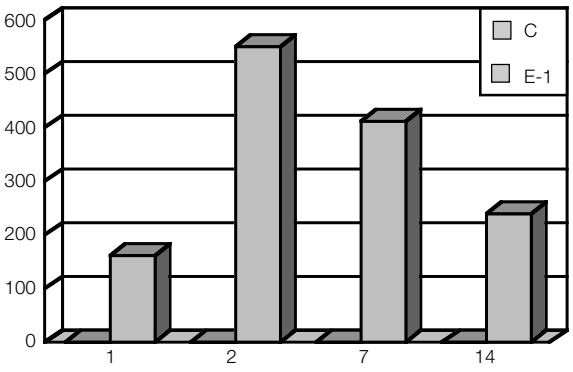

Fig. 1. SAA concentration ((g/ml) in patients with a less severe clinical course of cerebral infarction (E1) on days 1, 3, 7 and 14.

In patients with a more severe clinical course of cerebral infarction, the concentration of SAA also was on a rise by day 3, whereafter a decline was observed. The highest increase of SAA concentration was recorded between days 1 and 3. According to days of determination, the mean SAA concentration was as follows: 232.3 ± 90.8 μg/ml on day 1; 585.0 ± 55.4 μg/ml on day 3; 573.2 ± 79.8 μg/ml on day 7; and 322.4 ± 90.8 μg/ml on day 14. There was a statistically significant difference ($p < 0.05$) in the SAA concentration between days 1 and 3 (352.6 ± 116.1, t=10.5) and days 7 and 14 (245.8 ± 81.4, t = 9.5), while the difference recorded between days 3 and 7 did not reach statistical significance (6.9 ± 103.2, t = 0.2; $p > 0.05$). The concentration of SAA was statistically significantly higher in this group of patients than in the control group of patients on all days of determination ($p < 0.05$; diff.: 228.9 ± 88.3, t = 8.9 on day 1; 581.5 ± 54.3, t = 37.0 on day 3; 569.5 ± 81.0, t = 23.3 on day 7; and 318.3 ± 8.8, t = 113.2 on day 14) (Fig. 2).

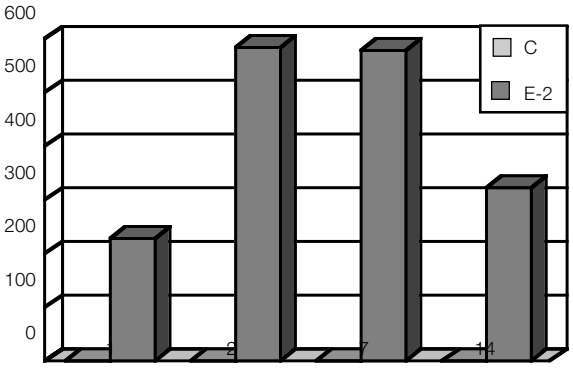

Fig. 2. SAA concentration ((g/ml) in patients with a more severe clinical course of cerebral infarction (E2) on days 1, 3, 7 and 14.

SAA concentration in patients with cerebral infarction complicated by infectious – inflammatory process was increased by day 14 from the onset of disease, with highest increase recorded between days 1 and 3. In this group of patients, the mean SAA concentration was 218.6 ± 60.4 μg/ml on day 1; 736.5 ± 109.6 μg/ml on day 3; 999.9 ± 169.7 μg/ml on day 7; and 1118.8 ± 132.2 μg/ml on day 14. The concentration of SAA was statistically significantly higher in this group of patients than in the control group of patients on all days of determination ($p < 0.05$; diff.: 215.7 ± 61.7, t = 13.5 on day 1; 733.7 ± 108.0, t = 26.2 on day 3; 997.8 ± 171.2, t = 21.0 on day 7; and 1116.0 ± 133.8, t = 26.3 on day 14). There was a statistically significant difference ($p < 0.05$) in the SAA concentration between days 1 and 3 (510.0 ± 124.5, t = 18.3) and days 3 and 7 (283.3 ± 201.6, t = 5.9), while the difference recorded between days 7 and 14 did not reach statistical significance (64.9 ± 136.5, t = 1.8; $p > 0.05$) (Fig. 3).

