Contrast induced acute kidney injury – is it a real problem these days?

Musiałowski P [1 A,B,C,D] , Musiałowska D [2 B,C,D*], Gościk E [1 E,F], Małyszko J [2 A,E,F]

1. Department of Paediatric Radiology, The Medical University of Bialystok Children's Clinical Hospital, Bialystok, Poland
2. 2nd Department of Nephrology and Hypertension with Dialysis Unit, Medical University of Bialystok, Poland

---

A - Conception and study design, B - Data collection, C –Data analysis, D - Writing the paper,
E – Review article, F - Approval of the final version of the article

---

ABSTRACT

Acute kidney injury (AKI) is proven risk factor associated with higher mortality and morbidity among hospitalized patients. The widespread use contrast media opens the discussion about the acute kidney injury as a result of used contrast - contrast induced nephropathy (CIN). CIN is defined as an acute, generally reversible decline in renal function that occurs 48-72 hours after intravascular injection of contrast medium. Pre-existing renal insufficiency is characterised independent risk factor for occurrence of CI-AKI, other factors, such as diabetes mellitus, hypertension, advanced age or hemodynamic instability increase the risk of AKI, but are not characterized as independent risk factors. Published new large retrospective single-center studies presented equal risk of AKI among patients receiving contrast enhanced computer tomography if compared to unenhanced computer tomography, based on serum creatinine levels. In our review we would like to present a persisting the problem of CIN after intravenous (iv) as well intra-arterial contrast media administration.

Key words: contrast media, contrast induced nephropathy, acute kidney injury

---

***Corresponding author:**
Dominika Musialowska
2nd Department of Nephrology and Hypertension with Dialysis Unit
The Medical University of Bialystok Clinical Hospital
M. Sklodowskiej-Curie 24a Street
15-276 Bialystok, Poland
e-mail: dmaciorkowska@poczta.onet.pl

Received: 28.04.2016
Accepted: 16.05.2016
Progress in Health Sciences
Vol. 6(1) 2016 pp 177-182

INTRODUCTION

Acute kidney injury (AKI) is proven risk factor associated with higher mortality and morbidity among hospitalized patients [1-4]. The aging population, growing percentage of people with diabetes mellitus and hypertension make the problem of kidney injury current and important. The one of the most important risk factors for acute kidney injury is chronic kidney disease (CKD), which estimated prevalence in Polish population is 5,8% [5-7]. Available data shows that about 16% of patients undergoing computer tomography (CT) suffer from CKD [6,8-9]. The increasing number of patients diagnosed with the use of CT, as also magnetic resonance and treated with percutaneous coronary interventions require the use of contrast media. The widespread use contrast media [10] opens the discussion about the acute kidney injury as a result of used contrast - contrast induced nephropathy (CIN). The first case report of CIN was observed in 1950's [11]. Nowadays contrast enhanced computer tomography is a standard diagnostic procedure. The incidence of CIN after contrast enhanced computer tomography is estimated at 2-12% depended on the analysed studies [12-19]. In our review we would like to present the problem of CIN after intravenous (iv) as well intra-arterial contrast media administration.

REVIEW

The pathogenesis of contrast induced acute kidney injury (CI-AKI) after contrast media

The pathogenesis of contrast induced acute kidney injury is complex. Iodinated contrast media injected intravenously or intra-arterial goes from vascular compartment to the extracellular space and is eliminated through glomerular filtration. It leads at the beginning to short increase of renal blood flow, followed by prolonged decreased blood flow resulting decreased glomerular filtration rate (GFR). It causes the ischemia of medulla, enlarges the damage of endothelial cells, among others the tubular epithelial cells [20-23]. The endothelium damage outside pre-existing clinical conditions is influenced by contrast media properties such as high osmolality and ionic strength [24-26]. Contrast media in in-vitro studies causes increase of renal tubular viscosity, tubular obstruction and elevates interstitial pressure [27].

Definition of contrast media induced acute kidney injury

In order to reduce the risk of CIN European Society of Urogenital Radiology as also American College of Radiology and the Canadian Association of Radiologists have published guidelines for iv contrast medium administration. All mentioned study groups concentrated on basal serum creatinine (SCr) levels, as a potential risk factor for later development of CIN [19,28,29].

