Kardiologia Polska 2015; 73, 3: 233–234; DOI: 10.5603/KP.2015.0050 ISSN 0022–9032

Early renal dysfunction after iodine contrast administration: new insight into the actual problem

Wczesne pokontrastowe pogorszenie czynności nerek: nowe spojrzenie na wciąż aktualny problem kliniczny

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Early renal dysfunction after scheduled coronarography or percutaneous transluminal coronary angioplasty is a clinically relevant issue. The term "contrast-induced acute kidney injury" was established by the Kidney Disease Improving Global Outcome (KDIGO) acute kidney injury (AKI) Guideline Work Group in the interdisciplinary guidelines concerning AKI published in 2012 [1]. According to KDIGO guidelines, the first stage of CI-AKI is reflected by an increase in serum creatinine (SCr) concentration by ≥ 0.3 mg/dL (26.5 μmol/L) or a 50% increase from baseline value, as well as urine output < 0.5 mL/kg/h for more than 6 h [1]. Even if CI-AKI diagnosis is based on the "sharpest criteria" described in the literature, there is still a group of patients in whom the deterioration of renal function after angiography is noticeable but not advanced enough to be interpreted as nephropathy [2, 3]. The hospitalisation time of patients undergoing scheduled coronarography or percutaneous transluminal coronary angioplasty is usually short and thereby insufficient for the renal function impairment to progress to a sufficient extent to meet current criteria of CI-AKI.

As a result of periprocedural intravenous hydration the creatinine level decreases, hence one should be concerned about the increase or even lack of changes in creatinine concentration (decrease or stability of glomerular filtration rate [GFR] alternatively) a few hours after administration of contrast medium. An early increase in serum creatinine level in particular patients can be associated not only with epithelium necrosis, but may also possibly indicate already apparent functional renal impairment, which will probably not meet the existing clinical criteria of CI-AKI.

Early estimation of SCr or GFR can be very effective in CI-AKI detection, as demonstrated by Ribichini et al. [4], which is one of just several studies describing such dependency. Ribichini et al. [4] showed that an increase in SCr after 12 h after contrast media administration is the most sensitive and specific risk factor for regular CI-AKI. The analysis was based on a prospective observational study performed with patients undergoing coronary angiography and angioplasty. The study included patients suffering from acute coronary syndrome, which may account for the large amount of patients (18% of the whole population) with in-hospital diagnosis of CI-AKI (according to the definition: increase in SCr by 25% from baseline level after 48 h). Moreover, 8% of the studied population was recognised as having contrast-induced kidney impairment 30 days after coronarography/coronaroplasty (so-called late nephropathy) [4].

An increase in creatinine level by 5% and 10% from base level was connected, respectively, with 70% and 80% of sensitivity and 70% and 85% of specificity for both CI-AKI during hospitalisation and persistent renal failure 30 days after the procedure [4].

In our own studies, we have shown that 12–18 h after coronarography/coronaroplasty with periprocedural hydration the majority of patients display a statistically significant increase in GFR as well as a decrease in SCr level. Nevertheless, when looking at absolute values, the increase in creatinine level, decrease in creatinine clearance, and variously defined reduction in GFR involved up to 28% of our patients [5]. Insufficient hydration, dehydration, or extensive loss of blood were excluded as possible reasons for such outcomes. Patients in whom contrast-induced early renal dysfunction was observed were characterised by lower base concentrations of creatinine and urea, as well as higher haemoglobin concentration and haematocrit. This warrants the conclusion that as regards

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factors commonly recognised as unfavourable, our group of patients with post-angiographic kidney function impairment was less burdened and generally healthier. Such dependence has not been described in literature so far. For the present, it cannot be excluded that that hospitalisation itself (even without implementing any medical procedures) can be connected with a statistically significant increase in creatinine level, which was described by Newhouse et al. [6], who pointed to the overactivation of patients sympathetic nervous system. As regards explanations of our findings, one should mention that patients with worse kidney function at baseline may be less prone to further toxic impairment in comparison to patients who have never been exposed to nephrotoxic agents. This is similar to ischaemic preconditioning in the heart, but it should be stressed that such a "habituation" phenomenon has been also already described in a renal context [4, 6, 7].

To sum up, contrast-induced kidney dysfunction should not be interpreted according to the criteria adopted for acute renal injury of other aetiology [1]. The early renal dysfunction can be observed just a few hours after implementation of iodine contrast and occurs regardless of prophylactic hydration. At the same time, it seems that patients who are at high risk of early renal dysfunction are those who are characterised by normal renal function at baseline. In the case of this group of patients, we should be particularly alert to any detected lack of SCr reduction in control laboratory tests performed after diagnostic procedures, including use of contrast medium; the lack of SCr reduction indicates the possibility of developing significant renal impairment and should result in adequate patient management that should then be oriented towards kidney protection therapy.

Acknowledgements

Special thanks to Simon Nowak for language service.

Conflict of interest: *none declared*

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