Prevention of contrast-induced nephropathy in patients undergoing percutaneous coronary interventions in everyday clinical practice

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Abstract

Introduction: Contrast nephropathy is a potentially serious complication of PCI, particularly in patients with chronic kidney disease – CKD.
Material and methods: Assessment of the effects of preventive measures on the prevalence of contrast-induced nephropathy in 529 consecutive patients with coronary artery disease undergoing percutaneous coronary interventions. In all the patients 24 h before PCI all nephrotoxic drugs (NSAIDs, diuretics, biguanidine derivatives in diabetic patients) were withdrawn and doses of ACEi were either withdrawn or reduced.
Results: Only 27.4-47.7% of patients with normal serum creatinine at admission had eGFR over 90 ml/min according to Cockcroft-Gault and MDRD formulae. In 39 patients we observed a rise in serum creatinine of more than 25% relative to baseline. More frequently they were females, hypertensive, PCI time was longer and contrast volume was higher. In 5 pts (0.9%) acute renal failure was observed (rise in creatinine more than 0.5 mg/dl relative to baseline). These patients received more contrast agent ($p<0.01$) and PCI was longer ($p<0.01$). In 21 patients we observed a significant fall in serum creatinine. They received significantly less contrast and low-osmolar contrast, and PCI was shorter. Estimated GFR and serum creatinine after PCI were not influenced by type of hydration (0.9% NaCl or 8.4% $NaHCO_3$).
Conclusions: Awareness of contrast nephropathy should be highlighted in primary care physician practice since some patients (with decrease in serum creatinine after PCI) were not well prepared for the procedure (mainly dehydrated). The prevalence of impaired renal function is relatively high in patients undergoing elective coronarography despite normal serum creatinine. The risk of contrast nephropathy is enhanced in these patients; therefore GRF should be estimated before coronarography. It is an inexpensive, reliable and widely available method. The most important prophylactic measure is adequate hydration, but low-osmolar contrast, contrast volume and duration of PCI should also be taken into account.

Key words: chronic kidney disease, contrast-induced nephropathy, percutaneous coronary interventions.

Introduction

The administration of radiocontrast media can lead to a usually reversible form of acute renal failure that begins soon after the contrast is administered [1-5]. In most cases, there are no permanent sequelae, but there is some

evidence that its development is associated with adverse outcomes. The reported incidence of radiocontrast-induced nephropathy varies widely, ranging from zero to over 50 percent. This variability results from differences in the presence or absence of risk factors (primarily renal insufficiency), definition of contrast-induced nephropathy, amount and type of agent administered, prospective or retrospective determination of incidence, the exact radiologic procedure, and whether other causes of acute renal failure unrelated to contrast media were excluded. Since interventional cardiologists are being asked more frequently to perform percutanous coronary intervention (PCI) on increasing numbers of patients, contrast nephropathy is a more frequent and potentially serious complication of PCI [6]. Peak creatinine levels typically occur 3 to 5 days after contrast administration and return to baseline (or a new baseline) in 1 to 3 weeks [7], when patients are discharged from the hospital. Unfortunately, creatinine is an unreliable indicator during acute changes in kidney function [8]. First, using serum creatinine to estimate true renal function has well-recognized inaccuracies and limitations [9]. A marked reduction in GFR can be present before it is reflected in a rise in serum creatinine (up to 50% of kidney function has already been lost before creatinine might change). Second, serum creatinine does not reflect kidney function during acute changes until a steady state have been reached, which could take several days as in the case of contrast nephropathy. There is no specific treatment once contrast-induced acute renal failure develops, and management must be as for any cause of acute tubular necrosis, with the focus on maintaining fluid and electrolyte balance. The best treatment of contrast-induced renal failure is prevention.

Taking all these data into consideration the aim of the study was to assess the effects of preventive measures on the prevalence of contrast-induced nephropathy in patients with coronary artery disease undergoing percutaneous coronary interventions.