Contrast-induced nephropathy (CIN) is defined as an acute, generally reversible decline in renal function that occurs 48-72 hours after intravascular injection of contrast medium [30,31]. CIN, depending on the rout of administration of contrast media, is classified as intravenous or intra-arterial CIN [19].

The definition of contrast induced AKI is based on the same criteria as other forms of AKI and except changes in serum creatinine concentration it includes also urine creatinine output changes [32]. The term CI-AKI is usually defined as the serum creatinine concentration rise of ≥0,5mg/dl (≥44 µmol/l) or a 25% increase from the baseline value occurs 24-48 hours, or up to 72 hours after intravenous or intra-arterial administration of contrast media and returns to the baseline within 14 days [29,32-34].

To standardize the definition for CIN The Acute Kidney Injury Network (AKIN) proposed new diagnostic criteria including the increase in serum creatinine level of>0.3 mg/dl (26.4 µmol/l) or a percentage increase in serum creatinine level of ≥50% from the baseline, or a reduction in urine output (oliguria of <0.5 ml/kg/h for > 6 hours) within 48 hours after contrast administration. The AKIN highlights the fact that these thresholds may be over-sensitive and cause more false-positive cases[34-37].

Risk factors for CI-AKI

These days the majority of radiological examinations are performed in outpatients. Also worth noting is aging population with coexisting many chronic diseases. In this situation it seems important to find patients with higher risk of CI-AKI in order to plan appropriate prevention procedures.

Pre-existing renal insufficiency, estimated in polish population on 5,8% [7] is as a risk factor above other risk factors for occurrence of CI-AKI [32,38]

The discussion about the threshold of creatinine or eGFR, below witch the risk of CIN increases, was the subject of various studies and meta-analysis. The CI-AKI Consensus Working Panel agreed that the prevention should be implemented among patients with baseline eGFR < 60 ml/min/1.73m^2 or even < 45 ml/min/1.73m^2 [32]. Large studies published in 2013 and 2014 proved that exposure to intravenous iodinated contrast media in patients with stable baseline eGFR ≥45 ml/min/1.73m^2 is not an independent nephrotoxic risk factor of AKI. Moreover, among patients with eGFR between 30-44 ml/min/1.73m^2 CI-AKI was observed less than expected [38-42]. What is worth a comment and need further analysis, that in only two of presented above study's authors characterized exposure on intravenous iodinated contrast material

as independent nephrotoxic risk factor in patients with eGFR < 30 ml/min/1.73m^2 [39,40].

Other risk factors predisposing the occurrence of CI-AKI, such as diabetes mellitus, hypertension, advanced age or hemodynamic instability increase the risk of AKI, but are not characterized as independent risk factors [38,41-43].

It is also important to differentiate the intravenous from intra-arterial administration of contrast media, which is related to higher risk of CI-AKI [19]. Patients undergoing angiographic procedures have higher risk of cardiovascular diseases or haemodynamic instability (hypotension, reduced cardiac output), as also the contrast media volume, depending on the procedure may be higher. Intra-arterial injection of contrast media causes higher iodine concertation in kidney capillaries, that might be related to later observed AKI [44-46]. It is important to notice, that through higher number of studies concentrating on cardiac angiography if compared to intravenous contrast media administration the risk of CIN among the second group might be over-estimated [47].

Abujudeh et al. as also Trivedi and Foley noticed that multiple, closely spaced iodinated intravenous contrast media doses may determine risk factor of higher CIN occurrence in studied populations [48,49]. There is no exact time interval which should be maintained between successive exposition on the contrast media. Some experts accept a 30-day limit in outpatients [28]. Repeated contrast media administration within 24-48 hours could positively influence the risk for CIN [50].

There are also contrast media related risk factors depending on contrast osmolality. Contrast media are divided into high-osmolar, low-osmolar and iso-osmolar contrast material. High-osmolar contrast media was identified as risk factor for CI-AKI, and because of this fact it is almost completely not in use any more [19,51,52].

Markers of AKI

To get performed a contrast enhanced computer tomography outpatients are obliged to submit current creatinine level. Already in 1998 Choyke et al. [53] elaborated a list of risk factors in order to identify patients with impaired renal function. On the basis of available literature as also experts opinion the paper published in 2014 present a list of risk factors, the presence of witch should require baseline serum creatinine concentration before the use of iodinated contrast medium administration. These factors include, among others: age >60, history of renal disease, hypertension or diabetes mellitus [28,54].

