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International Journal of Radiology & Radiation Therapy

Research Article
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Characteristics of patients with self-described gadolinium deposition disease

Abstract

Background: Gadolinium deposition disease (GDD) is a recently described entity that has not yet achieved wide recognition in the medical community.

Introduction: To evaluate individual characteristics in patients with self-diagnosed GDD.

Material and Methods: An anonymous survey was posted on websites hosted by self-described gadolinium toxicity groups. Descriptive statistical analyses were performed.

Results: 161 subjects were evaluated (125 females; age: 50.4± 13.2yrs). The age at onset was 46.5±12.8yrs. There were significant differences between genders (p<0.0006). The majority (83.2%) were White, genetic Central/Northern European. The presence of gadolinium was primarily confirmed by urine tests (95.6%). Before the first GBCA-enhanced MRI, 14.3% had been diagnosed with an autoimmune disease, increasing to 32.3% after the presumed diagnosis of GDD. The majority (70.1%) received multiple injections of GBCAs. In these, symptoms developed after an earlier injection, and overall symptoms became progressively worse following additional administration. The most frequent agent in subjects with 1 GBCA injection was Gadavist (n=17). In cases with multiple injections, the same agent was administered in 17 subjects, and the most frequent was Gadavist (n=8).

Conclusions: All GBCAs may cause the disease. Most participants were White females, and an increased autoimmune disease association exists. GDD may develop after single or multiple GBCA administrations, and the disease worsens with each successive administration. Recognizing these patients is critical to avoid further GBCAs and consequent disease worsening.

Keywords: gadolinium deposition disease, gadolinium, gadolinium-based contrast agents, toxicity, survey

Volume 12 Issue 6 - 2025

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Received: November 07, 2025 | **Published:** November 18, 2025

Introduction

To date, nephrogenic systemic fibrosis (NSF) represents the only well-established clinical entity related to the toxic effects of gadolinium. It is limited to patients with severe renal failure, in whom most have received a linear gadolinium-based contrast agent (GBCA) with lower chemical stability.[1] Although the entity had been initially reported in 1997.[2], it took almost a decade to clarify an association with GBCAs.[3,4]

A novel form of gadolinium toxicity has been described for at least eight years despite normal/near-normal renal function. This new clinical entity related to GBCA administration was termed "Gadolinium deposition disease" (GDD).[5-8] Criteria that have been proposed for this entity include 1. Symptom development shortly after administration of GBCA (often within minutes but up to one month); 2. Evidence of gadolinium presence in the body beyond 30 days; 3. Symptoms that are new and not previously experienced by patients (*i.e.*, no symptoms that could be explained by pre-existing conditions); 4. clearly defined symptoms (most often include clouded mentation/brain fog, skin and skin-substrate burning, pins and needles sensations, bone pain, and glove and sock distribution of skin discoloration, pain, or tissue thickening).[5-10]

In initial descriptions, clinical features showed consistency among patients. Recently other authors have proposed another designation called Symptoms Associated with Gadolinium Exposure (SAGE), acknowledging that a reaction to GBCA exists.[11,12] Recent investigations have been published regarding therapy with the currently most stable available chelator DTPA,[13,14] measurement of cytokines,[15-17] and antimitochondrial lab findings.[18] in GDD patients. Nevertheless, to date, no study has described the demographic characteristics of these patients. Therefore, the present study is intended to ascertain whether certain demographic features or factors existed to identify whether particular patient populations may be more susceptible to acquiring the disease. To generate meaningful data, the study relied on group leaders of the various online forums of alleged sufferers of presumed GDD to reach out to their members to participate in a survey.

Material and methods

The survey was anonymous, and all participants were recruited online from gadolinium toxicity support groups. All participants in the survey were informed of the purpose of the study.

The survey was posted using an online survey development cloud-based software (SurveyMonkey.com, San Mateo, CA).[19] An electronic link to the survey was posted to online support groups in North America and Europe.

