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Inflammatory response and postoperative kidney failure in patients with diabetes type 2 or impaired glucose tolerance undergoing heart valve surgery

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Abstract

Background: Diabetes type 2 (DM) or impaired glucose tolerance (IGT) are linked with a 3-fold increased risk of renal failure after heart valve surgery. The increase of proinflammatory cytokines is detected in patients with DM or IGT, moreover cardiac surgery promotes the proinflammatory response, which may be responsible for the development of postoperative kidney failure.

Aim: To assess the impact of perioperative pro- and antiinflammatory reaction after heart valve surgery and other clinical parameters on the risk of postoperative acute kidney injury in patients with DM or IGT.

Methods: Thirty patients with DM or IGT, without fibrate or statin treatment, with a mean LDL-cholesterol below 129 mg/dL, ejection fraction > 45%, in NYHA class II and III, referred for surgery due to acquired heart valve disease entered the study. Patients with acute or chronic inflammatory conditions, coronary artery disease or creatinine clearance below 50 mL/min were excluded. Serum creatinine, glycosylated hemoglobin, LDL-cholesterol and interleukin-10 as well as TNF-α were assessed before surgery. Interleukin-10 and TNF-α were also measured 4 hours after weaning from cardiopulmonary bypass. Moreover, serum creatinine and hemoglobin were measured 18 ± 2 hours after surgery. The relationship between postoperative creatinine clearance, its postoperative change and other parameters was assessed. These parameters included: age, weight and body mass index, pre- and postoperative serum level of TNF-α and interleukin-10, preoperative concentration of LDL-cholesterol and glycosylated hemoglobin, duration of cardiopulmonary bypass and postoperative hemoglobin.

Results: The significant postoperative decrease of creatinine clearance was noted in the study group. Eight (27%) patients developed postoperative kidney failure, of them 2 (6.5%) patients required hemodialysis. The level of TNF-α and interleukin-10 increased significantly postoperatively. A significant correlation between duration of cardiopulmonary bypass and postoperative decrease of creatinine clearance was noted ($R = 0.43$, $p = 0.02$). A non-significant trend towards correlation between preoperative TNF-α and postoperative decrease of creatinine clearance was observed ($R = -0.36$, $p = 0.05$).

Conclusions: Postoperative kidney failure with the incidence of 27% is a frequent finding in patients with DM or IGT operated due to acquired heart valve disease. The postoperative proinflammatory response is not involved in the development of this complication. The correlation between postoperative decrease of creatinine clearance and duration of cardiopulmonary bypass was noted. The trend toward the link between postoperative kidney failure and preoperative proinflammatory status was seen.

Key words: acquired heart valve disease, diabetes, postoperative kidney failure, cytokines

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INTRODUCTION

In the recent years, increasing prevalence of valvular heart disease (VHD) has been observed, particularly degenerative aortic valvular stenosis. Surgical treatment of the ageing population of VHD patients who suffer from numerous comorbidities, among which diabetes is one of the most prominent, represents a true challenge in contemporary cardiology and cardiac surgery. Patients with type 2 diabetes have more than triple the risk of kidney failure after surgery for VHD.

On the other hand, postoperative kidney failure requiring haemodialysis, carries 8 times higher mortality risk in the early postoperative period [1–4]. For that reason, in the group of diabetic patients treated surgically for VHD, in order to reduce morbidity and mortality, identification of mechanisms and risk factors of this complication seems very important.

It has been observed, that in diabetics and in patients with impaired glucose tolerance (IGT), activation of proinflammatory cytokines occurs, and it has been demonstrated that TNF-α plays an important role in the development of diabetic nephropathy [5–9]. Moreover, cardiac surgery itself promotes and enhances proinflammatory response [10]. This results from surgical injury, blood contacting the artificial surfaces during cardiopulmonary by-pass (CPB), endotoxaemia and ischaemia. It seems that excess proinflammatory response may be the reason of particularly high percentage of postoperative kidney failure in this patient group.

Current literature shows that there are numerous factors related to postoperative kidney failure in a general population of patients undergoing surgery. These include age, heart failure, preoperative kidney failure, infective endocarditis, reoperation, non-elective surgery, haemodilution, mean arterial blood pressure during CPB and prolonged CPB duration, as well as preoperative diuretic use and postoperative catecholamine administration [11, 12].

The aim of the study was to assess the impact of the pro and anti-inflammatory response in the pre- and postoperative periods, taking into account other clinical data, on the risk of postoperative kidney failure in the group of type 2 diabetics and patients with impaired glucose tolerance, undergoing surgery for VHD.