Acute kidney injury is diagnosed in everyday practice through serum creatinine levels measurements in time intervals. It is important to mention that serum creatinine changes are slowly following the onset of AKI. The steady state may be reached days after the exposure to nephrotoxic factor. Based on serum creatinine eGFR is estimated. Unfortunately GFR reduction is observed when up to 50% of kidney function has been lost. Concluding serum creatinine, despite its widespread availability, is not a good marker for AKI [55,56]. The question remains open if creatinine as also eGFR are proper markers for detection of patients with higher risk of AKI.

Many available studies concentrate on intra-arterial contrast supply, especially in invasive cardiology procedures, which are associated with higher risk of CI-AKI than computer enhanced tomography. It may lied to overestimation of the CI-AKI risk in patients undergoing intravenous contrast media exposure. Such apparently similar, but in practice different studies should not be compared because of different kidney exposure on the contrast media [19,57].

Crucial studies were published by McDonald et al., who questioned the concept of CI-AKI after contrast media exposure. Over than 10,000 patients were retrospectively analyzed in this multiple propensity score matched trials, each trial with over 10000 patients. In the large retrospective single-center study they observed equal risk of AKI among patients receiving contrast enhanced computer tomography if compared to unenhanced computer tomography. AKI was based on serum creatinine level differences. The results suggest rather coincidental AKI in patients receiving contrast media, than statistically significant differences independently from basal renal function. [39,40]. The authors noticed some study limitations, among others the study population, who consisted on inpatients, as also exclusion of patients receiving multiple doses of contrast media. Published in 2013 systemic review, based on studies concerning AKI incidence among patients underwent enhanced and unenhanced CT, showed similar risk of AKI in both group of patients. Even the risk of dialysis and mortality was comparable in both groups [38]. Newhouse et al. [58] pointed out the problem of misinterpretation of serum creatinine levels. Daily variations in serum creatinine concentration might be interpreted as AKI.

CONCLUSIONS

The debate about CI-AKI is still open and even more serious because of rising number of contrast enhanced computer tomography and angiography procedures. The anxiety of CI-AKI often leads to a refusal of performing the procedure, commonly in outpatients, what results prolongation of diagnostic procedures. Observed and analyzed since over 60 years problem of CI-AKI seems to be less serious than it was presumed. Non-controlled studies possibly exaggerated the risk of CI-AKI especially after intravenous exposure. New control

studies assume no risk of AKI after contrast media exposure in patients with proper renal function. The question of risk in patients with chronic kidney disease still remains open.

Conflicts of interest

The authors declare that they have no conflicts of interest.

