Original Research

Effects of Gadopentetate Dimeglumine and Gadodiamide on Serum Calcium, Magnesium, and Creatinine Measurements

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Purpose: To investigate the in vivo effects of gadodiamide (Gd-DTPA-BMA) and gadopentetate dimeglumine (Gd-DTPA) on the laboratory measurements of serum calcium, magnesium, and creatinine.

Materials and Methods: Medical records from 1993 to 2004 were reviewed to identify inpatients for whom laboratory data were available regarding serum calcium, creatinine, and magnesium levels before and within one day after gadodiamide and gadopentetate dimeglumine enhanced MRI. Patients who underwent both gadolinium (Gd)-enhanced MRI and iodinated contrast-enhanced examinations on separate days within a six-month period were also identified to compare changes in serum creatinine.

Results: Serum creatinine did not increase in 2788 cases following gadopentetate dimeglumine and gadodiamide injection. By comparison, serum creatinine increased from 1.21 to 1.28 mg/dL following iodinated contrast, and there were 20 cases (2.6%) of contrast-induced nephrotoxicity ($P < 0.01$). Gadopentetate dimeglumine did not affect serum calcium or magnesium measurements. Following 1157 gadodiamide-enhanced examinations, measured serum calcium spuriously dropped from 8.65 to 8.33 mg/dL ($P < 0.0001$) and 34 patients had spurious critical hypocalcemia (<6 mg/dL). Of 60 patients with high-dose gadodiamide injection and renal insufficiency, 36.7% ($N = 22$) had spurious critical hypocalcemia immediately post MRI. In 216 patients with renal insufficiency, the mean serum magnesium level increased slightly from 1.69 to 1.77 mEq/L following gadodiamide injection ($P < 0.0001$).

Conclusion: Gd-based contrast agents are safe for MRI and MR angiography (MRA), and do not induce nephrotoxicity. However, gadodiamide interferes with serum calcium and magnesium measurements—particularly at high doses and/or with renal insufficiency.

Key words: gadopentetate dimeglumine; gadodiamide; calcium; magnesium; creatine
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THE EFFICACY OF GADOLINIUM (Gd) as an intravenous contrast agent for magnetic resonance imaging (MRI) and MR angiography (MRA) is well established in clinical practice for both anatomical and physiological assessments, as well as for diagnosing a wide spectrum of diseases. At approved doses, Gd-based paramagnetic contrast agents have a favorable safety profile (1–5). Gd chelates are considered to have no nephrotoxicity, and therefore they can be used at high doses in azotemic patients (6). Gd-enhanced MRI is preferred over iodinated contrast-enhanced computed tomography (CT) in patients who are at risk for developing renal failure. However, the in vitro interference between Gd chelates and the serum assays for calcium, magnesium, and other electrolyte measurements is well documented (7–12), and serum creatinine elevation following high-dose Gd administration has been reported (13–14). It is widely accepted that in patients with renal dysfunction, Gd chelates may linger in the serum for longer periods of time, thereby increasing the chance of toxicity or laboratory interferences. Gadodiamide is known to interfere with colorimetric assays for serum calcium measurements (package insert, Nycomed Amersham) (9–12).

At our hospital we have experience with both gadopentetate dimeglumine (from 9/22/1993 to 2/28/1997, and 8/18/2003 to 8/26/2004) and gadodiamide (from 3/1/1997 to 8/17/2003). The aim of this retrospective study was to investigate the possible in vivo effects of these FDA-approved Gd chelates on laboratory measurements of serum calcium, magnesium, and creatinine in a typical hospital-based population, and to determine the clinical importance of such effects.

MATERIALS AND METHODS

Patients and Data Collection

Medical records from 9/22/1993 to 8/26/2004 were reviewed to identify inpatients for whom laboratory data were available regarding serum calcium, creatinine, and magnesium levels before and within one day after Gd-enhanced MRI. This study was approved by the local institutional review board. Informed consent was not required. During this 11-year period, two different Gd-based contrast agents—gadodiamide (Gd-DTPA-BMA or Omniscan; Amersham Health, now GE