METHODS

Patients

In the years 2007–2008, 30 type 2 diabetes and IGT patients with ejection fraction > 45% and NYHA II/III heart failure, who were selected for elective valve replacement surgery for acquired VHD, were included in the study. Patients in whom mean LDL level did not exceed 129 mg/dL and who were not previously treated with either a statin or a fibrate were included. Patients with a chronic inflammatory condition, significant coronary artery stenosis, chronic kidney disease with creatinine clearance (ClCr) < 50 mL/min were not included. The ClCr was calculated based on Cockroft-Gault formula. Postoperative kidney injury was defined as a ≥ 50% increase of serum creatinine one day after surgery in comparison to their pre-operative values. All patients expressed their written consent for participation in the study. Local ethical committee approved the study protocol. Clinical characteristics of the study group are presented in Table 1.

Laboratory workup

In the preoperative period serum creatinine, glycated haemoglobin, LDL-cholesterol and interleukin-10 and TNF-α levels were measured. At exactly 4 hours post CPB cessation, interleukin-10 and TNF-α measurements were repeated. Moreover, within the first 24 hours following surgery (18 ± 2 h after CPB cessation on the average) serum creatinine and haemoglobin were measured. Time gap between the baseline and the postoperative creatinine measurements was 72 hours.

Statistical analysis

Correlation between postoperative ClCr and the difference between its pre- and postoperative values and numerous other parameters was studied. Among these parameters age, body mass, body height, pre- and postoperative TNF-α and interleukin-10, preoperative LDL level, preoperative glycated haemoglobin, CPB duration and first day postoperative haemoglobin were assessed.

The statistical assessment of Pearson's or Spearman's correlation coefficients was carried out with SAS package, depending on the normality of data distribution. A p value < 0.05 was

Table 1. Characteristics of the study group

Age [years]	67 ± 9
Male/female	14 (46%)/16 (54%)
Body mass index	29 ± 4
Aortic valve disease	22 (73%)
Mitral valve disease	6 (20%)
Mitral and aortic valve disease	2 (7%)
NYHA II heart failure	22 (73%)
NYHA III heart failure	8 (27%)
Arterial hypertension	24 (80%)
Type 2 diabetes	25 (83%)
Impaired glucose tolerance	5 (17%)
Diabetes treatment:	
Insulin	5 (20%)
Sulphonylurea derivatives	9 (36%)
Biguanides	4 (16%)
Glycated haemoglobin level [%]	6.2 ± 1.7
LDL concentration [mmol/L]	3 ± 0.8
Creatinine clearance [mL/min]	74 ± 18

Table 2. Comparison of pre- and postoperative creatinine clearance, pro- and antiinflammatory response level, cardiopulmonary bypass duration and hemoglobin concentration

	Preoperative	24 hours after surgery	p
Creatinine clearance [mL/min]	74 ± 18	56 ± 24	0.001
TNF-α concentration–median; range [pg/mL]	2.15 (0.6–4.97)	2.47 (0.8–10.6)	0.05
Interleukin-10 concentration–median; range [pg/mL]	0.78 (0.78–70)	50 (4.1–70)	0.001

considered significant. Parametric analysis was carried out with Student t-test for paired data because preoperative and postoperative data in individual patients were compared.

RESULTS

In the entire group, significant decrease in creatinine clearance during the first 24 hours postoperatively was observed. Acute postoperative kidney injury was noted in 8 (27%) patients. Of these, 4 (13%) patients were in stage 1, two patients were in stage 2 and three patients were in stage 3 of the acute kidney injury. Of these patients, 2 (6,5%) required haemodialysis in the postoperative period.

In the entire study group a significant increase of TNF-α and interleukin-10 was found in the postoperative period (Table 2). The CPB duration was 118 ± 37 min and haemoglobin concentration was 9.88 ± 1.2 mmol/L.

A significant positive correlation was found between CPB duration and the postoperative ClCr decrease (R = 0.43, p = 0.02). A trend fowards a correlation between postoperative ClCr decrease and pre-operative TNF-α (R = –0.36, p = 0.05; Fig. 1) was noted. No correlation was found neither between ClCr and ClCr pre- *vs* postoperative difference nor between other parameters analysed (Tables 3, 4). No statistical significance was found in relation to selected clinical and biochemical parameters in the group of patients in whom acute postoperative kidney injury occurred in comparison with the patients in whom this complication was not observed (Table 5).