REFERENCES

1. Levy EM, Viscoli CM, Horwitz RL. The effect of acute renal failure on mortality. A cohort analysis. JAMA 1996 May;275(19):1489-94.
2. Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. J Am Soc Nephrol. 2005 Nov;16(11):3365-70.
3. Weisbord SD, Chen H, Stone RA, Kip KE, Fine MJ, Saul MI, Palevsky PM. Associations of increases in serum creatinine with mortality and length of hospital stay after coronary angiography. J Am Soc Nephrol. 2006 Oct;17 (10):2871-7.
4. Barrett J. Contrast nephrotoxicity. J Am Soc Nephrol 1994 Aug;5(2):125-37.
5. Mehran R, Nikolsky E. Contrast-induced nephropathy: definition, epidemiology, and patients at risk. Kidney Int Suppl, 2006 Apr; (100):S11–5.
6. Murakami R, Hayashi H, Sugizaki K, Yoshida T, Okazaki E, Kumita S, Owan C.. Contrast-induced nephropathy in patients with renal insufficiency undergoing contrast-enhanced MDCT. Eur Radiol, 2012 Oct;22(10):2147-52.
7. Zdrojewski Ł, Zdrojewski T, Rutkowski M, Bandosz P, Krol E, Wyrzykowski B, Rutkowski B. Prevalence of chronic kidney disease in a representative sample of the Polish population: results of the NATPOL 2011 survey. Nephrol Dial Transplant. 2016 Mar;31(3):433-9.
8. Chadban SJ, Briganti EM, Kerr PG, Dunstan DW, Welborn TA, Zimmet PZ, Atkins RC. Prevalence of kidney damage in Australian adults: the AusDiab kidney study. J Am Soc Nephrol. 2003 Jul;14(7Suppl 2):S131-8.
9. Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, Van Lente F, Levey AS. Prevalence of chronic kidney disease in the United States. JAMA, 2007 Nov;298(17):2038-47.
10. Katzberg RW, Haller C. Contrast-induced nephrotoxicity: clinical landscape. Kidney Int Suppl. 2006 Apr;(100)S3–7.
11. Bartels ED, Brun GC, Gammeltoft A, Gjorup PA. Acute anuria following intravenous pyelography in a patient with myelomatosis. Acta Med Scand. 1954;150(4):297-302.
12. Lee J, Cho JY, Lee HJ, Jeong YY, Kim CK, Park BK, Sung DJ, Kang BC, Jung SI, Lee EJ, Yi BH, Park SJ, Kim JC, Jung DC, Sung CK, Kim Y, Lee Y, Kim SH, Yoon SK, Park BJ, Kim SH; Korean Society of Urogenital Radiology (KSUR); Korean Society of Radiology. Contrast-induced nephropathy in patients undergoing intravenous contrast-enhanced computed tomography in Korea: a multi-institutional study in 101487 patients. Korean J Radiol. 2014 Jul-Aug;15(4):456-63.
13. Davenport MS, Khalatbari S, Cohan RH, Dillman JR, Myles JD, Ellis JH. Contrast material-induced nephrotoxicity and intravenous low-osmolality iodinated contrast material: risk stratification by using estimated glomerular filtration rate. Radiology. 2013 Sep; 268(3):719-28.
14. Kooiman J, Sijpkens YW, van Buren M, Groeneveld JH, Ramai SR, van der Molen AJ, Aarts NJ, van Rooden CJ, Cannegieter SC, Putter H, Rabelink TJ, Huisman MV. Randomised trial of no hydration vs. sodium bicarbonate hydration in patients with chronic kidney disease undergoing acute computed tomography-pulmonary angiography. J Thromb Haemost. 2014 Oct;12(10):1658-66.
15. Andreucci M, Solomon R, Tasanarong A. Side effects of radiographic contrast media: pathogenesis, risk factors, and prevention. Biomed Res Int. 2014;2014:741018.
16. Lima FO, Lev MH, Levy RA, Silva GS, Ebril M, de Camargo EC, Pomerantz S, Singhal AB, Greer DM, Ay H, González RG, Koroshetz WJ, Smith WS, Furie KL. Functional contrast-enhanced CT for evaluation of acute ischemic stroke does not increase the risk of contrast-induced nephropathy. AJNR Am J Neuro-radiol. 2010 May;31(5):817-21.
17. McGillicuddy EA, Schuster KM, Kaplan LJ, Maung AA, Lui FY, Maerz LL, Johnson DC, Davis KA. Contrast induced nephropathy in elderly trauma patients. J Trauma. 2010 Feb;68 (2):294-7.
18. Kuhn MJ, Chen N, Sahani DV, Reimer D, van Beek EJ, Heiken JP, So GJ. The PREDICT study: a randomized double-blind comparison of contrast-induced nephropathy after low- or isoosmolar contrast agent exposure. AJR Am J Roentgenol. 2008 Jul;191(1):151-7.
19. Stacul F, van der Molen AJ, Reimer P, Webb JA, Thomsen HS, Morcos SK, Almén T, Aspelin P, Bellin MF, Clement O, Heinz-Peer G. Contrast Media SafetyCommittee of Euro-pean Society of Urogenital Radiology (ESUR). Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines. Eur Radiol. 2011 Dec;21(12):2527-41.
20. Andreucci M, Solomon R, Tasanarong A. Side effects of radiographic contrast media: patho-