The survey comprised 20 open questions accessible over 20 consecutive days (Appendix A). Instructions were given that individuals were only to take the survey once if they belonged to more than one advocacy group, and this was judiciously checked with the survey supervisor. Participants had the option to leave any questions unanswered.

Statistical analysis

Descriptive statistical analyses were performed to characterize the data and determine the responses' frequencies, including

demographics, kidney function, autoimmune disorders, number and types of GBCAs, tissue tests, sites and patterns of pain, and other somatic reactions and/or changes. The student *t*-test for independent samples was used whenever appropriate.

These calculations were performed using StatsToDo open-source Software.[20]

Results

A total of 183 subjects responded to the survey. For study entry, inclusion required the confirmation of gadolinium in fluids or tissues beyond one month of the GBCA administration and the presence of normal/near-normal kidney function. Seventeen subjects did not confirm the presence of gadolinium, 6 of these had pre-existent decreased kidney function, and five others declared low self-described kidney function. Thus these 22 subjects were excluded from this study.

Our final population included 161 subjects. One hundred and twenty-five subjects (77.6%) were females (age: 52.3± 12.2 yrs.), and 36 (22.4%) were males (age: 44.1 ± 13.2 yrs.). There were statistical differences between genders ($p<0.0006$). The age at onset of GDD was 48.3 ± 12.2 yrs. and 40.1 ± 14.9 yrs. for females and males, respectively.

Concerning racial identity/ancestry, the majority was White, genetic Central/Northern European (n= 134; 83.2%), and White, Southern European (includes Hispanic) was the second most common (n=15; 9.3%). The remaining 7.5% represented occurrences in other races.

Concerning the fluid/tissue tests that found the presence of gadolinium > 1 month after administration of a GBCA, 154/161 subjects had urine tests, 24/161 had blood tests, 14/161 had skin/dermal tissue tests, and 10/161 were additionally tested for other tissues that showed the presence of gadolinium.

Kidney function was normal/near-normal (eGFR >60 mL/min/1.73m^2) in the majority of the respondents (145/161, 90.0%), while 16/161 (9.9%) subjects were uncertain of their kidney function status but unaware of personal kidney disease.

There was a strong family history of autoimmune disease in 28 subjects (17.4%). Before the first MRI with contrast, 23 subjects (14.3%) had been diagnosed with an autoimmune disease, while the diagnosis of autoimmune disease increased to 52 subjects (32.3%) after GBCA-enhanced MRI (Table 1).

Table 1 Autoimmune disease list

Autoimmune disease	Number of subjects
Psoriasis	4
Lichen Planus	2
Crohn's Disease	2
Celiac Disease	5
Multiple Sclerosis	4
Sjögren's Syndrome	5
Fibromyalgia	11
Hashimoto's Thyroiditis	12
Guillain-Barré Syndrome	1
Vitiligo	1
Chronic Lyme Disease	4
Sarcoidosis	1
Lupus	3
Antiphospholipid Syndrome	1
Raynauds	1
Rheumatoid Arthritis	3
Wegener's Granulomatosis	1
Amyloidosis	1
Pernicious Anemia	1
Alopecia Areata	1
Systemic Sclerosis	1

Note: 8 subjects had multiple autoimmune disorders.

Twenty-six subjects (16.1%) had pre-existent or developed after GBCA injection allergies/reactions to other metals. One hundred thirteen subjects had no allergies/reactions to other metals, and in 22 subjects, responses were unclear.

The majority of subjects (n=113) had received more than one injection (70.1%), and 48 (29.8%) received only one injection of GBCA. Each injection of GBCA reflected that they obtained follow up MRIs which included another GBCA injection. Enumerating data on an injection-by-injection basis: 48 subjects (29.8%) received one injection; 14 subjects (8.7%) received 2; 24 subjects (14.9%) received 3; 18 subjects (11.2%) received 4. Noteworthy, 10 subjects (6.2%) received between 11-15 injections of GBCA; 2 subjects (1.2%) received between 16-20 injections; 5 subjects (3.1%) received between 21-25 injections; and 4 subjects (2.5%) received >25 injections (Table 2).