Material and methods

A cohort of 529 (350 M, 179 F) consecutive patients undergoing PCI (coronary angiography and/or coronary angioplasty, males were significantly more prevalent than females, 66.4% vs 33.6 %, $p<0.001$) due to ischaemic heart disease in the Department of Invasive Cardiology was studied. Normal serum creatinine was found in 491 patients (less than 1.5 mg/dL in males and less than 1.2 mg/dL in females). Clinical data were obtained from hospital clinical records. We evaluated the patients' basal clinical variables: demographic, risk factors, family history in regard to coronary artery disease, serum creatinine on admission and 24 after procedure at discharge, lipids, glucose, blood pressure, medications. Serum creatinine was measured by the standard laboratory method (Jaffe) in the central laboratory. We assessed kidney function according to the simplified MDRD [10] and Cockcroft-Gault [11] formulae before the procedure and 24 hours after the procedure at discharge. Type of PCI, time of the procedure, volume and kind of contrast agent were taken from the procedure files. According to DOQI guidelines stages of chronic kidney diseases were as follows [12]:

- Stage 1 – Kidney damage (kidney damage is defined as 'pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies') with normal or increased eGFR (>90 ml/min),
- Stage 2 – mild decrease in eGFR (60-89 ml/min),
- Stage 3 – moderate decrease in eGFR (30-59 ml/min),
- Stage 4 – severe decrease in eGFR (15-29 ml/min),
- Stage 5 – kidney failure (<15 ml/min or dialysis).

Neither physicians taking care of the patients at the department nor the physician at the catheterisation laboratory were aware of the aim of the study. All the patients admitted to the department of invasive cardiology were recommended to drink about 2 litres of still water within 24 hours periprocedurally. High-risk patients (with established chronic kidney disease, diabetes, hypovolaemic, hypotensive, with cardiogenic shock, and intraaortic balloon counterpulsation) were administered intravenous hydration as prescribed by the physician. Isotonic (0.9 percent) saline at a rate of 3 mL/kg per hour for 1 hour before, then 1 mL/kg per hour during and for 6 hours after the angiogram or three 50 mL ampules of 1 meq/mL sodium bicarbonate to 850 mL of 5% dextrose in water administered as a bolus of 3 mL/kg per hour for one hour prior to contrast, followed by a 1 mL/kg per hour infusion for six hours afterwards. Metformin, diuretics, non-steroidal anti-inflammatory drugs or other nephrotoxic agents were withdrawn 24 hours before the procedure. ACE inhibitors were either withdrawn (when blood pressure permitted) or reduced by 50% 24 hours before the procedure.

Data given were analysed using Statistica 6.0 computer software. Data were expressed as means ±SD or %. The examination of the distribution normality of variables was done using W-Shapiro-Wilk test. Comparisons between groups were done by t-test, Wilcoxon rank sum test or χ^2 test as appropriate. Values of $p< 0.05$ were taken as statistically significant.

Results

Clinical and biochemical characteristics of patients with contrast-induced nephropathy (CIN) and without CIN are presented in Table I. When contrast nephropathy was defined as an increase in serum creatinine by >25% of the baseline level 24 hours after contrast exposure, the prevalence of CIN was 7.9% (39 patients). These patients were

Table I. Clinical and biochemical characteristics of patients with contrast-induced nephropathy (CIN, defined as a rise in serum creatinine by 25% of the baseline value) and without CIN

Patients with normal serum creatinine (n=491)	With CIN (N=39)	Without CIN (N=452)
Age (years)	61.51±9.25	61.10±9.66 NS
Body mass index BMI (kg/m^2)	28.67±3.41	28.59±4.15 NS
Hypertension (%)	89.7	76.3*
Low-osmolar contrast (%)	30.8	36.7 NS
Haemoglobin – Hb- (g/L)	14.35±? 1.01	14.30±1.37 NS
Haematocrit – Ht- (%)	42.17±2.86	41.96±3.88 NS
creatinine (mg/dL) before PCI	0.84±0.19	1.00±0.20**
24 hours after PCI	1.18±0.37	0.98±0.23**
GFR (MDRD, ml/min) before PCI	92.83±22.05	77.95±18.84**
24 hours after PCI	65.86±18.78	79.69±18.13**
GFR (Cockcroft-Gault, ml/min) before PCI	103.37±1.12	89.47±28.89**
24 hours after PCI	76.44±23.45	91.14±28.12**
Duration of PCI (min)	51.97±29.04	47.24±20.52 NS
Contrast volume (ml)	151.89±84.62	142.46±68.39 NS
$NaHCO_3$ (%)	0	4.4 NS
NaCl 0.9% (%)	10.3	10.6 NS
Serum fasting glucose (mg/dL)	104.37±29.14	111.83±39.48 NS
diabetes (%)	30.8	25.9 NS
EF (%)	50.17±12.6	50.40±11.44 NS