genesis, risk factors, prevention. Biomed Res Int. 2014;2014:741018.
21. Murphy S.W Barrett B.J, Parfrey P.S. Contrast nephropathy. J Am Soc Nephrol. 2000 Jan;11 (1):177-82.
22. Detrenis S, Meschi M, Musini S, Savazzi G. Lights and shadows on the pathogenesis of contrast-induced nephropathy: state of the art. Nephrol Dial Transplant. 2005 Aug;20(8):1542-50.
23. Heyman SN, Rosen S, Rosenberger C. Renal parenchymal hypoxia, hypoxia adaptation, and the pathogenesis of radiocontrast nephropathy. Clin J Am Soc Nephrol. 2008 Jan;3(1):288-96.
24. Sendeski MM. Pathophysiology of renal tissue damage by iodinated contrast media. Clin Exp Pharmacol Physiol, 2011 May;38(5):292-99.
25. Heinrich MC, Kuhlmann MK, Grgic A, Heckmann M, Kramann B, Uder M. Cytotoxic effects of ionic highosmolar, nonionic monomeric, and nonionic iso-osmolar dimeric iodinated contrast media on renal tubular cells in vitro. Radiology. 2005 Jun;235(3):843-49.
26. Andreucci M, Faga T, Pisani A, Sabbatini M, Michael A. Acute Kidney Injury by Radiographic Contrast Media: Pathogenesis and Prevention. BioMed Res Int. 2014;362725.
27. Chang CF, Lin CC. Current concepts of contrast-induced nephropathy: a brief review. J Chin Med Assoc. 2013 Dec;76(12):673-81.
28. American College of Radiology ACR Manual on Contrast Media Version 10.1 2015 [Internet]. [cited 2016 Mar 12] Available from: http://www.acr.org/quality-safety/resources/ contrast-manual.
29. Owen RJ, Hiremath S, Myers A, Fraser-Hill M, Barrett B. Consensus Guidelines for the Prevention of Contrast Induced Nephropathy. Canadian Association of Radiologist Web site [Internet]. [cited 2016 Mar 12] Available from: http://www.car.ca/ uploads/standards% 20 guidelines/20110617_en_ prevention_cin.pdf
30. Morcos SK. Contrast medium-induced nephrontoxicity. In: Dawson P, editor. Textbook of Contrast Media. Isis Medical Media Ltd; 1999.
31. Barret. Contrast nephrotoxicity. J Am Soc Nephrol. 1994 Aug;5(2):125-37.
32. Kellum JA, Lameire N, et al. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl, 2012;2:124-38.
33. Harjai KJ, Raizada A, Shenoy C, Sattur S, Orshaw P, Yaeger K, Boura J, Aboufares A, Sporn D, Stapleton D. A comparison of contemporary definitions of contrast nephronpathy in patients undergoing percutaneous coronary intervention and a proposal for a novel nephropathy grading system. Am J Cardiol. 2008 Mar 15;101(6):812–19.
34. Molitoris BA, Levin A, Warnock DG, Joannidis M, Mehta RL, Kellum JA, Ronco C, Shah S. Improving outcomes from acute kidney injury. J Am Soc Nephrol. 2007 Jul;18(7):1987–94.
35. Molitoris BA, Levin A, Warnock DG, Joannidis M, Mehta RL, Kellum JA, Ronco C, Shah SV; Acute Kidney Injury Network working group. Improving outcomes of acute kidney injury: report of an initiative. Nat Clin Pract Nephrol. 2007 Aug;3(8):439-42.
36. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A; Acute Kidney Injury Network. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007;11(2):R31.
37. Waybill MM, Waybill PN. Contrast media-induced nephrotoxicity: identification of patients at risk and algorithms for prevention. J Vasc Interv Radiol. 2001 Jan;12(1):3-9.
38. McDonald JS, McDonald RJ, Comin J, Williamson EE, Katzberg RW, Murad MH, Kallmes DF. Frequency of acute kidney injury following intravenous contrast medium administration: a systemic revive and meta-analysis. Radiology, 2013 Apr;267(1):119-28.
39. Davenport MS, Khalatbari S, Cohan RH, Dillman JR, Myles JD, Ellis JH. Contrast material-induced nephrotoxicity and intravenous low-osmolality iodinated contrast material: risk stratification by using estimated glomerular filtration rate. Radiology. 2013 Sep;268(3):719-28.
40. Davenport MS, Khalatbari S, Cohan RH, Dillman JR, Myles JD, Ellis JH.Contrast material-induced nephrotoxicity and intravenous low-osmolality iodinated contrast material. Radiology 2013 Apr;267(1):94-105.
41. McDonald JS, McDonald RJ, Carter RE, Katzberg RW, Kallmes DF, Williamson EE. Risk of intravenous contrast material-mediated acute kidney injury: a propensity score-matched study stratified by baseline-estimated glomerular filtration rate. Radiology 2014 Apr:271(1):65-73.
42. McDonald RJ, McDonald JS, Bida JP, Carter RE, Fleming CJ, Misra S, Williamson EF, Kallmes DF. Intravenous contrast material-induced nephropathy: causal or coincident phenomenon? Radiology. 2013 Apr;267(1):106-18.
43. Mehran R, Nikolsky E. Contrast-induced nephropathy: definition, epidemiology, and patients at risk. Kidney Int Suppl. 2006 Apr;100: S11–15.
44. Dong M, Jiao Z, Liu T. Effect of administration rout on the renal safety of contrast agents: a meta-analysis of randomized controlled trials. J Nephrol, 2012 May-Jun; 25(3):290-301.
45. Karlsberg RP, Dohad SY, Sheng R. Iodixanol Peripheral Computer Tomographic Angiography Study Investigator Panel. Contrast

