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C-reactive protein in patients with coexistent periodontal disease and acute coronary syndromes

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Abstract

Aim: To evaluate periodontal disease (PD) influence on changes in high-sensitivity C-reactive protein (hsCRP) concentrations in patients with acute coronary syndromes and coexistent PD.

Materials and Methods: Dental examinations were carried out in a group of 50 consecutive patients, less than 60 years old, hospitalized as a result of acute coronary syndromes. The patients were divided into two groups on the basis of own-constructed combined PD score (group 2: more advanced; and group 1: less advanced PD) as well as clinical attachment loss (CAL) – group 4: CAL >3 mm; group 3: CAL $\leqslant 3$ mm. Blood samples for hsCRP estimation were taken at admission, after 10/12 days and long term after acute coronary syndromes.

Results: A statistically significant decrease in hsCRP was observed among three consecutive blood sample examinations in groups 2 and 4, whereas it was only seen between examination 1 and examination 2 in groups 1 and 3.

Conclusions: Although no statistically significant difference of hsCRP was found between studied groups, patients with less advanced PD, either estimated with the use of own-constructed combined score or on the basis of CAL, have significantly longer diminution of inflammatory response monitored with hsCRP concentrations.

Key words: acute coronary syndromes; C-reactive protein; inflammatory response; periodontal disease

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In recent years, concepts of the pathogenesis of atherosclerosis and cardiovascular events, like acute coronary syndromes, have broadened from a lipid-centric view of aetiology to appreciation of the importance of inflammatory processes, including chronic periodontal disease (Czerniuk et al. 1999, Beck et al. 2005). The role of infection is believed to serve as a critical inflammatory stimulus that contributes to both atherogenesis and acute events via coronary plaque destabilization (Opolski et al. 2002). This inflammatory process can involve the vasculature directly or indirectly via modulation of haemostasis or hepatic activation of the acute-phase response that leads to increased circulating levels of acute-phase reactants such as C-reactive protein (Offenbacher & Beck 2005). Findings from many research groups clearly support relations of chronic oral infections and the inflammation of periodontitis as contributors to, or triggers for, systemic inflammatory responses (Beck & Offenbacher 2002, Ebersole et al. 2002, D'Aiuto et al. 2004a). The potential role of periodontal disease as a possible chronic source of infection and inflammation is supported by findings indicating its association with elevated serum C-reactive protein and IL-6. Moreover, periodontal therapy studies have shown a lowering of C-reactive protein (D'Aiuto et al. 2005b, Montebugnoli et al. 2005). Self-reported gingivitis, dental caries and tooth loss are associated with stable coronary heart disease (Ylostalo et al. 2006).

Only a few studies have been conducted on inflammatory response in acute coronary syndrome patients with coexistent periodontitis (Persson et al. 2005). We have previously reported the outcomes of the study on tumour necrosis factor α and IL-1 concentrations in patients with such coexistence (Czerniuk et al. 2004). The aim of this study was to evaluate the state of the oral cavity and the influence of periodontal disease on the intensity and dynamics of the inflammatory response, measured by changes in the serum concentration of high-sensitivity C-reactive protein (hsCRP) in patients with acute coronary syndromes and coexistent periodontal disease.

Materials and Methods

The study involved a group of 50 patients below 60 years of age consisting

of nine females and 41 males (mean age 51 years) admitted to Coronary Care Unit, First Department of Cardiology, University Hospital, Warsaw, Poland, with chest pain and an initial diagnosis of acute coronary syndrome. All patients had chest pain for less than 12 h and were treated according to contemporary standards of acute coronary syndrome therapy, receiving the same standard doses of drugs in secondary prevention. The study was carried out according to the ethical standards of the 1964 Helsinki Declaration and was approved by the Ethics Board of Medical University School of Warsaw, Poland. All patients signed informed consent forms.

We examined all the patients during the first 24 h of their stay at the unit. Dental examinations were carried out, indicating the condition and number of retained teeth and state of dental prostheses together with treatment needs regarding prosthetics and oral surgery. In all patients, there was a finding of chronic generalized periodontitis. A history regarding tobacco smoking was obtained, revealing 78% smokers among the target group, with a daily average of 20 cigarettes.