**p<0.05, **p<0.001 CIN vs non-CIN*
NS – not significant

Table II. Estimated GRF according to Cockcroft-Gault and MDRD in patients with and without CIN (defined as a rise in serum creatinine by 25% of the baseline value)

e GFR- (ml/min)	Number	Before PCI	24 hours after PCI
Cockcroft-Gault in patients with CIN	N=39	103.37 (±27.33)	76.44 (±23.45)*
MDRD in patients with CIN		92.83 (±22.05)	65.86 (±18.78)*
Cockcroft-Gault in patients without CIN	N=452	89.47 (±28.89)#	91.14 (±28.12)*
MDRD in patients without CIN		77.95 (±18.84)#	79.69 (±18.13)*

**p<0.001 GFR before PCI vs after PCI*
#p<0.001 GFR before PCI in patients with CIN vs patients without CIN

significantly more often hypertensive (p<0.05) with similar serum creatinine, haemoglobin, BMI and higher eGFR than patients without CIN (Table II). In Table III eGFR at admission according to both Cockcroft-Gault and MDRD formulae is presented. It is noteworthy that only 27.4-47.7% of patients with normal serum creatinine at admission had eGFR over 90 ml/min. In Table IV type of contrast agent administered in relation to stage of CKD is given.

Effects of hydration

Among 39 patients who develop CIN only 4 patients (10.3%) were given intravenous hydration periprocedurally. A similar percentage of patients without CIN were administered intravenous hydration. Intravenous hydration was significantly more often given to patients with elevated serum creatinine (44.7%, 17 out of 38 with elevated serum creatinine) when compared to patients with normal serum creatinine (7.2%, 35 out of 491, p<0.001). Similarly, sodium bicarbonate was significantly more often given to patients with elevated serum creatinine (28.6%) when compared to patients with normal serum creatinine (2.7%, p<0.001).

In none of the patients administered sodium bicarbonate did CIN develop.

Effects of risk factors on CIN

Chronic renal failure (defined at admission as having elevated serum creatinine over 1.5 mg/dl in males and 1.2 mg/dl in females) was found in 38 patients, more frequently in males (11%) than in females (2.6%, p<0.01). In this group of patients elevated serum creatinine before PCI (1.55±0.19 mg/dL) decreased significantly after the procedure (1.35±0.15 mg/dL, p<0.001). Namely, in 21 patients elevated serum creatinine before PCI was lower after the procedure. These patients with elevated serum creatinine before PCI more frequently were given low-osmolar contrast (p<0.001), PCI duration was shorter (39±17 min vs 48±21 min, p<0.01) and contrast volume was diminished (119±63 vs 145±79 ml, p<0.05).

Prevalence of diabetes did not differ between groups with and without CIN, whereas hypertension was more prevalent in patients who developed CIN.

When contrast nephropathy was defined as an increase in serum creatinine by 0.5 mg/dl of the baseline level 24 hours after contrast exposure, prevalence of CIN was 0.9% (5 patients). Mean serum creatinine before PCI was 1.04±0.16 mg/dl and 1.9±0.34 mg/dl 24 hours afterwards. Estimated GRF according to MDRD was 70.49±21.53 ml/min before PCI, and 36.15±18.45 ml/min 24 hours afterwards, whereas eGFR according to Cockcroft-Gault formula was 79.38±20.21 ml/min before PCI and 43.61±19.54 ml/min 24 hours after PCI. These patients were of similar age, BMI with similar haemoglobin, haematocrit to patients without CIN. In patients who develop CIN defined as a rise in serum creatinine over 0.5 mg/dl of the baseline, duration of the procedure was longer (91.0±30.8 min vs 47.1±19.7 min, p<0.05) and contrast volume higher (230±92 ml vs 142±65 ml, p<0.01).