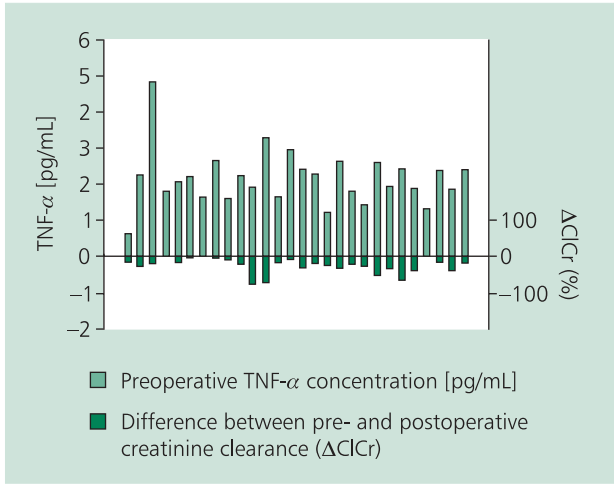

Figure 1. Individual values of preoperative TNF-α and change (in %) of creatinine clearance after surgery

DISCUSSION

To date, two pathomechanisms were identified as responsible for organ injury after cardiac surgery with CPB. These include systemic inflammatory reaction syndrome (SIRS) and ischaemia/reperfusion-related injury. The SIRS can be initiated by blood contacting synthetic surfaces of the biopumps used for CPB. This results in complement activation, platelet,

Table 3. Correlation between postoperative creatinine clearance and selected clinical and biochemical parameters

	Correlation coefficient	P value
Age	–0.19	0.29
Body mass	–0.16	0.37
Height	–0.19	0.29
Glycated haemoglobin	–0.28	0.26
Postoperative haemoglobin level	0.16	0.39
LDL-cholesterol concentration	0.19	0.31
Preoperative TNF-α concentration	–0.36	0.05
Postoperative TNF-α concentration	–0.16	0.39
Preoperative interleukin-10 concentration	–0.15	0.44
Postoperative interleukin-10 concentration	–0.1	0.61

Table 4. Correlation between cardiopulmonary bypass duration, difference between pre- and postoperative TNF-α and interleukin-10 concentration, and postoperative creatinine clearance change

	Correlation coefficient	P value
Cardiopulmonary bypass duration	0.43	0.02
Difference between pre- and postoperative TNF-α concentration	–0.05	0.77
Difference between pre- and postoperative and interleukin-10 concentration	–0.08	0.68

Table 5. Comparison of selected clinical and biochemical parameters of patients with postoperative kidney failure and patients without such complication

	Patients with acute postoperative kidney failure n = 22 (73%)	Patients without acute postoperative kidney failure n = 8 (27%)
Age [years]	72 ± 8	66 ± 9
Body mass index	29 ± 4	28 ± 4
LDL concentration [mmol/L]	3.04 ± 0.4	3.04 ± 0.9
Glycated haemoglobin [%]	6.4 ± 1.2	6.2 ± 1.9
Preoperative creatinine clearance [mL/min]	68 ± 17	76 ± 18
Creatinine clearance within 24 h postoperatively [mL/min]	29 ± 13	66 ± 20*
Preoperative TNF-α [pg/mL]	2.4 ± 0.5	2.2 ± 0.8
Postoperative TNF-α [pg/mL]	2.9 ± 0.9	3 ± 2
Preoperative interleukin-10 [pg/mL]	0.78 ± 0.01	4 ± 14
Postoperative interleukin-10 [pg/mL]	56 ± 23	40 ± 24
Cardiopulmonary bypass [min]	107 ± 39	122 ± 37
Haemoglobin concentration within 24 h postoperatively [g/dL]	9.56 ± 0.9	10 ± 1.2

*$p < 0.05$; NS

neutrophil, monocyte and macrophage activation and an increase in cytokine and leukotriene levels. Increase of endothelial permeability also occurs, followed by migration of the activated leukocytes to the extravasal space [13].

Organ damage secondary to ischemia is, in turn, related to perfusion alterations at the microcirculation level, resulting from vasoconstriction. It has been demonstrated that in type 2 diabetes and in impaired glucose tolerance, activation of proinflammatory reaction occurs. This results from protein glycation. The end-products of the process are capable of binding to specific receptors which can be found, among other cells, on the macrophages. This leads to their activation and subsequent cytokine release, including TNF-α, interleukin-1 and interleukin-6 among others [14, 15]. The increase of TNF-α level results in intraglomerular cellular infiltration and fibrin deposition. Moreover, vasoconstriction and secondary hipoperfusion are observed. These biological actions of proinflammatory cytokines result in glomerular filtration rate decrease.

