Eur J Med Res (2005) 10: 149-154

Renal Effects of Gadopentetate Dimeglumine in Patients with Normal and Impaired Renal Function

U. Hoffmann[1], M. Fischereder[2], A. Reil[1], M.Fischer[1], J. Link[3], B. K. Krämer[1]

[1]Department of Internal Medicine II, University of Regensburg,
[2]Department of Internal Medicine, Ludwig-Maximilians-University Munich,
[3]Institute of Radiology, University of Regensburg, Germany

Abstract: Gadolinium chelates are widely used in magnetic resonance imaging as contrast medium in patients with nephropathy. However, only few studies have investigated the effect of gadolinium on serum creatinine concentration and estimated GFR as surrogate markers of renal function. This study was performed to evaluate the effect of gadopentetate dimeglumine in a dose sufficient for diagnostic and interventional purposes on renal function in a large sample of patients.

We analyzed serum creatinine and serum-urea levels before and after the administration of gadopentetate dimeglumine in patients with normal and patients with pre-existing impaired renal function. Age, height, body mass, sex, medication and preexisting illnesses such as diabetes, renal artery stenosis and heart disease were monitored.

In 181 patients with normal renal function, there was no statistically significant change in serum creatinine concentration after the administration of gadopentetate dimeglumine (at baseline: 0.72 ± 0.18 mg/dl, after gadolinium: 0.73 ± 0.22 mg/dl). In contrary, serum creatinine levels decreased significantly after the administration of gadolinium in 198 patients with pre-existing renal impairment (1.82 ± 1.03 mg/dl before and 1.72 ± 1.03 mg/dl after gadolinium) ($p<0.01$). According to this surrogate marker of renal function, the change of estimated GFR in patients with normal baseline renal function was not significant, while in patients with impaired renal function, GFR increased after the administration of gadolinium ($p<0.001$).

The high diagnostic value of gadolinium contrast media is associated with a very small risk of adverse reactions. Our findings show that the administration of gadolinium even is associated with a decrease of serum creatinine in patients with pre-existing renal impairment. In conclusion, the use of gadolinium-based contrast media may be considered as a safe alternative in patients with impaired renal function for whom use of iodine-based contrast agents is prone to a high rate of radiocontrast-induced nephropathy.

Key words: Gadolinium; creatinine; renal function; nephropharmacology

Introduction:

Radiocontrast-induced nephropathy is a critical consideration during the performance of diagnostic and interventional radiological procedures in patients with renal impairment [1, 2]. In contrast to conventional contrast-based angiography, there are several imaging modalities that do not use iodinated contrast. These include gadolinium-enhanced magnetic resonance angiography [3]. Gadolinium-based contrast agents have minimal toxicity in their standard use in MR imaging [4, 5, 6, 7, 8]. They are cleared from the body by glomerular filtration and are dialyzable [9, 10, 11].

For gadopentetate dimeglumine, the overriding majority of adverse events consist of transient and relatively minor symptoms and reactions. The incidence of severe reactions after the injection of gadopentetate dimeglumine is considerably lower than that for iodinated contrast agents, both ionic and nonionic [5]. Nevertheless, data exist showing nephrotoxic side effects of gadolinium, especially when injected in relatively high doses resulting in a substantial osmotic load to the kidneys or when the renal arteries are directly exposed to these hypertonic solutions [12, 13].

This study was performed to evaluate the influence of gadopentetate dimeglumine injection on serum creatinine concentration and on estimated GFR when administered during routine clinical use in patients with normal and in patients with pre-existing reduced renal function not treated with renal replacement therapy.

Patients, Methods, Statistics

Data from 3634 patients undergoing magnetic resonance imaging (MRI) without receiving iodinated contrast agents during the blood sample collecting period were retrospectively analyzed at the University Hospital of Regensburg. Gadopentetate dimeglumine-based contrast material (0.5 mmol/ml; Magnevist, Schering, Germany) was administered undiluted. Normal serum creatinine levels were defined for men as 0.50 – 1.10 mg/dl and for women as 0.50 – 0.90 mg/dl. Serum creatinine levels of patients with pre-existing impaired renal function were within 1.11 – 5.00 mg/dl for men and 0.91 – 5.00 mg/dl for women. None of the patients had renal replacement therapy. Furthermore, a change in any medication or the distribution of i.v. fluids during the analyzed period was an exclusion criterium. Only patients with obtained serum creatinine levels just before and within 1-8 days after the administration of gadolinium were monitored. Finally, 181 pa-