Patients with a fall in eGFR after PCI (but not with CIN)

In 108 patients (22.1%) a fall in e GFR by 10 ml/min was observed, and in 45 patients (9.2%) eGFR decreased by at least 20 ml/min. In patients with eGFR fall by 10 ml/min intravenous hydration was prescribed less frequently (p<0.01); none of the patients received sodium bicarbonate. Diabetes was diagnosed in 29.6% and prevalence was not different from patients without fall in eGFR (25.6%). However, prevalence of hypertension in patients with a fall in eGFR almost reached statistical significance (83.3% vs 75.5%, p=0.08) when compared to patients without an observed fall in eGRF after PCI. Low-osmolar contrast was administered less frequently to patients with a fall in eGFR (p<0.01), contrast volume was significantly higher (p<0.05) and duration of PCI was significantly longer (p<0.05). In patients with a fall in eGFR by more than 20 ml/min, diabetes was more prevalent (40% vs 25.15%, p<0.05), but not hypertension (86.7% vs 76.3%, NS). In addition, in these patients a rise in serum creatinine was observed 24 hours after PCI (p<0.01). There were no statistically significant differences in gender, BMI, haemoglobin or haematocrit, between patients with a fall in eGFR by 20 ml/min and patients without this fall. Differences in contrast volume and PCI duration did not reach statistical significance (p=0.07 and p=0.10, respectively).

Table III. Estimated GFR in patients with normal serum creatinine

a – according to MDRD formula at admission in studied patients

eGFR	N=491	Gender (female/male)	
>90 ml/min	47.7%	31%	56.2%
89-60 ml/min	38.5%	45%	35.2%
59-30 ml/min	13.2%	22%	8.6%
29-15 ml/min	0.6%	1.8%	–
<15 ml/min	–	–	–

b – according to Cockcroft-Gault formula at admission in studied patients

eGFR	N=491	Gender (female/male)	
>90 ml/min	27.5%	14%	34.4%
89-60 ml/min	57.2%	62%	54.7%
59-30 ml/min	14.6%	22.1%	10.9%
29-15 ml/min	0.64%	1.2%	–
<15 ml/min	0.2%	0.6%	–

Table IV. Type of contrast agent administered in relation to stage of CKD, i.e. eGFR according to MDRD

Low-osmolar N=190 (38.7%)	High-osmolar N=301 (61.3%)
I stage CKD – 23.7%	29.9%
II stage CKD – 52.5%	60.2%
III stage CKD – 22.2%	9.9%
IV stage CKD – 1.0%	0
V stage CKD – 0.5%	0

Patients with a fall in serum creatinine 24 hours after the procedure

In 17 patients a fall in serum creatinine (more frequently in males, 68.1%, p<0.05) was observed. In these patients intravenous hydration was prescribed more frequently (35.35% vs 9.7%, p<0.001) with sodium bicarbonate (11.8% vs 3.8%, p<0.01). They received less contrast agent (p<0.05) and duration of the procedure was shorter (p<0.05).

Discussion

It is increasingly appreciated that chronic renal dysfunction alone is an independent factor for the development of coronary artery disease. In addition,

patients with CKD have enhanced mortality after an acute coronary syndrome and after PCI with or without stenting. Studies have shown that a GFR (glomerular filtration rate) less than 60 ml/min is a harbinger of premature cardiovascular death [13]. Since interventional cardiologists are being asked more frequently to perform PCI on increasing numbers of patients with significant co-morbidities such as CKD and/or diabetes, we should think about renal function in our cardiological patients as well as about contrast-induced nephropathy as a potentially serious complication of PCI. There is no specific treatment once contrast-induced acute renal failure develops, and management must be as for any cause of acute tubular necrosis, with the focus on maintaining fluid and electrolyte balance. The best treatment of contrast-induced renal failure is prevention: the use of lower doses of contrast [3, 14-16] and avoidance of repetitive studies that are closely spaced (within 48 to 72 hours), avoidance of volume depletion or nonsteroidal antiinflammatory drugs, both of which can increase renal vasoconstriction, the administration of intravenous saline or possibly sodium bicarbonate and the use of low or iso-osmolar nonionic contrast agents. Patients with ischaemic heart disease often exhibit some degree of renal dysfunction due to concomitant diabetes, hypertension or congestive heart failure, despite normal serum creatinine. In our study, up to 70% of patients with normal serum creatinine undergoing PCI exhibited impaired renal function, i.e. GFR below 90 ml/min. In up to 15% GFR was below 60 ml/min, which is considered significant impairment of renal function (i.e. CKD 3 stage or lower). The prevalence of CKD is high and similar to our previously published papers [17, 18].