medium-induced acute kidney injury: comparison of intravenous and intra-arterial administration of iodinated contrast medium. J Vasc Interv Radiol, 2011 Aug; 22(8):1159-65.
46. James MT, Ghali WA, Knudtson ML, Ravani P, Tonelli M, Faris P, Pannu N, Manns BJ, Klarenbach SW, Hemmelgarn BR; Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease (APPROACH) Investigators. Association between acute kidney injury and cardiovascular and renal outcomes after coronary angiography. Circulation. 2011 Feb 1;123(4):409-14.
47. Katzberg RW, Newhouse JH. Intravenous contrast medium-induced nephrotoxicity: is the medical risk really as great as we have come to believe? Radiology 2010 Jul;256(1):21–8.
48. Abujudeh HH, Gee MS, Kaewlai R. In emergency situations, should serum creatinine be checked in all patients before performing second contrast CT examinations within 24 hours? J Am Coll Radiol. 2009 Apr; 6(4):268-73.
49. Trivedi H, Foley WD. Contrast-induced nephropathy after a second contrast exposure. Ren Fail. 2010;32(7):796-801.
50. Guidelines on the Use of Iodinated Contrast Media in Patients With Kidney Disease 2012. Digest Version. JSN, JRS and JCS Joint Working Group. [cited 2016 Mar 12] Available from: https://www. jstage.jst.go.jp/ article/ circj/ 77/ 7/ 77_CJ-66-0069/_article
51. Bruce RJ, Djamali A, Shinki K, Michel SJ, Fine JP, Pozniak MA. Background fluctuation of kidney function versus contrast-induced nephrotoxicity. AJR Am J Roentgenol. 2009 Mar;192(3):711-8.
52. Moos SI, van Vemde DN, Stoker J, Bipat S. Contrast induced nephropathy in patients undergoing intravenous (IV) contrast enhanced computed tomography (CECT) and the relationship with risk factors: a meta-analysis. Eur J Radiol. 2013 Sep;82(9):e387-99.
53. Choyke PL, Cady J, DePollar SL, Austin H. Determination of serum creatinine prior to iodinated contrast media: is it necessary in all patients? Tech Urol. 1998 Jun;4(2):65-9.
54. Tippins RB, Torres WE, Baumgartner BR, Baumgarten DA. Are screening serum creatinine levels necessary prior to outpatient CT examinations? Radiology. 2000 Aug;216(2):481-4.
55. Malyszko J. Biomarkers of Acute Kidney Injury in Different Clinical Settings: A Time to Change the Paradigm? Kidney Blood Press Res. 2010; 33(5):368–82.
56. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute Dialysis Quality Initiative workgroup. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care. 2004 Aug;8(4):R204-12.
57. Katzberg RW, Barrett BJ. Risk of iodinated contrast material induced nephropathy with intravenous administration. Radiology.2007 Jun;243(3):622-8.
58. Newhouse JH, Kho D, Rao QA, Starren J. Frequency of serum creatinine changes in the absence of iodinated contrast material: implications for studies of contrast nephrotoxicity. AJR Am J Roentgenol. 2008 Aug; 191(2):376-82.