The initial diagnosis of acute coronary syndrome was confirmed in all patients. Depending on the electrocardiogram picture, the final diagnosis was acute ST-segment elevation myocardial infarction in 32 individuals and unstable angina or acute non-ST-segment elevation myocardial infarction in 18 individuals. The discharge diagnosis in the latter group was based on troponin criteria: troponin cut-off: troponin I $<1{,}5$ ng/dl – unstable angina– (11 cases); troponin I >1.5 ng/dl – non-ST-elevation myocardial infarction (seven cases). Maximally marked values for creatinine kinase (CK_{max}) and maximally marked values for creatinine kinase for MB fraction ($CKMB_{max}$) values were compared between groups to assess the extent of myocardial infarction. All patients were treated according to contemporary guidelines, with implementation of standard pharmacotherapy [routinely, acetylsalicylic acid (ASA) 75 mg, clopidogrel 75 mg, simvastatin 40 mg daily to all patients]. The groups only differed in the use of angiotensin-converting enzyme inhibitors and β-blockers.

Blood samples were taken from all 50 patients for hsCRP estimation. Examinations were carried out during admission to the unit (acute-phase examination 1), subsequently at 10 to 12 days of hospitalization (examination 2) and long-term observation after 3 months of hospitalization (examination 3). Long-term observation was also carried out after 6 months in 44 patients (examination 4). The samples were centrifuged and the obtained serum was frozen and stored at a temperature of -80°C until estimation. hsCRP serum levels were assessed by means of a commercial high-sensitivity ELISA kit (R & D Systems, Inc., Minneapolis, MN, USA, lower detection limit of 0.25 mg/l) according to the manufacturer's instructions.

The second part of the examination – for verification and definitive confirmation of the initial diagnosis of chronic periodontitis – was continued at the Department of Oral Medicine and Periodontology, Warsaw Medical University School. This was carried out approximately within 1 month after the acute coronary syndrome episode. The following measurements were made – probing depth (PD) using a periodontal probe type WHO 621, clinical attachment loss (CAL), level of root furcation and tooth mobility using the three-degree Entin's scale (Aleksandrov 1988) – in a way similar to that used in other studies (Fleszar et al. 1980, Czerniuk et al. 2004). The simplified plaque index (PI) and bleeding index (BI) were measured, showing the relationship of bleeding gingival units to all tooth surfaces (Ainamo & Bay 1975, Page & Schroeder, 1976). All four measurements (PI, BI, PD and CAL) were carried out for each tooth, and then their mean values were determined for each patient, respectively. Chronic generalized periodontitis was diagnosed in all patients on the basis of clinical examination in accordance with the 1999 American Academy of Periodontology classification (Armitage 2004).

Each of the four parameters (PI, BI, PD and CAL) was evaluated on a scale of 1–4 (1 = the lowest value; 4 = the highest value). The points from each parameters were then added and used to determine the extent of periodontal disease in the own-constructed combined risk score (lowest score possible = 4 points; highest score = 16 points). The 50 patients were divided into two groups on the basis of their score: group 1 (below median), $\leqslant 9$ points ($N = 27$) – less advanced periodontitis; group 2 (above median), >9 points ($N = 23$) – more advanced periodontitis.

Because CAL is a particularly significant periodontal parameter, the patients were also divided on the basis of attachment loss: group 3 – CAL $\leqslant 3$ mm ($N = 19$) and group 4 – CAL >3 mm ($N = 31$). The groups 1 *versus* 2 and 3 *versus* 4 did not differ, or differed only slightly, regarding other coronary risk factors.

Changes in serum concentrations of hsCRP were used as the primary outcome variable. To test normal distribution, the Kolmogorov–Smirnov test was used. Variables not normally distributed were logarithmically transformed before being used in parametric comparative analysis. All estimated values were averaged and expressed as mean ± standard deviation (SD) and were considered significantly different if $p<0.05$ (Student's *t*-test). Medians were used for hsCRP concentrations. Wilcoxon's paired rank-sum test was used to analyse hsCRP between examinations. Mann–Whitney U-test was used for within-group comparisons. A stepwise logistic regression model was developed to analyse the effect of baseline characteristics on any observed association with hsCRP concentration changes. Variables with significance $p<0.1$ were added to the model. SAS version 8.1 (Chicago, IL, USA) was used as a statistical package for analysis.

Results

Examination of a group of 50 patients with periodontal disease and coexisting acute coronary syndrome showed that mean values for PI and BI were very high, respectively: PI, 72% in females and 42% in males; BI, 86% in females and 78% in males.