tients with normal renal function and 198 patients with impaired renal function could be included in our study. The following data were extracted from the patients data sheets: Sex, age, height, body mass and medication. The patients with an elevated serum creatinine level were further divided into 2 groups: Patients with a baseline creatinine level of 1.51 – 3.00 mg/dl (n = 89) and patients with a baseline creatinine level of 3.01 – 5.00 mg/dl (n = 23). Patients with and without diabetes, renal artery stenosis, heart disease, and patients taking ACE inhibitors, NSAIDS or diuretics were analyzed as subgroups

Glomerular filtration rate (GFR) was estimated by Cockcroft and Gault's formula [14] in both patients with normal (n = 104) and patients with impaired renal function (n = 183).

Descriptive data are expressed as mean ± standard deviation. Differences in serum creatinine levels before and after gadolinium were recorded and a paired-sample t-test was used to test for significance under the null hypothesis that the mean of the distribution is equal to zero. In case of an extremely skewed difference a nonparametric signed-rank test was requested instead. Classification and regression trees were used for partitioning recursively subjects into distinct subgroups based on the outcome in creatinine levels. With this multivariable technique the strongest risk factors for an increase in creatinine were sequentially determined. P values <0.05 were considered as statistically significant.

Results

No patient included in our analysis showed a rise in serum creatinine of more than 0.5 mg/dl after the administration of gadopentetate dimeglumine.

Patients with Normal Renal Function:

The mean age of the patients with normal serum creatinine (n=181) was 51.9 ± 15.8 years. Gadolinium was given at a mean dose of 0.12 ± 0.04 mmol/kg body weight. Before the administration of gadolinium, mean serum creatinine concentration was 0.72 ± 0.18 mg/dl and 0.73 ± 0.22 mg/dl after gadolinium. In 96 men, it was 0.81 ± 0.17 mg/dl before and 0.83 ± 0.21 mg/dl after and in 85 women, it was and 0.62 ± 0.14 mg/dl before and 0.63 ± 0.18 mg/dl after the administration of gadolinium. The little trend for an increase in serum creatinine concentration in all these groups was not statistically significant (Fig. 1).

GFR was estimated in the patients with normal renal function before (119.0 ± 42.8 ml/min.) and after (119.1 ± 43.4 ml/min.) the administration of gadolinium (not significant).

In men, GFR was 117 ± 42 ml/min. before and 116 ± 44 ml/min. after and in women 121.1 ± 43.9 ml/min. before and 122.3 ± 43.1 ml/min. after the administration of gadolinium (not significant) (Fig. 2).

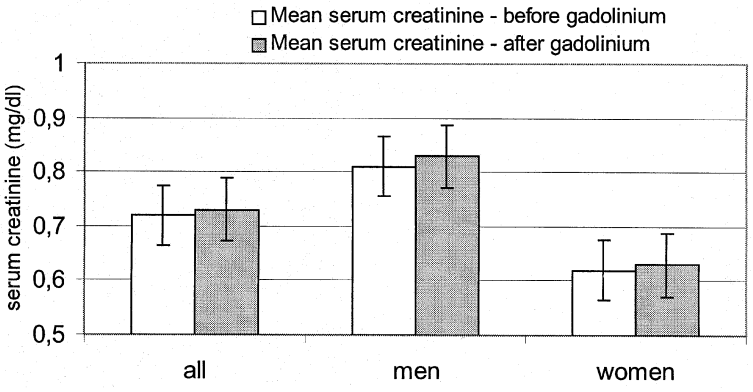

Fig. 1. Patients with normal renal function before and after the administration of gadolinium. Mean serum creatinine levels in all patients (n = 181). Subgroups: men (n = 96) and women (n = 85). There is no significanct change.