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Table 1
Serum Calcium, Magnesium, and Creatinine Measurements After Gadolinium Administration

	n	Ca pre	Ca post	*P* value	n	Mg pre	Mg post	*P* value	n	Cr pre	Cr post	*P* value
Gadodiamide												
Total	1157	8.65	8.33	<0.0001	1126	1.64	1.64	0.47	1157	1.19	1.20	0.51
High dose[a]	113	8.57	7.19	<0.0001	111	1.64	1.64	0.97	113	1.38	1.40	0.44
Creatinine > 1.5	217	8.52	8.19	<0.0001	216	1.69	1.77	<0.0001	217	3.27	3.33	0.35
Gadopentetate dimeglumine												
Total	1182	8.89	8.87	0.48	966	1.61	1.62	0.54	1631	1.24	1.21	0.005
High dose[a]	97	8.96	8.94	0.65	94	1.63	1.61	0.49	97	2.22	2.13	0.22
Creatinine > 1.5	188	8.83	8.83	0.99	166	1.68	1.68	0.80	254	3.62	3.49	0.01

[a]High dose is defined as contrast dose of > 0.1 mmol/kg.

Biosciences, Princeton, NJ, USA) and gadopentetate dimeglumine (Gd-DTPA or Magnevist; Berlex Laboratories, Monteville, NJ, USA)—were used. Patients who had undergone Gd-enhanced MRI were identified from MR logbooks, and data on each patient's age and gender, type and dose of Gd contrast administered, and the ending time of the MR examination were recorded. The Gd injection time was estimated to be 15 minutes before the end of the MRI examination. Gd contrast doses of >0.1 mmol/Kg were categorized as "high dose."

Electronic laboratory records were reviewed to obtain serum calcium, magnesium, and creatinine data. The measurement time was recorded by the phlebotomist at the time blood was drawn, and entered into the electronic record so that the intervals between Gd injection and laboratory measurement could be determined. In our institution the ortho-creso-pthalein (OCP) colorimetric assay was used for all total serum calcium measurements. This assay was performed on a Technicon Autoanalyzer (Tarrytown, NY, USA) between 1993 and 1998. After 1998 a Hitachi 747-100 analyzer (Roche Boehringer Mannheim, Indianapolis, IN, USA) was used, but the assays were identical. Serum calcium measurements of <6 mg/dL were considered to be "critical" values. Serum creatinine was measured on the same analyzers using Jaffe's method. An increase in serum creatinine level of >0.5 mg/dL (instead of >25% from baseline level) was defined as a significant change. The numbers of patients with a critically low calcium level and/or a serum creatinine increase of >0.5 mg/dL following Gd administration were recorded. Electronic medical charts of these particular cases were reviewed to determine possible reasons for changes in calcium or creatinine levels, as well as the impact of laboratory results on the clinical management of the patients. Our laboratory does not have critical values for magnesium, which is measured with the Calmagite assay.

Medical records were also reviewed to identify patients who had undergone both Gd-enhanced MRI and iodinated contrast-enhanced examinations, including computed tomography (CT) and X-ray digital subtracted angiography (DSA) on different days within six months before or after the Gd-enhanced MR study. Patients who exhibited changes in serum creatinine measurements before and within one day after iodinated contrast injection were investigated for comparison with the changes in serum creatinine following Gd contrast agents.

Statistical Analysis

Each Gd-enhanced MR examination was treated as an independent event. The statistical significance of changes in serum calcium, magnesium, and creatinine measurements following gadodiamide or gadopentetate dimeglumine administration was analyzed using a paired Student's *t*-test. In patients with renal insufficiency (serum creatinine level > 1.5 mg/dL), the statistical significance of the changes in calcium and magnesium levels following contrast administration was calculated by using Student's *t*-test. Similarly, the significance of the effect of high-dose Gd contrast administration on the laboratory results was calculated. The changes in serum creatinine following Gd-based and iodinated contrast media injections on different days were compared for all patients in both examinations, and for patients with renal insufficiency.

RESULTS

The changes in mean serum calcium, magnesium, and creatinine measurements following Gd-based contrast agent administration are listed in Table 1.

Serum Calcium

A total of 2339 examinations were identified as having serum calcium data before and within 24 hours after the MR examination. In 1182 MR examinations gadopentetate dimeglumine was administered at a mean dose of 17.3 mL, and in 1157 MR examinations gadodiamide was used at a mean dose of 17.6 mL.