Proinflammatory effects of TNF-α are partially neutralised by interleukin-10 which inhibits lymphocyte T and neutrophil activity [16–18]. This results in lowered production of the proinflammatory cytokines. This phenomenon was demonstrated in an animal model. It was shown that TNF-α plays a key role in postreperfusion kidney injury by stimulating cellular infiltration of renal parenchyma and activation of apoptosis. These destructive effects are counterbalanced by interleukin-10 [19]. Clinical manifestation of these processes is a net effect of the balance between pro- and anti-inflammatory cytokines.

Chello et al. [14] demonstrated adhesion activation of leukocytes within 24 hours post revascularisation with CPB in type 2 diabetic patients. The opinion according to which in type 2 diabetic or IGT patients with initially elevated cytokine levels, i.e. in patients particularly prone to excess proinflammatory response after cardiac surgery with use of CPB, this mechanism would play a key role in the development of postoperative kidney failure, seems warranted. On the other hand, in view of our results, only the high preoperative TNF-α level showed a trend for correlation with postoperative drop of ClCr. We did not demonstrate that excessive proinflammatory response after cardiac surgery with use of CPB contri-

buted to the development of this complication. The lack of protective effect of preoperative and postoperative interleukin-10 levels for the risk of postoperative kidney failure should also be noted.

Observations made by Morariu et al. [13] were similar. They found no effect of preoperative administration of dexamethasone on the incidence of postoperative kidney failure within 24 hours post surgical revascularisation, despite significant inhibition of proinflammatory response. It is interesting that these authors found a significant correlation between glomerular and tubular injury and higher glucose levels in patients without history of diabetes, in whom dexamethasone was administered. Taking this into consideration, it seems that high level of the proinflammatory response in type 2 diabetic patients prior to surgery through biological actions of TNF-α causes microstructural and perfusion changes in the kidney. Surgery-related hyperglycemia, resulting in impaired endothelial NO production, enhances the already present but clinically silent changes of renal parenchymal perfusion and morphology. This, in turn, promotes postoperative kidney failure, as described by Bohlen et al. [20] and Jin et al. [21]. Prolonged CPB duration, augmenting hyperglycemia and perfusion pressure alterations, was previously found to be an important risk factor of this complication, which was further confirmed by our study [11, 12].

Perioperative statin treatment, as the recently published retrospective observation by Fedoruk et al. [22] showed, significantly lowered the surgical risk in a general population of patients undergoing surgery for VHD. In their study group, 32% of the patients were type 2 diabetics. It seems that, by lowering preoperative proinflammatory reaction, statins can diminish postoperative kidney failure in patients with type 2 diabetes. However, this needs to be confirmed in a prospective study of this patient population.

Limitation of the study

The present study included small number of patients, which was due to very scrict inclusion and exclusion criteria. These criteria were applied to avoid confounding influence of other proinflammatory conditions or anti-inflammatory interventions such as statin and fibrate treatment which could have had disturbed the proinflammatory influence of the surgical intervention on the baseline proinflammatory activation in this patient group. Hence, to minimise the impact of other factors on the results, the number of the patients was restricted in order to increase group homogeneity. Only patients with no history of statin or fibrate treatment were included in the study. Current European Society of Cardiology guidelines allow for a short term refraining from implementation of drug therapy in diabetic patients with lower cardiovascular risk and LDL-cholesterol level of < 3.4 mmol/L (131 mg/dL). In order to avoid unavoidable drop-outs from the study group, patients with ClCr of $< 50\%$ were excluded. In univariate analysis a relationship between CPB duration and postoperative acute kidney injury was found. On the other hand, the preoperative TNF-α concentration showed a trend for correlation, so multivariable analysis was not performed.

CONCLUSIONS

Postoperative acute kidney failure in type 2 diabetics and in IGT patients undergoing cardiac surgery for acquired VHD is a common clinical finding and can be seem in 27% of these patients. There is a relationship between CPB duration and the risk of acute kidney injury. The preoperative proinflammatory status, measured by TNF-α concentration, maybe related to postoperative renal failure. The inflammatory response resulting from surgical intervention with the use of CPB doesn't seem play a role in the development of renal failure.