The mean PD was 2.91 mm in females and 2.36 mm in males. The mean CAL ranged between 3.11 mm in females and 3.8 mm in males (Table 1). Periodontal and clinical characteristics for groups 1 and 2 as well as for groups 3 and 4 are shown in Table 2.

The study showed non-significant raised mean and median hsCRP concentrations in all examinations when the group with more advanced periodontitis was compared with the group with less advanced disease (group 2 *versus* 1). This was also seen when comparing the group with CAL >3 mm (group 4) with CAL $\leqslant 3$ mm patients

Table 1. Condition of periodontium and oral mucous membrane, and mean level of clinical parameters in acute coronary syndrome patient population

Gender	Plaque index (%)	Bleeding index (%)	Cigarette smoking (%)	Pathological changes on mucous membrane (%)	Clinical attachment loss (mm)	Probing depth (mm)
Females ($N = 9$)	72	86	78	22	3.11 ± 0.73	2.91 ± 0.88
Males ($N = 41$)	42	78	78	15	3.8 ± 0.9	2.36 ± 1.01

Table 2. Periodontal and clinical characteristics of patients taking part in the study

Characteristics	Group 1 ($N = 27$)	Group 2 ($N = 23$)	*p* value groups 1 *versus* 2	Group 3 ($N = 19$)	Group 4 ($N = 31$)	*p* value groups 3 *versus* 4
Plaque index (%)	38.78 ± 18.73	84.39 ± 15.04	<0.01	44.92 ± 27.58	68.86 ± 25.49	<0.01
Bleeding index (%)	25.30 ± 21/13	79.43 ± 17.40	<0.01	31.42 ± 31.82	61.71 ± 29.23	<0.01
Clinical attachment loss (mm)	2.43 ± 0.70	3.34 ± 0.83	<0.01	1.93 ± 0.35	3.41 ± 0.59	<0.01
Probing depth (mm)	2.98 ± 0.86	4.24 ± 0.59	<0.01	3.12 ± 0.92	3.82 ± 0.93	<0.01
Age (years)	49.7 ± 5.8	52.4 ± 4.9	NS	48.6 ± 6.2	52.4 ± 4.5	0.02
Gender (*N* males)	22	19	NS	16	25	NS
Arterial hypertension (%)	59	39	NS	59	45	NS
Diabetes (%)	4	26	0.02	11	16	NS
Cigarette smoking (%)	70	87	NS	90	71	NS
Hypercholesterolaemia (%)	59	61	NS	63	58	NS
CK_{max} (U/l)	1088 ± 1510	1543 ± 1493	NS	896 ± 1179	1544 ± 1642	NS
$CKMB_{max}$ (U/l)	77 ± 86	113 ± 94	NS	66 ± 68	110 ± 99	NS

CK_{max}, mean maximally marked values for creatinine kinase; $CKMB_{max}$, mean maximally marked values for creatinine kinase for MB fraction (values in international units).

Table 3. Median, mean and range of the mean concentrations of hsCRP (mg/l) in groups 1 and 2 as well as in groups 3 and 4

Serum hsCRP concentration (mg/l)	Group 1 ($N = 27$)	Group 2 ($N = 23$)	Group 3 ($N = 19$)	Group 4 ($N = 31$)	*p* value groups 1 *versus* 2 and *p* value groups 3 *versus* 4
Examination 1					NS
Median	17.50	22.00	15.50	18.00	
Mean (± SD)	32.55 (± 35.73)	42.47 (± 50.60)	36.34 (± 41.59)	37.39 (± 44.25)	
Examination –2					NS
Median	4.19	4.01	4.39	4.01	
Mean (± SD)	7.50 (± 10.5)	10.56 (± 17.44)	8.38 (± 12.66)	9.13 (± 14.84)	
Examination –3					NS
Median	2.27	3.23	1.40	2.80	
Mean (± SD)	3.73 (± 4.62)	5.04 (± 9.66)	3.58 (± 5.57)	4.45 (± 8.22)	
Examination –4					NS
Median	2.19	3.90	2.72	2.33	
Mean (± SD)	2.81 (± 2.54)	4.26 (± 3.54)	3.95 (± 3.80)	3.14 (± 2.69)	

SD, standard deviation; statistical significance of difference between groups was tested using the Mann–Whitney test; NS, not significant.

(group 3), as shown in Table 3. A statistically significant decrease in hsCRP was observed among three consecutive blood sample examinations in groups 2 and 4, whereas it was only seen between examination 1 and examination 2 in groups 1 and 3, respectively (Figs 1 and 2).