Table 2 Number of GBCA injections

Number of injections	Number of subjects
1	48
2	14
3	24
4	18
5	9
6	7
7	3
8	5
9	7
10	5
11 - 15	10
16 - 20	2
21 - 25	5
> 25	4

GBCA, Gadolinium-based contrast agent

GBCA, Gadolinium-based contrast agent

In subjects who had received multiple injections, individuals considered that the disease arose after the first injection in 35 subjects (30.7%), after the second in 18 (15.9%), the third in 16 (14.1%), and fourth in 8 (7%). It was unusual for initial symptoms to arise beyond the sixth injection (Table 3). In 89 of the 113 (78.7%) individuals who received multiple injections, symptoms developed after an earlier injection than the last reported. In all of these 89, overall symptoms worsened after each additional administration.

In subjects who had 1 GBCA injection, the most frequent agents were Gadavist (n=17), Magnevist (n=8), and Dotarem (n=7). The other agents were Omniscan (n=3), ProHance (n=2), MultiHance (n=3) and Eovist (n=1). Seven respondents did not know the brand of the GBCA.

Citation: Semelka RC, Ramalho M. Characteristics of patients with self-described gadolinium deposition disease. *Int J Radiol Radiat Ther.* 2025;12(6):142–146. DOI: 10.15406/ijrrt.2025.12.00437

Table 3 Onset of the disease in subjects with multiple injections

Onset of the disease (# GBCA injection)	Number of subjects
1	35
2	18
3	16
4	8
5	9
6	6
7	0
8	1
9	1
10	2
11 to 15	-
16 to 20	-
21 to 25	-
> 25	-
Unsure	17

GBCA, Gadolinium-based contrast agent

GBCA, Gadolinium-based contrast agent

In cases with multiple injections of contrast, the same agent was administered for each MRI in 17 subjects. Magnevist (n=4); Gadavist (n=8); Dotarem (n=1); MultiHance (n=2); ProHance (n=1); Uncertain (n=1). Sixty-seven subjects had different GBCAs, of which 29 did not know all the administered agents.

One hundred and sixteen subjects (72%) sought medical treatment within the first month after the GBCA administration. (21 subjects (13%) on the same day; 49 (30.4%) in the first week; and 46 (28.5%) > one week and < 1 month). Five subjects (3.1%) did not seek medical treatment.

Discussion

Several interesting features were observed in the subjects who participated in the survey. The highest disease frequency was seen in females (nearly 78%), concurring with earlier smaller surveys.[6,8] Semelka et al.,[6,7] had previously opined with their earlier experience with this disease that it shared features with acute hypersensitivity reactions to GBCAs. Both appear to be more commonly observed in females.[20,21] Most of the subjects that self-described the disease were genetic White Central/Northern Europeans. This very high occurrence in central and northern European origin is similar to other diseases, such as Genetic Hemochromatosis.[22,23] It may suggest a related genetic susceptibility for the development of the disorder. A higher occurrence in females is also seen in other diseases, such as fibromyalgia.[24] A recent investigation postulated fibromyalgia might be an autoimmune disorder.[25] Interestingly, the symptoms of GDD are comparable to those of fibromyalgia.[24] One paper.[26] presented a case of fibromyalgia that emerged after exposure to GBCAs and exacerbated after additional exposures, which we suppose might be a case of GDD. Moreover, a recent report hypothesizes the relation of fibromyalgia and unexplained chronic widespread pain reported after trauma, surgery, or medical illness to the retention of gadolinium in the body.[27] We also believe that GDD may appear as a fibromyalgia-like disease triggered by Gd exposure; however, this should be verified in future investigations.