The mean measured serum calcium level did not change significantly following gadopentetate dimeglumine administration (pre = 8.89 mg/dL; post = 8.87 mg/dL; $P = 0.48$). This indicates that gadopentetate dimeglumine does not interfere with OCP colorimetric serum calcium measurements. In 97 patients with high-dose injection (>0.1 mmol/kg body weight) and 188 patients with preexisting renal insufficiency, no change in serum calcium measurements following gadopentetate dimeglumine injections was observed ($P = 0.65$ and 0.99, respectively). A critical drop in the serum calcium level following gadopentetate dimeglumine administration was found in one case out of 1182 studies (<0.1%) in which a high dose (60 mL) was used for MRA. Reviewing the medical records of this individual

Table 2
Serum Creatinine in Patients Receiving both Gd and Iodinated Contrast Agents

	n	Pre	Post	Post-pre	*P*-value
Gadopentetate dimeglumine	447	1.29	1.26	–0.03	0.005
Iodinated contrast		1.25	1.33	0.08	<0.0001
Gadodiamide	321	1.15	1.17	0.02	0.28
Iodinated contrast		1.15	1.22	0.07	0.005
All gadolinium	768	1.27	1.25	–0.02	0.22
All iodinated contrast		1.21	1.28	0.06	<0.0001
With renal insufficiency (Cr > 1.5)					
Gadolinium	111	4.02	3.92	–0.23	0.27
Iodinated contrast		3.26	3.55	0.29	0.0004

did not identify any obvious explanation for hypocalcemia. The measurements were repeated and after two hours the calcium level returned to 7.8 mg/dL without any treatment, which suggests that this was a specimen/laboratory error.

Following 1157 gadodiamide-enhanced examinations, the mean measured serum calcium level dropped from 8.65 mg/dL to 8.33 mg/dL ($P < 0.0001$). An even greater decrease in measured serum calcium level was noticed after high-dose (>0.1 mmol/kg) injections ($N = 113$; pre = 8.57 mg/dL; post = 7.19 mg/dL; $P < 0.0001$) as well as in the patients with renal insufficiency ($N = 217$; pre = 8.52 mg/dL; post = 8.19 mg/dL; $P < 0.0001$). In 34 examinations (2.9%), measured serum calcium decreased by at least 2 mg/dL down to <6 mg/dL after gadodiamide injection at a mean dose of 37.4 mL. Since day-to-day changes in serum calcium are small (<1.9 mg/dL), a decrease greater than 2 mg/dL cannot be explained by random variation (12). No correcting analyses were performed. Ten patients with critical hypocalcemia post gadodiamide were treated with calcium (iv, $N = 8$; p.o.: $N = 2$). One of these patients developed atrial fibrillation immediately following the iv calcium, and the following day suffered a hemisphere stroke. Clinically significant interference was found in 19.5% ($N = 22$) of the patients who received a high-dose injection and 13.4% ($N = 29$) of the patients with renal dysfunction. In 60 patients with both high-dose injection and renal insufficiency, the incidence of significant serum calcium decrease was 36.7% ($N = 22$). Eight of these patients were on dialysis.

Magnesium

In our hospital the reference range for normal serum magnesium is 1.5–1.9 mEq/L. Serum magnesium data were available for 2092 MR examinations, 966 of which involved gadopentetate dimeglumine injection (mean dose = 18.1 mL) and 1126 of which involved gadodiamide injection (mean dose = 17.6 mL). There was no statistically significant change in the mean serum magnesium level following either gadopentetate dimeglumine (pre = 1.61 mEq/L; post = 1.62 mEq/L; $P = 0.54$) or gadodiamide (pre = 1.64 mEq/L; post = 1.64 mEq/L; $P = 0.47$). Magnesium levels did not change even after high-dose injections. However, in 216 patients with renal insufficiency, the mean serum magnesium level increased from 1.69 to 1.77 mEq/L following gadodiamide injection ($P < 0.0001$). The number of patients with renal failure who received gadopentetate dimeglumine was 266, and in this population the change in the mean serum magnesium level was not statistically significant ($P = 0.80$). Serum magnesium increased from normal to a level that was above the normal range in 122 examinations (52 after gadopentetate dimeglumine ($P < 0.01$) and 70 after gadodiamide ($P < 0.01$) injection). However, since there is no critical value for serum magnesium, no doctors were called as a result of the spuriously elevated magnesium.

Creatinine

Pre- and post-MRI serum creatinine data were available for 2788 examinations. The mean gadopentetate dimeglumine dose used in 1631 examinations was 16.6 mL. The mean gadodiamide dose administered in 1157 examinations was 17.5 mL. MR examinations were identified in patients who had also undergone iodinated contrast injection on a separate day and had serum creatinine data before and after both contrast-enhanced examinations. Pre- and post-study serum creatinine data for both studies were available for 768 subjects (Table 2). This allowed us to directly compare Gd and iodinated contrast effects on serum creatinine while controlling for patient variability.