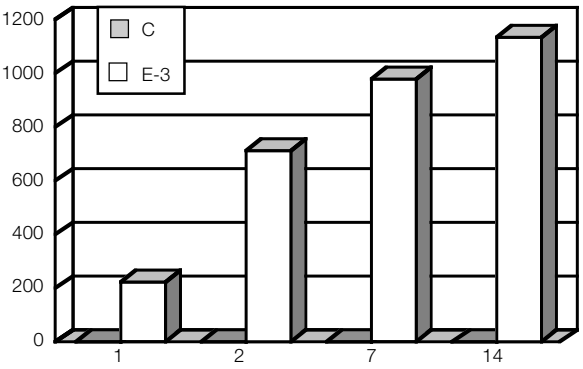

Fig. 3. SAA concentration ((g/ml) in patients with cerebral infarction complicated by infectious - inflammatory process (E3) on days 1, 3, 7 and 14.

Comparative analysis of SAA concentration in patients with cerebral infarction not complicated and complicated by infectious – inflammatory process showed highest SAA concentration in patients with cerebral infarction not complicated by infectious – inflammatory process with a more severe clinical course and lowest SAA concentration in patients with cerebral infarction with a less severe course of disease on day 1. On days 3, 7 and 14 of the disease, highest SAA concentration was recorded in patients with cerebral infarction complicated by infectious – inflammatory process, and lowest SAA concentration in patients with cerebral infarction with a less severe course of disease and not complicated by infectious – inflammatory process. The difference in SAA concentration between the cerebral infarction pa-

tients with and without a complication of an infectious – inflammatory process was especially pronounced on day 14.

Comparative analysis of SAA concentration in patients with cerebral infarction not complicated by infectious – inflammatory process with a less and more severe clinical course of disease (E1 *vs* E2) according to days of determination showed statistically significant differences ($p < 0.05$) on days 1 (74.1 ± 91.9, $t = 2.7$), 7 (161.6 ± 108.0, $t = 4.9$), and 14 (71.7 ± 67.3, $t = 3.3$). On day 3, the difference in SAA concentration did not reach statistical significance (34.8 ± 86.8, $t = 1.3$; $p > 0.05$).

Comparative analysis of SAA concentration in patients with cerebral infarction not complicated by infectious – inflammatory process with a less and more severe clinical course of disease (E1 *vs* E2) according to days of determination showed statistically significant differences ($p < 0.05$) on days 1 (74.1 ± 91.9, $t = 2.7$), 7 (161.6 ± 108.0, $t = 4.9$), and 14 (71.7 ± 67.3, $t = 3.3$). On day 3, the difference in SAA concentration did not reach statistical signficance (34.8 ± 86.8, $t = 1.3$; $p > 0.05$).

Comparative analysis of SAA concentration according to days of determination between patients with cerebral infarction not complicated by infectious – inflammatory process with a less and more severe clinical course of disease, and those with cerebral infarction complicated by infectious – inflammatory process revealed statistically signficant differences ($p < 0.05$; diff. E1 *vs* E3: 65.2 ± 59.6, $t = 3.9$ on day 1; 203.4 ± 119.6, $t = 6.1$ on day 3; 617.2 ± 170.0, $t = 12.5$ on day 7; and 912.7 ± 137.3, $t = 19.9$ on day 14; and E2 *vs* E3: 160.7 ± 111.3, $t = 5.0$ on day 3; 449.5 ± 173.6, $t = 8.1$ on day 7; and 798.2 ± 141.5, $t = 14.9$ on day 14). Only the E2 *vs* E3 difference recorded on day 1 (14.98 ± 90.3, $t = 0.5$) did not reach statistical significance ($p > 0.05$) (Fig. 4).