In this survey, 14.3% of subjects described that they had a pre-existent autoimmune disease before developing the symptoms, which increased to 32.3% after GBCA-enhanced MRI. By definition, autoimmune disease arises from an abnormal immune response to a normal body part. In comparison to our population, approximately 7% (24 million) to 15% (50 million) of people in the United States are affected by autoimmune diseases.[28-30] The cause of autoimmune disease is generally considered unknown. Some autoimmune diseases have a genetic basis, and individual cases may be triggered by infections or other environmental factors.[31] Our assumption regarding the much higher incidence of autoimmune diseases in our subjects than in the general population is that subjects with presumed GDD may also have a higher susceptibility to autoimmune conditions, and the two may be related. As observed in this study on GDD, autoimmune diseases demonstrate a higher prevalence in females (7.1% for females vs. 3.0% for males).[32] Based on this initial study, we cannot conclude that the possible development of GDD is associated with or can cause other additional autoimmune conditions; however, it is worth further investigation. Another explanation for the increase in diagnoses of autoimmune diseases, especially when reported after the development of presumed GDD, may rely on the closer medical evaluation of these patients after the development of symptoms, which may have uncovered an autoimmune condition that had already been present. Another possibility is that the GBCA could represent the environmental factor that elicited the autoimmune disease.

In our population, 26 subjects (16.1%) had a history of or developed hypersensitivity to other metals. This is only marginally greater than reported in the general population, ranging from 10% to 15%.[33,34] Metal hypersensitivity is a common immune disorder, and it has been reported that some heavy metals, such as gold, cadmium, and mercury, may also induce human autoimmune diseases.[35]

The findings of this study should not be misconstrued to suggest that GBCAs should be avoided in all patients, as GBCAs remain vital for many MRI studies. It should also be recognized that alternative contrast agents might bear their own adverse reactions, many not yet known. Our opinion is that at this stage, the focus should be on: i) the subjects at risk for possibly developing GDD, ii) methods to avoid getting the disease, and iii) treatment methods for individuals with the disease.

As observed in earlier surveys,[6,8] a sizable percentage of presumed GDD arose from the single GBCA-enhanced MRI study that subjects underwent, which in the present study was approximately 30%. A novel critical observation is that many subjects acquired disease after their second, third, and fourth GBCA-enhanced study, having undergone multiple GBCA-enhanced studies. However, subjects continued to be sent by their physicians for additional GBCA-enhanced MRI exams. Their disease progressed in severity with each additional study in 78.7% of the cases. Another crucial point of this survey is that 72% of subjects sought medical advice and treatment within the first month following the GBCA injection. Therefore, we believe that physicians must be aware of the existence of GDD and its clinical features. If nothing else is done for them, it is important that sufferers not undergo additional GBCA-enhanced MRI to investigate what may well be GDD. This may be the simplest and most important aspect of managing this disease: when symptoms develop, the patient should not receive additional GBCAs on future MR examinations, as sufferer's report that symptoms get progressively worse with each successive GBCA injection.

One critical aspect of previous studies is that patients with GDD develop flares after chelation with DTPA. This is attributed to the re-ignition of more severe symptoms due to a host immune response to the remobilization of Gd into the vascular circulation.[13-17] Furthermore, individuals who do not have GDD but have received previous

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GBCA injections do not experience a flare reaction when chelated with DTPA.[17] The finding that with each successive GBCA injection following the development of GDD, the patient worsens also support causation. That chelation with an effective chelator (DTPA) causes a flare reaction, aligns with the most critical Bradford Hill criterion for causation: when the agent is given again, symptoms worsen. Few drugs meet this most critical of criteria, because in most cases to knowingly give a patient again a drug that causes severe reaction is unethical. The process of chelation remobilizes Gd from tissue to be eliminated by the kidneys, so in essence this creates the effect that a patient experiences the drug again. Furthermore, individuals who do not have GDD but have received GBCA do not experience flare reactions when chelated with DTPA.[17,36] Our reported findings herein show that with each successive GBCA injection following the development of GDD, the patient becomes worse, directly supporting causation.

Our study showed that all GBCAs appear to have caused symptoms in patients complaining of GDD. Of solitary agents use (unconfounded) GBCA injections, most were macrocyclic agents. This finding differs from the initial studies,[8] in which the GBCAs were predominantly linear agents. Also, a recent paper from Semelka et al.,[36] and a paper from Shahid et al.,[12] reported that MultiHance showed a high rate of SAGE/GDD. Our opinion of the high representation of macrocyclic agents may reflect the increasing usage trend of this class of GBCA rather than linear agents, because of stability concerns, and not that macrocyclic agents are more likely to cause GDD.