In our study the prevalence of CIN was 7.9% and did not differ from other reports [19-21]. In patients who developed CIN, defined as an increase in serum creatinine over 25% of the baseline value, duration of PCI was significantly longer than in patients without CIN. When CIN was defined as a rise in serum creatinine over 0.5 mg/dl of the baseline value, the important risk factor of developing CIN was not only duration of PCI but also the contrast volume. Moreover, in some patients we observed a fall in serum creatinine 24 hours after PCI. These patients were probably with preexisting CKD, and they were inadequately hydrated before the procedure, despite obligatory hydration (either oral or intravenous) before PCI. In several studies the problem of hydration was evaluated; however, the optimal hydration solution (isotonic saline, half-normal saline, or isotonic sodium bicarbonate) for prevention of contrast nephropathy is unclear. Hydration with isotonic saline may be superior to one-half normal saline. This was shown in a study of 1620 patients provided with either isotonic or one-half isotonic saline at a rate of 1 mL/kg starting on the morning of the procedure [22].

Since alkalinization may protect against free radical injury, the possibility that sodium bicarbonate may be superior to isotonic saline was examined in a prospective trial of 119 patients with and without renal insufficiency who were randomly assigned to receive infusions of either isotonic (154 meq/L) sodium chloride or bicarbonate [23]. These results suggest that the infusion of isotonic sodium bicarbonate may be beneficial [24].

In our study, high-risk patients (elevated serum creatinine at admission) received more frequently low-osmolar contrast, lower contrast volume, and were more frequently intravenously hydrated either with isotonic saline or with sodium bicarbonate. In this group a fall in serum creatinine after PCI was observed, suggesting inadequate ambulatory care. The safety and efficacy of oral hydration for the prevention of contrast nephropathy remains uncertain as presented in two conflicting trials [25, 26]. In one study, 36 patients with plasma creatinine concentration ≥1.4 mg/dL who required PCI were randomly assigned to an outpatient regimen or an inpatient regimen [25]. There was no difference in the maximal change in plasma creatinine concentration between the two groups. In a second trial, 53 patients were randomly assigned to either unrestricted oral fluids or to normal saline at 1 mL/kg per hour for 24 hours beginning 12 hours prior to the scheduled catheterization [26]. Acute renal failure was significantly more common with oral hydration (35% versus 4%). In a recent study of Mueller et al. [21] the combination of intravenous and oral volume supplementation resulted in a very low incidence of CIN following PCI in 425 consecutive patients with mean GFR of 89 ml/min.

We also focused on the effect of the type of contrast agent and prevalence of CIN in our patients. Low-osmolar contrast was mainly given to patients with impaired renal function (stage 3 and 4 CKD). Most studies have evaluated the relative effectiveness of nonionic low osmolar agents versus ionic hyperosmolar agents. The ionic low-osmolar agent also appears to be associated with a lower risk of contrast nephropathy compared to ionic hyperosmolar agents [27].

Conclusions

Our findings may have important implications for the clinical management of patients undergoing PCI. The "window of opportunity" is narrow in contrast nephropathy and the time to introduce proper treatment after initiating insult is limited, particularly when patients are discharged within 24 hours after PCI. Optimal therapy to prevent contrast-induced acute renal failure remains uncertain. Patients with near-normal renal function are at little risk and few precautions are necessary other than avoidance of volume depletion. Use of lower doses of contrast and low osmolar or iso-osmolar nonionic contrast, avoidance of repetitive, closely spaced studies (e.g. <48 hours apart), and avoidance of volume depletion and nonsteroidal

antiinflammatory drugs are simple measures to prevent CIN. If there are no contraindications to volume expansion, isotonic intravenous fluids prior to and continued for several hours after contrast administration should be recommended.

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