The mean serum creatinine level dropped from 1.24 mg/dL to 1.21 mg/dL after gadopentetate dimeglumine injections ($P = 0.005$). In 97 examinations with high-dose gadopentetate dimeglumine injection, the drop in mean serum creatinine level was even greater (from 2.22 mg/dL to 2.13 mg/dL); however, it was not statistically significant ($P = 0.22$). In 254 examinations in patients with renal insufficiency, there was a transient decrease in serum creatinine following gadopentetate dimeglumine injection (pre = 3.62 mg/dL; post = 3.49 mg/dL; $P = 0.01$).

Following gadodiamide injection, there was no change in the mean serum creatinine measurements (pre = 1.19 mg/dL; post = 1.20 mg/dL; $P = 0.51$), even with high doses (pre = 1.38 mg/dL, post = 1.40 mg/dL; $P = 0.44$) or in patients with renal insufficiency (pre 3.27 mg/dL, post = 3.33 mg/dL; $P = 0.35$).

In 768 cases with both contrast-enhanced MR and iodinated contrast-enhanced examinations, the mean serum creatinine level was unchanged after Gd contrast administration (pre = 1.27 mg/dL; post = 1.25 mg/dL; $P = 0.22$) but significantly increased after iodinated contrast injection (pre = 1.21 mg/dL; post =

Table 3
Creatinine Increase ≥ 0.5 mg/dL in Patients Receiving both Gd and Iodinated Contrast

Contrast	n	%
Gadopentetate dimeglumine	0	0
Gadodiamide	0	0
Iodinated[a]	20	2.6

[a]Note that patients at risk for contrast induced nephrotoxicity usually did not receive iodinated contrast.

1.28 mg/dL; $P < 0.0001$). An increase (≥0.5 mg/dL) in serum creatinine was identified after 17 Gd administrations. A review of the medical charts revealed that 13 of 17 MR studies were performed on hemodialysis patients. One patient was identified to have leukemia and was receiving chemotherapy, which was considered the most likely reason for renal impairment. In the remaining three patients, the creatinine level was already rising before contrast-enhanced MRI was performed. Thus, there were no patients for whom Gd injection was believed to have caused renal insufficiency. The mean serum creatinine level dropped from 1.27 mg/dL to 1.25 mg/dL following Gd administration ($P = 0.005$), whereas it increased from 1.21 mg/dL to 1.28 mg/dL ($P < 0.0001$) following iodinated contrast administration. In 111 patients with renal insufficiency, the increase in the mean serum creatinine level was 0.27 mg/dL within the first 24 hours following iodinated contrast administration ($P = 0.0004$). After iodinated contrast injection, 36 patients experienced a creatinine increase greater than 0.5 mg/dL (Table 3) despite hydration and other efforts to minimize the iodinated contrast nephrotoxicity. Sixteen of these patients were on dialysis. Therefore, the creatinine increase can only be explained by iodinated contrast injection in 20 cases (2.6%).

DISCUSSION

The data from 2788 Gd-enhanced MR examinations confirm that gadodiamide can interfere with serum calcium and magnesium assays. However, there is no evidence that gadodiamide or gadopentetate dimeglumine induced nephrotoxicity. Indeed, for gadopentetate dimeglumine, there was a transient decrease of mean serum creatinine, which was a statistically significant difference compared to gadodiamide. The amount of saline flush in Gd-enhanced MRI is typically too small to have a diuretic renal effect. This slight difference in effect on creatinine may be explained by some difference between the two compounds. One difference is that gadopentetate dimeglumine is ionic, whereas gadodiamide is non-ionic. This difference relates to the presence of amino groups in gadodiamide, as opposed to carboxyl groups in gadopentetate dimeglumine. Another important difference is that gadopentetate dimeglumine has a stronger chelate thermodynamic stability with less in vivo release of free Gd cations.