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Wpływ reakcji zapalnej na rozwój niewydolności nerek u osób z cukrzycą typu 2 lub upośledzoną tolerancją glukozy po leczeniu kardiochirurgicznym z powodu nabytych wad zastawkowych serca

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Streszczenie

Wstęp: Chorzy ze zwężeniem zastawki aortalnej i współistniejącą cukrzycą typu 2 lub nietolerancją glukozy są narażeni na 3-krotnie większe ryzyko wystąpienia niewydolności nerek po chirurgicznej operacji zastawkowej. Niewydolność nerek wymagająca zastosowania hemodializy jest związana z 8-krotnym wzrostem pooperacyjnej śmiertelności. Zaobserwowano, że u chorych z nieprawidłową gospodarką węglowodanową dochodzi do aktywacji produkcji cytokin prozapalnych; ponadto sama operacja kardiochirurgiczna stymuluje reakcję prozapalną. Wydaje się, że nasilona reakcja prozapalna może stanowić przyczynę szczególnie wysokiego odsetka występowania pooperacyjnej niewydolności nerek w tej grupie chorych.

Cel: Celem pracy była ocena wpływu reakcji pro- i przeciwzapalnej w okresie okołooperacyjnym na ryzyko wystąpienia pooperacyjnego ostrego uszkodzenia nerek u pacjentów z cukrzycą typu 2 lub nietolerancją glukozy poddanych leczeniu kardiochirurgicznemu z powodu nabytych wad zastawkowych serca.

Metody: Badaniem objęto 30 chorych z nietolerancją glukozy lub cukrzycą typu 2, z frakcją wyrzutową > 45%, niewydolnością serca w II i III klasie według NYHA, zakwalifikowanych do planowej operacji z powodu nabytej wady zastawkowej serca. Do badania włączono pacjentów ze średnim stężeniem cholesterolu frakcji LDL nieprzekraczającym 129 mg/dl, którzy nie otrzymywali statyn ani fibratów. Wyłączono osoby z ostrym lub przewlekłym stanem zapalnym, z istotnymi zwężeniami w naczyniach wieńcowych, z klirensem kreatyniny < 50 ml/min. Przed operacją oznaczono stężenie kreatyniny, hemoglobiny glikowanej, cholesterolu frakcji LDL, interleukiny-10 i TNF-α. Po 4 godzinach od zakończenia krążenia pozaustrojowego oznaczono ponownie stężenia interleukiny-10 i TNF-α. Po 18 ± 2 godzinach od zakończenia krążenia pozaustrojowego oznaczono stężenie kreatyniny i hemoglobiny. Pooperacyjną niewydolność nerek zdefiniowano jako 50-procentowy wzrost kreatyniny w 1. dobie po leczeniu kardiochirurgicznym w stosunku do stężenia wyjściowego. Przeanalizowano związek między pooperacyjnym klirensem kreatyniny oraz różnicą między jego wartościami w okresie przed- i pooperacyjnym a parametrami, takimi jak: wiek, masa ciała, wskaźnik masy ciała, przed- i pooperacyjne stężenie TNF-α oraz interleukiny-10, przedoperacyjne stężenie cholesterolu frakcji LDL, hemoglobiny glikowanej, czas trwania krążenia pozaustrojowego i stężenie hemoglobiny w 1. dobie po operacji.

Wyniki: Zaobserwowano znamienny spadek klirensu kreatyniny w 1. dobie po leczeniu kardiochirurgicznym. Odnotowano wystąpienie pooperacyjnej niewydolności nerek u 8 (27%) chorych. Spośród tej grupy 2 (6,5%) osób wymagało stosowania hemodializy. W całej badanej grupie stwierdzono znamienny statystycznie wzrost stężenia TNF-α oraz interleukiny-10 w okresie pooperacyjnym. Wykazano znamienną statystycznie dodatnią korelację między czasem trwania krążenia pozaustrojowego a pooperacyjnym spadkiem klirensu kreatyniny ($R = 0.43$, $p = 0.02$). Zaobserwowano trend w powiązaniu pooperacyjnego spadku klirensu kreatyniny z przedoperacyjnym stężeniem TNF-α ($R = –0,36$; $p = 0,05$).

Wnioski: Pooperacyjna niewydolność nerek u pacjentów z cukrzycą typu 2 lub nietolerancją glukozy po leczeniu kardiochirurgicznym z powodu nabytej wady zastawkowej serca jest częstym zjawiskiem klinicznym i dotyczy 27% operowanych. Odpowiedź zapalna po operacji w krążeniu pozaustrojowym nie odgrywa roli w powstawaniu tego zjawiska. Stwierdzono korelację między czasem trwania krążenia pozaustrojowego a ryzykiem rozwoju pooperacyjnej niewydolności nerek. Jednocześnie zaobserwowano trend w kierunku związku między przedoperacyjnym stanem prozapalnym a pooperacyjnym spadkiem klirensu kreatyniny w tej grupie chorych.

Słowa kluczowe: nabyte wady zastawkowe, cukrzyca, pooperacyjna niewydolność nerek, cytokiny

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