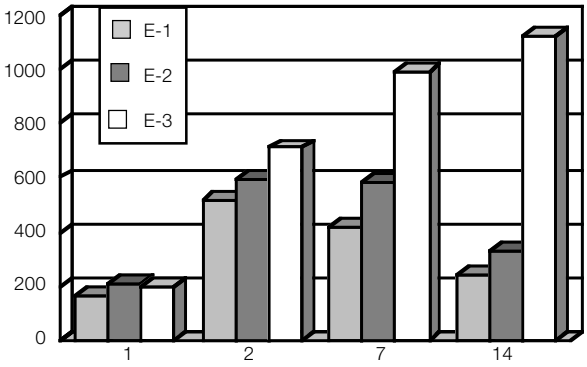

Fig. 4. Comparative analysis of SAA concentration in patients with cerebral infarction on days 1, 3, 7 and 14.

Discussion

Our own investigations showed a cerebral tissue damage due to ischemia to result in acute phase response inducing an SAA concentration increase in the blood of patients. Previous studies of the acute phase SAA pattern have pointed to its high responsivity in different disease processes[2-4,9,18,20]. Investigations of the behavioral pattern of acute phase proteins (SAA, C-reactive protein, α1-antichymotripsin, α1-acid glycoprotein, haptoglobin) have shown them to rise in the blood of patients with cerebral infarction[23,24]. According to Syrjanen et al.[2] and Marhaug et al.[18], the increase in the concentrations of SAA and other acute phase proteins is higher in myocardial infarction than in cerebral infarction. The higher concentrations of SAA in patients with non-complicated cerebral infarction with a more severe clinical course, recorded in the present study in comparison to SAA concentration in patients with non-complicated cerebral infarction with a less severe clinical course indicated a significant dependence of SAA concentration on the patient's clinical condition. Marhaug and Dowton[4] also report on a higher concentration of SAA in patients with a more severe clinical course of rheumatic disease. Widespread cerebral infarction foci leading to a more severe clinical course of the disease may cause major damage to the blood – brain barrier, which may be the reason for differences in the release of acute phase mediators and in SAA concentration between patients with a less and more severe clinical course[25]. Studies in patients with myocardial infarction showed a positive correlation between SAA concentration and infarction focus size[18]. There are no such investigations on cerebral infarction. Syrjanen et al.[2] failed to demonstrate any dependence between SAA concentration and localization of the focus of infarction as far as the supratentorial and subtentorial brain structures are considered. The role of infectious – inflammatory process in the etiopathogenesis of cerebral infarction has not yet been fully explained. Earlier studies have pointed to a higher activity of acute phase response in patients in whom an infectious – inflammatory process anticipated cerebral infarction or concurred during or after the stroke period. The possible effect of vascular inflammation during the course of infection on their occlusion and origin of cerebral infarction has been considered[2]. Our studies have confirmed previous observations that the course of cerebral infarction superimposed by an inflammatory process increases the activity of acute

phase response, expressing itself in a higher and prolonged elevation of SAA concentration in the patient's blood. In relation to this, monitoring of SAA concentration in the blood of patients may be a sensitive indicator of infectious – inflammatory complications occurring during the course of cerebral infarction. It should be remembered that a chronic inflammatory process causing hematologic stress syndrome, characterized among others by elevated levels of acute phase proteins, including serum amyloid A protein, may be the cause of secondary amyloid in the vessel walls, which in turn results in a damage to their basal membrane[12,26]. Arakawa et al.[27] describe a patient with relapsing cerebral infarction episodes in the course of secondary amyloidosis concomitant with rheumatoid arthritis. Secondary amyloidosis may be the etiologic factor for ischemic stroke, so efficient treatment of all chronic infectious – inflammatory processes is of utmost importance.

Table 1. SAA concentrations in the groups of patients with cerebral infarction (μg/ml)

Group	day 1 mean±SD	day 3 mean±SD	day 7 mean±SD	day 14 mean±SD
E1	158.96±5.24	533.22±87.71	404.90±65.09	236.39±52.41
E2	224.2±90.86	585.00±55.49	573.29±79.87	322.49±6.70
E3	193.8±25.84	683.61±179.5	956.17±148.14	1034.26±151.99
E4	243.25±66.16	770.11±75.39	1061.90±153.78	1164.01±105.37

E1 = patients with cerebral infarction not complicated by infectious - inflammatory process with less severe clinical course; E2 = patients with cerebral infarction not complicated by infectious - inflammatory process with more severe clinical course; E3 = patients with cerebral infarction complicated by infectious - inflammatory process with less severe clinical course; E4 = patients with cerebral infarction complicated by infectious - inflammatory process with more severe clinical course.