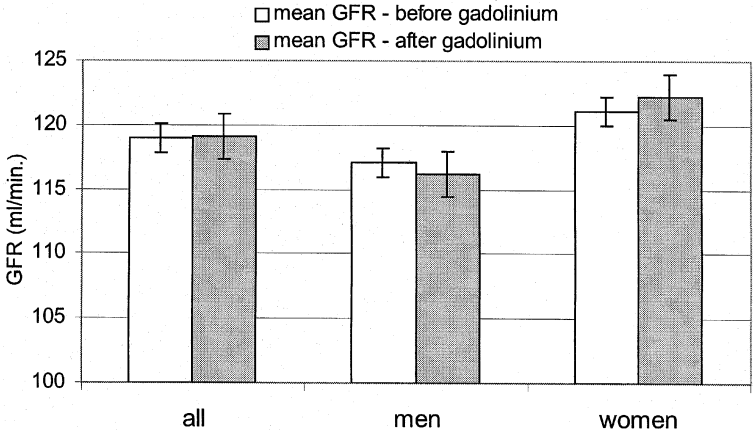

Fig. 2. Patients with normal renal function before and after the administration of gadolinium. Mean GFR in all patients (n = 104). Subgroups: men (n = 54) and women (n = 50). There is no significant change.

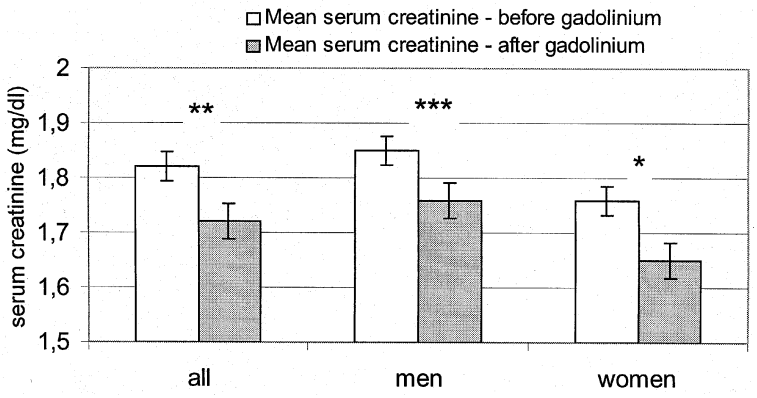

Fig. 3. Patients with impaired baseline renal function before and after the administration of gadolinium. Mean serum creatinine levels in all patients (n = 198). Subgroups: men (n=128) and women (n = 70).
* p< 0.05, ** p< 0.01, *** p<0.001.

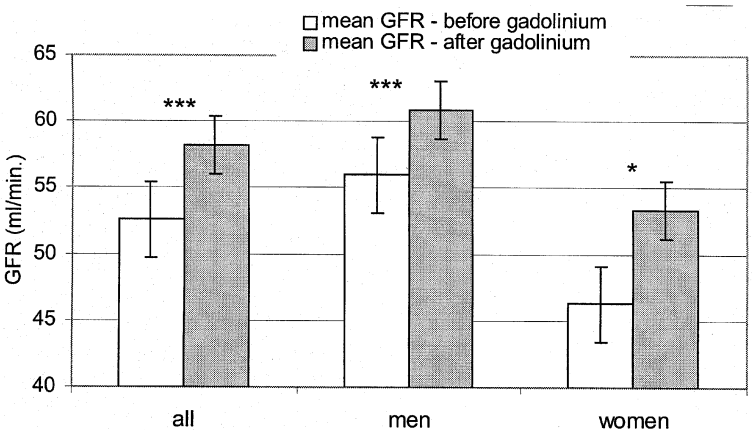

Fig. 4. Patients with impaired baseline renal function before and after the administration of gadolinium. Mean GFR in all patients (n=183). Subgroups: men (n = 119) and women (n = 64).
*** p < 0.001, * p < 0.05.

Patients with Pre-existing Renal Impairment:

In patients with pre-existing renal impairment, mean age was 60.0 ± 14.1 years. The administered mean dose of gadolinium was 0.14 ± 0.05 mmol/kg. The mean serum creatinine concentration (n=198) decreased significantly from 1.82 ± 1.03 mg/dl before to 1.72 ± 1.03 mg/dl after the administration of gadolinium ($p<0.01$). In men (n = 128), mean serum creatinine level was 1.85 ± 0.94 mg/dl before and 1.76 ± 0.96 mg/dl after ($p<0.001$) and in women (n=70), it was 1.76 ± 1.16 mg/dl before and 1.65 ± 1.13 mg/dl after the administration of gadolinium ($p<0.05$) (Fig. 3).