In a stepwise logistic regression including diabetes, age, smoking, hypercholesterolaemia, advanced periodontal disease described with the own-created score and CAL values, only periodontal parameters turned out to be an independent predictor of hsCRP change (Table 4).

Discussion

The present study investigated the dynamics of hsCRP concentrations in patients ⩽60 years old with coexisting periodontal disease and acute coronary syndrome. We found that patients with less advanced periodontal disease have a significantly faster decline of inflammatory response compared with the group with more advanced periodontal disease, when hsCRP is monitored both short and long term after acute coronary syndrome.

It is hard to elucidate whether hsCRP decreases reflect mainly the natural course of inflammatory response after acute coronary syndromes or whether it is connected with the drugs introduced routinely in these patients in secondary prevention, which have important anti-inflammatory features (statins and ASA in all patients; in those patients 40 mg of simvastatin per day and 75 mg of ASA were given). Nevertheless, patients with more advanced periodontal disease displayed significantly longer diminution of the inflammatory response monitored with serum hsCRP concentrations in this study.

C-reactive protein remains an important marker of long-term prognosis after

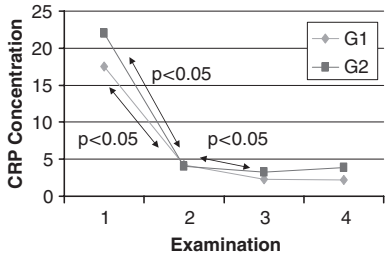

Fig. 1. Change in median serum concentrations of high-sensitivity C-reactive protein (hsCRP) over time in group 1 (G1) and group 2 (G2).

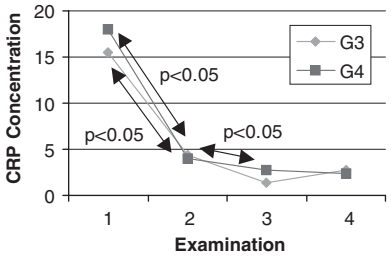

Fig. 2. Change in median serum concentrations of high-sensitivity C-reactive protein (hsCRP) over time in group 3 (G3) and group 4 (G4).

acute coronary syndromes since 90 years of the last century (Liuzzo et al. 1994). However, no detailed recommendations exist concerning the optimal time point to estimate hsCRP level after myocardial infarction or unstable angina. No broader studies have been devoted to the subject of hsCRP dynamics after acute coronary syndromes in patients with coexistent periodontal disease. In one recently published study of this kind, hsCRP concentrations were estimated in 40 acute myocardial infarction survivors of similar age as reported in our study. The mean serum hsCRP level in myocardial infarction patients was 40.2 mg/l (a similar value as seen in examination 1 in our study), which was much higher than in the control non-myocardial infarction, age-matched group (7.9 mg/l). In that study, half of the myocardial infarction patients were diagnosed with periodontal disease and had significantly higher hsCRP levels than myocardial infarction patients who did not have coexistent periodontal disease (50.7 *versus* 30.7 mg/l, $p < .001$) (Deliargyris et al. 2004). We were not able to conduct such an analysis, as all our patients were diagnosed with periodontal disease. In the Deliargyris study, periodontal disease remained an independent risk factor for elevated hsCRP after the multivariable linear regression model was introduced. We report similar findings in this study. We know that some other factor, like hypercholesterolaemia, might influence hsCRP dynamics after acute coronary syndromes; however, studies with more subjects are needed to explore this correlation (Filipiak et al. 2001).

Because of the multi-factorial nature of dental infection and acute coronary syndromes, confirming a causal association is difficult, and the published results are conflicting. However, meta-analysis of prospective and retrospective follow-up studies has shown that periodontal disease may increase the risk of cardiovascular disease by approximately 20% (Meurman et al. 2004). Periodontitis may add to the inflammatory burden of the individual and may result in increased levels of cardiovascular risk based on serum hsCRP concentrations, as was shown in one of the recent studies (D'Aiuto et al. 2004b). Similar conclusions were drawn from the Health Professional Follow-up Study in a sample consisting of 468 men (Joshipura et al. 2004).