Table 2. Differences in SAA concentrations (μg/ml) in patients with cerebral infarction between days of disease onset

Group	Difference		
	Days 1-3	Days 3-7	Days 7-14
E1	374.26* t=2.63	128.32* t=2.48	168.51* t=2.62
E2	360.8* t=2.63	11.71 t=1.28	250.8* t=2.43
E3	489.8* t=2.90	272.56* t=2.83	78.0* t=2.31
E4	526.86* t=2.75	290.89* t=5.23	102.11 t=0.44

*Student's t-test; *statistically significant difference p<0.05*

Table 3. Comparative analysis of SAA concentration ((g/ml) between different groups of patients with cerebral infarction and control patient group

Day	Diff. E1-E2	Diff. E1-E3	Diff. E1-E4	Diff. E2-E3	Diff. E2-E4	Diff. E3-E4	Diff. C-E1	Diff. C-E2	Diff. C-E3	Diff. C-E4
1	65.24	34.84	84.29	30.4	19.05	49.45	156.1*	221.3*	190.9*	240.4*
3	51.78	150.3*	236.8*	98.61*	185.1*	86.5*	530.4*	582.1*	680.7*	767.2*
7	168.3*	551.2*	657.0*	382.8*	488.6*	908.1*	402.0*	570.4*	953.3*	1059*
14	86.1*	797.8*	927.6*	711.7*	841.5*	129.7*	233.5*	319.6*	1031*	1161*

*Duncan test, *statistically significant difference p<0.05*

Conclusions

1. In patients with cerebral infarction not complicated by infectious – inflammatory process, a statistically significant increase of SAA concentration was observed, which was especially pronounced on day 3 from the onset of disease.
2. In patients with cerebral infarction complicated by infectious – inflammatory process with a less and more severe clinical course, a statistically significant increase of SAA concentration was recorded by day 14 from the onset of disease.
3. Differences in SAA concentrations between cerebral infarction patients with a less and more severe clinical course may be helpful in monitoring the disease process dynamics.
4. Differences in SAA concentrations between groups of patients with cerebral infarction complicated and not complicated by infectious – inflammatory process suggest that SAA may be a sensitive indicator of inflammatory complications occurring during the course of the disease.

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Sažetak

PROGNOSTIČKA VRIJEDNOST PRAĆENJA RAZINE SERUMSKOG PROTEINA AMILOID A U BOLESNIKA S MOŽDANIM INFARKTOM

J. Iłzecka i Z. Stelmasiak

Serumski protein amiloid A (SAA) je plazmatski protein akutne faze koji raste u odgovoru na tkivno oštećenje. Cilj ovoga ispitivanja bio je istražiti SAA u krvi bolesnika s moždanim infarktom. Ispitivanje je obuhvatilo 60 osoba, od toga 45 bolesnika s cerebralnim infarktom i 15 kontrolnih osoba. SAA je mjeren pomoću metode ELISA, i to 1., 3., 7. i 14. dana nakon nastupa ishemijskog moždanog udara. Povišenje SAA zabilježeno je do 3. dana, nakon čega je uslijedilo snižavanje njegove razine ukoliko nije bilo infektivnih komplikacija. Koncentracija SAA bila je statistički značajno povišena 1., 3., 7. i 14. dana ($p<0,05$), međutim, taj je porast bio veći u skupini bolesnika s težim kliničkim manifestacijama bolesti. Statistički značajno povišenje SAA zabilježeno je do 14. dana u bolesnika s pratećim infektivnim procesom. Ispitivanje je pokazalo da koncentracija SAA ovisi o kliničkoj težini ishemijskog udara. SAA je osjetljiv rani pokazatelj mogućih infektivnih komplikacija.

Ključne riječi: *moždani infarkt, komplikacije; moždani infarkt, krv; amiloidni protein SAA, analiza; infekcija, komplikacije*