The mean GFR increased from 52.6 ± 24.5 ml/min. before to 58.2 ± 30.4 ml/min. after the administration of gadolinium ($p<0.001$) in the patients with impaired renal function. In men, GFR was 56.0 ± 24.1 ml/min. before and 60.8 ± 28.7 ml/min. after ($p<0.001$) and in women, it was 46.2 ± 24.1 ml/min. before and 53.3 ± 32.9 ml/min. after gadolinium ($p<0.01$) (Fig. 4).

Subgroup analysis of patients with baseline-creatinine concentrations between 1.51 and 3.00 mg/dl (n = 89) showed a mean serum creatinine level of 2.60 ± 1.10 mg/dl before and 2.46 ± 1.12 mg/dl after the administration of gadolinium ($p < 0.01$). In the group of patients with a baseline creatinine of 3.01 – 5.00 mg/dl (n = 23), the difference did not reach statistical significance (4.23 ± 0.80 mg/dl before and 4.11 ± 0.85 mg/dl after gadolinium).

Subgroups

The results regarding the comparison of subgroups are shown in Table 1.

Statistically significant decreases of serum creatinine concentration after the administration of gadolinium were seen only in patients with impaired renal function but without diabetes or without heart disease or without taking NSAIDs. Furthermore, there was a significant decrease of serum creatinine concentration in both subgroups with reduced renal function with or without diuretics or with or without ACE inhibitors. Classification and regression trees were used for partitioning recursively subjects into distinct subgroups based on the outcome in creatinine levels. With this multivariable technique no groups at high risk to develop an increase in creatinine could be determined.

Discussion

Many studies have been carried out to examine the clinical safety of the four gadolinium-based contrast agents currently available [6, 7, 12, 15, 16].

Most of them focused on allergic adverse reactions of gadolinium without investigation of a potential change in renal function after its administration. Allergic reactions as nausea or vomiting, hives, diffuse erythema or skin irritation, periorbital edema or respiratory symptoms are reported to occur in 372 of 15496 patients (2,4 %) undergoing MR imaging [17] or in 36 of 21000 patients (0,2 %) studied by Murphy et al.

Table 1. Changes of creatinine in mg/dl (mean +/- SD) before and after the administration of gadolinium in subgroups. ns = non significant.

Subgroups of patients	n	creatinine mg/dl (mean ± SD) baseline	after gadolinium	p
no diabetes, normal renal function	158	0.73 ±0.17	0.74 ± 0.21	ns
no diabetes, reduced renal function	129	1.85 ± 1.07	1.72 ± 1.03	p<0.001
diabetes, normal renal function	23	0.67 ± 0.24	0.69 ± 0.25	ns
diabetes, reduced renal function	69	1.76 ± 0.93	1.73 ± 1.02	ns
renal artery stenosis, normal renal function	2	0.88 ± 0.06	1.09 ± 0.20	ns
renal artery stenosis, reduced renal function	30	2.46 ± 1.25	2.51 ± 1.26	ns
no heart disease, normal renal function	122	0.72 ± 0.18	0.73 ± 0.20	ns
no heart disease, reduced renal function	95	1.69 ± 0.85	1.56 ± 0.83	p<0.001
heart disease, normal renal function	10	0.77 ± 0.19	0.78 ± 0.22	ns
heart disease, reduced renal function	37	1.82 ± 0.94	1.85 ± 0.98	ns
no ACE inhibitors, normal renal function	145	0.71 ± 0.19	0.72 ± 0.21	ns
no ACE inhibitors, reduced renal function	100	1.8 ± 1.03	1.69 ± 1.04	p<0.01
ACE inhibitors, normal renal function	36	0.75 ± 0.17	0.81 ± 0.25	ns
ACE inhibitors, reduced renal function	94	1.83 ± 1.02	1.75 ± 1.02	p<0.05
no NSAIDs, normal renal function	118	0.72 ± 0.18	0.74 ± 0.23	ns
no NSAIDs, reduced renal function	110	1.81 ± 0.98	1.66 ± 0.96	p<0.05
NSAIDs, normal renal function	63	0.72 ± 0.20	0.72 ± 0.21	ns
NSAIDs, reduced renal function	87	1.82 ± 1.07	1.78 ± 1.11	ns
no diuretics, normal renal function	140	0.72 ± 0.18	0.73 ± 0.20	ns
no diuretics, reduced renal function	67	1.35 ± 0.56	1.28 ± 0.60	p<0.05
diuretics, normal renal function	41	0.72 ± 0.19	0.75 ± 0.27	ns
diuretics, reduced renal function	130	2.05 ± 1.12	1.94 ± 1.22	p<0.002

[18]. Data collected after 90473 administrations of iodinated contrast media in comparison to 28340 administrations of gadolinium showed that mild and moderate adverse events are more common with iodinated radiocontrast media (rate of adverse reactions: 0.43 %) than with gadolinium MR contrast media (0.067 %) [19]. Changes in serum creatinine levels were not assessed in these studies.