Although there are case reports of rising serum creatinine levels following Gd injection at high doses (14), many studies have demonstrated a favorable safety profile for Gd-based contrast agents, even at high doses and in patients with renal insufficiency (3,15–17). Most of the high-dose studies involved patients with vascular disease. These patients often already have some deterioration in their renal function. For example, diabetic patients commonly have atherosclerotic disease affecting the larger arteries (e.g., the aorta, carotid artery, coronal artery, or peripheral arteries) as well as microvascular disease affecting the renal parenchyma. In such patients, X-ray DSA or CT angiography with iodinated contrast agents should be avoided as long as possible to prevent further damage to the kidneys. Some authors have reported using Gd instead of iodinated contrast during interventional procedures, and verified the safety of Gd (2,18–20). However, the off-label use of Gd is not universally recommended (21). The sensitivity and specificity of MRA in diagnosing vascular diseases are approaching the accuracy of standard DSA in diagnosing large vessel stenoses. Therefore, it is very important to be familiar with the side effects and adverse reactions of Gd-based contrast media. A limitation of this study is the lack of detailed documentation about the type and dose of the iodinated contrast media used in the patients of our study population. Nevertheless, the data are compelling that Gd has significantly less nephrotoxicity than iodinated contrast.

It is well documented that Gd-based contrast agents can show both positive and negative interference with cation assays. The affected analytes include angiotensin-converting enzyme (ACE), calcium, iron, magnesium, total iron binding capacity (TIBC), and zinc. In a previous in vitro study of gadodiamide, gadopentetate dimeglumine, gadoversetamide, and gadoteridol, two of these contrast agents—gadodiamide and gadoversetamide—had negative interference with colorimetric assays for serum ACE, calcium, and zinc; positive interference with magnesium and TIBC assays; and both positive and negative interference with iron assays (7).

This study confirms prior observations regarding the interference of gadodiamide contrast agents with clinical chemistry tests, particularly colorimetric calcium assays (7,9,10), which are used at many (but not all) hospitals. Compared to gadopentetate dimeglumine, gadodiamide produces clinically significant negative interference with colorimetric assays for serum calcium measurements. The incidence of false "critical" calcium drops is higher in patients with high-dose injections and/or renal insufficiency compared to those with low-dose injections and normal renal function. In 22 of 60 patients (36.7%) with renal insufficiency who received high-dose gadodiamide injections, the colorimetric serum calcium measurement dropped to below 6 mg/dL, the critical value for which chemistry laboratory technicians are required to stat page the ordering physician for an immediate clinical investigation into this potentially life-threatening condition. Another problem is that patients with hypercalcemia (e.g., due to occult malignancy) may be missed due to gadodiamide interference causing the serum calcium to drop into the normal range.

This spurious hypocalcemia is likely caused by dissociation of Gd ions from their ligands and complexation with the chromophore (OCP) used in the calcium

assay. This interference is proportional to the Gd chelate concentration in the plasma sample (12). It is maximized by factors that lead to a high concentration of gadodiamide in the blood for extended periods of time, including high doses and use in patients with renal dysfunction, who may require several days to eliminate the gadodiamide from the circulation.

The likely reason for magnesium interference is the alkaline conditions of the Synchron LX-20 assay, which catalize release of Gd from the chelator and allows association between the Gd^{++} ions and the chromophore. This creates a colored complex that resembles the magnesium-chromophore complex, resulting in a positive interference. Our data show that the interference with this serum magnesium assay is minor and could only be demonstrated statistically for patients with renal insufficiency. This finding is consistent with previous results from an in vitro study (7). Gadopentetate dimeglumine did not interfere with magnesium measurements, consistent with its higher thermodynamic stability with stronger binding of the Gd to the chelator.

In conclusion, although all Gd chelates are considered to have a similar and favorable safety profile, we observed a clinically significant difference between gadodiamide and gadopentetate dimeglumine regarding the commonly used laboratory assays. Gadodiamide is more likely to produce significant interference with laboratory measurements. Of all of the affected analytes, calcium measurement interference is the most likely to lead to unnecessary treatment and adverse effects on patient care. If the measured serum calcium levels are important for actual clinical management and follow-up in particular cases, an agent other than gadodiamide should be preferred for MR studies; otherwise, a non-OCP colorimetric method or ionized calcium measurement must be utilized to eliminate the possibility of mismanagement. It is also important to recognize potential interference with analytes other than calcium and magnesium, such as ACE, iron, TIBC, and zinc in serum samples from patients who have recently undergone Gd-enhanced MRI. We attempted to examine all laboratory measurements pertaining to positive cations that are similar to Gd. However, serum tests for zinc, iron, TIBC, and ACE were performed so infrequently that it was impossible to obtain a statistically meaningful data pool. Thus, we can conclude that interference with these tests is very rare.

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