The more severe the periodontal disease, estimated either in our own combined score or in the CAL parameter, the longer the diminution of the inflammatory response monitored with serum hsCRP concentrations (compare groups 2 *versus* 1 and group 4 *versus* 3). The conclusions from the classic ARIC (Atherosclerosis Risk in Communities) study, undertaken in a large >5500 cohort population, were quite different. In that study, the mean hsCRP level was 7.6 mg/l among people with extensive periodontal pockets (>30% of sites with pocket depth ⩾4 mm), approximately one-third greater than that for people with less extensive periodontal pockets (5.7 mg/l) (Slade et al. 2003). The larger the samples of patients, the more significant the differences in hsCRP among patients with different extents of periodontal disease. Thus, we were not able to report the significant difference in hsCRP levels among the studied groups in this paper.

Several limitations of this study have to be raised. Firstly, the main limitation is the small group of patients being studied. Secondly, the study included 50 patients with nine females and 41 males; thus there could be a substantial gender bias in the data presented. Thirdly, 78% of our patients were smokers with a daily average of 20 cigarettes. This was of some concern regarding the generalizability of our findings. Smoking has an important impact on dental plaque, although it probably does not influence the local concentrations of inflammatory markers (Erdemir et al. 2004). There are some data to postulate that it may even suppress inflammatory response (Apatzidou et al. 2005). Fourthly, other variables, e.g. hsCRP genotype or concomitant high serum titres to *Porphyromonas gingivalis*, may affect its concentrations in patients with periodontal disease (D'Aiuto et al. 2005a, Dye et al. 2005).

This study found no difference in hsCRP between periodontal disease groups but there was evidence of difference in diminution of hsCRP during coronary care. The clinical meaning of

Table 4. Independent predictors of hsCRP change between examination 1 and examination 2

Variable	Unadjusted odds/hazard ratio* (95% CI)	*p* value	Adjusted† odds/hazard ratio* (95% CI)	*p* value
Periodontal disease described with the own-created score >9 points	5.02 (3.17–8.13)	<0.0001	4.78 (2.35–9.74)	<0.0001
CAL values >3 mm	2.54 (1.10–4.28)	0.029	2.03 (0.87–6.0)	0.076

*Odds ratio for significant hsCRP concentration change.

†Adjusted for age, diabetes, smoking, gender, CK_{max}, $CKMB_{max}$, hypercholesterolemia

CI, confidence interval; CK_{max}, mean maximally marked values for creatinine kinase; $CKMB_{max}$, mean maximally marked values for creatinine kinase for MB fraction (values in international units).

this difference suggests the impact of periodontal disease' severity on the clinical course of acute coronary syndrome survivors. Our findings suggest once more that periodontal disease has an influence on inflammatory response due to acute coronary syndromes. These findings are consistent with our previous reports concerning tumour necrosis factor α and IL-1 concentrations in those patients (Czerniuk et al. 2004). Similar observations were also seen with fibrinogen concentrations, reported elsewhere (Czerniuk et al. 2002).

Conclusions

In conclusion, although no statistically significant difference in hsCRP was found between acute coronary syndrome patients with less and more severe periodontal disease, patients with less advanced periodontal disease, estimated on the basis of either the own combined score or CAL, have significantly longer diminution of the inflammatory response monitored with serum hsCRP concentrations. It might be suggested that in patients with more advanced periodontal disease – contrary to those with less advanced disease – it is too early to estimate hsCRP values 2 weeks after acute coronary syndromes as they do not usually come back to the background values.

Recently, it has been widely accepted that daily doses of statins (standard secondary prevention drugs for acute coronary syndrome patients) should be titrated not only on the basis of lipid profile but also on the basis of hsCRP target (Ridker et al. 2005). Therefore, cardiologists should be conscious of concomitant periodontal disease when titrating hsCRP-lowering therapy in acute coronary syndrome survivors. The design and findings of this study are too limited to make such a broad statement and recommendations. It should be noted, however, that an additional study in larger samples of patients might further investigate and confirm these findings.

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Clinical Relevance

Scientific rationale for the study: High-sensitivity C-reactive protein (hsCRP) serves as the marker for monitoring inflammatory response in acute coronary syndromes. The influence of coexistent periodontal diseases on hsCRP in this situation is not clearly defined.

Principal findings and practical implications: No statistically significant difference in hsCRP between acute coronary syndrome patients with less and more severe periodontal disease was found. However, during coronary care, there was some evidence that more advanced periodontal disease may implicate longer diminution in hsCRP as compared to less severe disease. Additional study in larger samples of patients will be needed to confirm these findings.