To date, only few studies examined the effect of gadopentetate dimeglumine on serum creatinine concentration and estimated GFR after the administration of this MR contrast agent in comparison to baseline creatinine levels. In view of the potential risk of gadolinium-induced nephrotoxicity due to e.g. prolonged half-life, possible compound-specific differences in nephrotoxicity, and a probable higher risk of gadolinium-induced nephrotoxicity with high contrast volume, we aimed to assess the risk of gadolinium in a large sample of patients with normal and impaired renal function receiving contrast volume averaging 0,12-0,14 mmol/kg body weight during routine clinical investigations.

In our study, there was no change of serum creatinine level after the administration of gadolinium in patients with normal renal function. Though renal insufficiency is the most frequent risk factor associated with iodinated contrast-induced nephrotoxicity, multivariate analysis reveals that diabetes, renal artery stenosis, heart failure, NSAIDs, dose of contrast media and volume depletion contribute to the threat [20, 21]. In our findings, pre-existing illnesses as diabetes, renal artery stenosis or heart diseases and drug intake i.e. ACE inhibitors, diuretics or NSAIDs had no unfavourable effect when gadopentetate dimeglumine was used. Analyzing all patients with pre-existing reduced renal function and in accordance with earlier work [17], there was even a significant decrease in serum creatinine after the administration of gadolinium, while in diabetics, patients with heart disease and renal artery stenosis or patients taking NSAIDS, the decrease of serum creatinine levels was absent, possibly pointing to an increased risk of these patient subgroups for gadolinium-induced nephrotoxicity. In parallel with serum creatinine concentration, estimated GFR was unchanged in patients with normal renal function but increased in patients with reduced renal function after the administration of gadolinium. Potential explanations for this observation include a vol-

ume repletion due to the osmotic load from gadopentetate dimeglumine as well as improvement in the underlying renal pathology as a consequence of radiologic imaging. One has to keep in mind that an increase in estimated GFR rests on the assumption, that changes in serum creatinine truely reflect changes in renal function. However, recent data suggest effects of e.g. N-acetylcysteine on serum creatinine without any effect on renal function [22, 23]. There are different possible explanations for a decrease of serum creatinine after the administration of gadolinium: First, gadopentetate dimeglumine truly improves GFR. Second, gadopentetate dimeglumine does not alter GFR but causes a decrease in serum creatinine through another mechanism. Especially in patients with impaired renal function, the extent of tubular secretion may contribute significantly to the total creatinine excretion. Effects of gadopentetate dimeglumine on muscle metabolism might also play a role. Extending this finding to the use of gadopentetate dimeglumine, nonspecific pharmacologic effects also have to be considered. Yet, further studies examining this effect have to be done. Nevertheless, these data clearly demonstrate that the use of gadopentetate dimeglumine in an average dose of 0.12-0.14 mmol/kg body weight is without detrimental effects on renal function.

Our data with the highest number of patients to date are confirming results of few small studies that have already been reported. Prince et al. recorded serum creatinine levels in 64 patients pre and post both gadolinium (0.2 to 0.4 mmol/kg) and iodinated contrast administration [7]. The mean change in serum creatinine after gadolinium in these 64 patients was – 0.07 mg/dl in comparison to + 0.35 mg/dl in the patients after iodinated contrast ($p = 0.002$). None of the patients had gadolinium contrast-induced renal failure while eleven of the 64 patients developed radiocontrast-induced nephropathy. In another small study, none of the 28 patients with renal insufficiency receiving gadolinium chelates (0,24 ± 0,13 mmol/kg) as contrast agent for x-ray digital subtraction angiography had contrast-induced acute renal failure [24]. Comparable to our study, mean serum creatinine decreased after the administration of gadolinium (2.6 ± 1.5 mg/dl before and 2.3 ± 1.0 mg/dl after gadolinium).

In accordance with these clinical trials suggesting absence of gadolinium nephrotoxicity, some in vivo-studies have shown no change in glomerular filtration rate with gadolinium in rats and dogs [25, 26, 27]. However, in one study carried out by Brillet et al. [28], a decreased creatinine clearance in isolated ischaemic rat nephrons after perfusion with gadolinium was noticed.

There is one critical point in all these studies including our own: comparing toxicity between gadolinium-based and iodinated radiocontrast media, concentrations or doses that attain the same quality of images should be compared. Nyman et al. suggested that at those doses for digital subtraction angiography, modern iodinated contrast media even result in a lower toxic load on the body than the presently available gadolinium chelates. In his analysis, a 40 % incidence of nephropathy induced by the gadolinium chelate gadodiamide was shown to occur with a high dose of up to 220 mmol given to patients with azotemia undergoing digital subtraction angiography [12]. A recent small study using high doses of gadobutrol (0,57 mmol/kg body weight) and iohexol (0,6 mmol/kg body weight) for angiography showed the same incidence of acute renal failure with both compounds in 21 patients with renal dysfunction [13].

Furthermore, two clinical cases of acute renal failure after the administration of high doses of gadolinium (0.44 mmol/kg and 0.34 ± 0.06 mmol/kg) have been reported [29, 30]. Nevertheless, prospective, randomized larger trials comparing different doses of gadolinium with iodinated contrasts agent to assess the same high image quality are still lacking.

In conclusion, serum creatinine levels and estimated GFR before and after the administration of gadopentetate dimeglumine contrast were analyzed in a large sample of patients with normal and impaired renal function in this study. Our findings show that the administration of gadolinium in a moderate dose even is associated with a decrease of serum creatinine in patients with pre-existing renal impairment. Thus, gadopentetate dimeglumine in a dose sufficient for diagnostic and interventional purposes may be considered as a safe contrast agent in patients with impaired renal function for whom the use of iodine-based contrast agents is prone to a high rate of radiocontrast-induced nephropathy.

REFERENCES

1. Solomon R (1998) Contrast-medium-induced acute renal failure. Kidney Int 53: 230-242
2. Krämer BK, Kammerl M, Schweda F, Schreiber M (1999) A primer in radiocontrast-induced nephropathy. Nephrol Dial Transplant 14: 2830-2834
3. Bush RL, Lin PH, Bianco CC, Lumsden AB, Gunnoud AB, Terramani TT, Brinkman WT, Martin LG, Weiss VJ (2002) Endovascular aortic aneurysm repair in patients with renal dysfunction or severe contrast allergy: Utility of imaging modalities without iodinated contrast. Ann Vasc Surg 16: 537-44
4. Niendorf HP, Alhassan A, Geens VR, Claub W (1994) Safety review of gadopentetate dimeglumine extended clinical experience after more than five million applications. Invest Radiol 29: 179-182
5. Runge VM (2001) Safety of magnetic resonance contrast media. Top Magn Reson Imaging 12: 309-14
6. Haustein J, Niendorf HP, Krestin G, Louton T, Schuhmann-Giampieri G, Clauss W, Junge W (1992) Renal tolerance of gadolinium-DTPA/dimeglumine in patients with chronic renal failure. Invest Radiol 27: 153-156
7. Prince MR, Arnoldus C, Frisoli JK (1996) Nephrotoxicity of high-dose gadolinium compared with iodinated contrast. J Magn Reson Imaging 6:162-6
8. Arsenault TM, King BF, Marsh JW Jr, Goodman JA, Weaver AL, Wood CP, Ehman RL (1996) Systemic gadolinium toxicity in patients with renal insufficiency and renal failure: retrospective analysis of an initial experience. Mayo Clin Proc 71: 1150-4
9. Schuhmann-Giampieri G, Krestin G (1991) Pharmacokinetics of GD-DTPA in patients with chronic renal failure. Invest Radiol 26: 975-9
10. Dorsam J, Knopp MV, Schad L, Piesche S, Carl S, Oesingmann N (1995) Elimination of gadolinium-DTPA by peritoneal dialyses. Nephrol Dial Transplant 10: 1228-1230

11. Choyke PL, Girton ME, Vaughan EM, Frank JA, Austin HA (1995) Clearance of gadolinium chelates by hemodialyses: an in vitro study. J Magn Reson Imaging 5: 470-472
12. Nyman U, Elmstahl B, Leander P, Nilsson M, Golman K, Almen T (2002) Are gadolinium-based contrast media really safer than iodinated media for digital subtraction angiography in patients with azotemia? Radiology 223: 311-318
13. Erley CM, Bader BB, Berger ED, Tuncel N, Winkler S, Tepe G, Risler T, Duda S (2004) Gadolinium-based contrast media compared with iodinated media for digital subtraction angiography in azotaemic patients. Nephrol Dial Transplant 19: 2526-2531
14. Cockcroft DW, Gault MH (1976) Prediction of creatinine clearance from serum creatinine. Nephron 16: 31-41
15. Carr JJ (1994) Magnetic resonance contrast agents for neuroimaging. Safety issues. Neuroimaging Clin N Am 4: 43-54
16. Hammer FD, Gofette PP, Malaise J, Mathurin P (1999) Gadolinium dimeglumine: an alternative contrast agent for digital subtraction angiography. Eur Radiol 9: 128-136
17. Nelson KL, Gifford LM, Lauber-Huber C, Gross CA, Lasser TA (1995) Clinical safety of gadopentetate dimeglumine. Radiology 196: 439-43
18. Murphy KJ, Brunberg JA, Cohan RH (1996) Adverse reactions to gadolinium contrast media: a review of 36 cases. Am J Roentgenol 167: 847-9
19. Cochran ST, Bmyea K, Sayre JW (2001) Trends in adverse events after IV administration of contrast media. Am J Roentgenol 176: 1385-8
20. Spinosa DJ, Matsumoto AH, Angle JF, Hagspiel KD, Leung DA, Hartwell GD (2001) Gadolinium contrast agents: their role in vascular and nonvascular diagnostic angiography and interventions. Tech Vasc Interv Radiol 4: 45-52
21. Barrett B, Carlisle E (1993) Metaanalysis of the relative nephrotoxicity of high- and low-osmolality iodinated contrast media. Radiology 188: 171-178
22. Hoffmann U, Fischereder M, Krüger B, Drobnik W, Krämer BK (2004) The value of N-acetylcysteine in the prevention of radiocontrast-induced nephropathy appears questionable. J Am Soc Nephrol 15:407-10
23. Hoffmann U, Banas B, Fischereder M, Krämer BK (2004) N-acetylcysteine in the prevention of radiocontrast-induced nephropathy: clinical trials and end points. Kidney Blood Press Res 27:161-6
24. Wagner HJ, Kalinowski M, Klose KJ, Alfke AH (2001) The use of gadolinium chelates for X-ray digital subtraction angiography. Invest Radiol 36: 257-65
25. Tervahartiala P, Kivisaari L, Kivisaari R, Cirtanen I, Standertskjold-Nordenstam CG (1991) Contrast media-induced renal tubular vacuolization. A light and electron microscopic study on rat kidneys. Invest Radiol 26: 882-7
26. Murphy KP, Szopinsky KT, Cohan RH, Mermillod B, Ellis JH (1999) Occurrence of adverse reactions to gadolinium-based contrast material and management of patients at increased risk; a survey of the American Society of Neuroradiology Fellowship Directors. Acad Radiol 6: 656-64
27. Katzberg RW, Sahler LG, Duda SW (1990) Renal handling and physiologic effects of the paramagnetic contrast medium Gd-DOTA. Invest Radiol 25: 714-719
28. Brillet G, Dubois M, Beaufils H, Bourbouze R, Eray G (1994) Renal tolerance of gadolinium-DOTA and gadolinium-DTPA in rats. Invest Radiol 29: 352-354
29. Gemery I, Idelson B, Reid S, Yucel EK, Pagan-Marin H, Ali S, Casserly L (1998) Acute renal failure after arteriography with a gadolinium-based contrast agent. Am J Roentgenol 171: 1277-8
30. Rieger J, Sitter T, Toepfer M, Linsenmaier U, Pfeifer KJ, Schiffl H (2002) Gadolinium as an alternative contrast agent for diagnostic and interventional angiographic procedures in patients with impaired renal function. Nephrol Dial Transplant 17: 824-28

Received: February 4, 2005 / Accepted: March 10, 2005

Address for correspondence:
Dr. med. Ute Hoffmann
Klinik und Poliklinik für Innere Medizin II
Klinikum der Universität
93042 Regensburg
Tel.: +49-941-9447301
Fax: +49-941-9447302
E-mail: Ute.hoffmann@klinik.uni-regensburg.de