The risk posed by the GBCA chemical structure (linear versus macrocyclic) in GDD appears different from that observed for NSF, where the latter occurs primarily following linear agents.

Concerning risk among the macrocyclic agents, Semelka et al.,[36] reported in an earlier study that Gadavist had the highest occurrence of GDD, which concurs with the results from this present survey. It may be that the higher concentration of Gd in the formulation of Gadavist compared to ProHance and Dotarem accounts for the higher rate of occurrence. Gadavist has double the gadolinium concentration compared with the other macrocyclic agents, which might account for the greater immunogenicity of Gadavist. We caution that some differences in GDD occurrence rates may reflect market share. This may reflect the low incidence of other linear agents causing GDD, such as Magnevist, Omniscan, and OptiMARK. Our opinion of why macrocyclic GBCAs result in GDD, even though the agent is almost certainly fully intact, is that it reflects the action of different immune cells that are principally responsible for NSF, which are CD34+ circulating fibrocytes. Acute hypersensitivity reactions (AHR) are experienced by all GBCAs, and this reaction typically arises within minutes to hours of GBCA injection, when even the least stable GBCAs are likely fully intact,[36] so disassembly of a GBCA is not feature of all toxic reactions. Previous studies.[15-17] support that T cells are the primary immune cells that elaborate cytokines and are the primary cells involved with GDD. Our current opinion is that the immune response of those who have already been administered a GBCA is a complex mixture of proinflammatory, inflammation-suppressing, and communication cytokines, and the proportions of each, and their change in concentration over time, reflect whether the patient has GDD or not, and the severity of GDD.[36]

A principal explanation for insufficient recognition of the disease to the present time is primarily because the patients have near-normal renal function. In some ways ironic, the earlier description of NSF has hampered physicians' preparedness to recognize GDD. The current common supposition by physicians is that since the patient has normal renal function, they cannot have a GBCA-related disease. GDD parallels heavy metal toxicity from other heavy metals, and it is NSF which is apparently unique to Gd. Although the primary feature of renal failure is prolonged retention of the GBCA, there must be additional contributing biochemical aspects, as yet not fully elucidated for NSF.[37,38]

Previous articles skeptical of GDD emphasized the concern that this entity may result in major medical malpractice lawsuits. Some editorials imply that this could be the primary motivator of the sufferers.[39] Nevertheless, in a recent study, several allopathic physicians have described the disease themselves, and they do not appear to be malingering or litigious.[40]

There are some limitations of our study. One limitation is that only individuals that have visited self-described gadolinium toxicity group websites were invited to participate in the survey. This limited outreach introduced a bias in the study; therefore, definite conclusions about the race and gender characteristics of patients with assumed GDD cannot be made. One should consider the possibility that the very high female preponderance and high central and northern European ancestry may represent that women are more likely to respond to surveys. Our 9 year clinical experience of treating patients with GDD with DTPA-chelation therapy shows similar demographics, with the majority being Genetic White central European women. We have not yet analyzed patients in detail in our clinical practice, and we anticipate performing this in the coming years. Another limitation is the use of patient self-reporting of disease. In the present environment of computer web-based medical management of subjects, this approach is now standard, and has been previously employed for this disease.[6,8,40] and other disease processes. Other researchers have concurred with our impression that this is an important modern trend in health care of greater patient involvement in their management.[41,42] Finally, the survey did not inquire as to the health status of individuals prior to undergoing MRI with GBCA, and the extent of pre-existent wellness or disease may have influence on the findings.

In summary, our survey-based study results suggest that most individuals who GDD are White genetic central/northern European females with an increased autoimmune propensity. GDD sufferers who have undergone multiple GBCA-enhanced studies develop disease not infrequently after the first or early in a series of multiple GBCA administrations. The disease worsens with each successive administration. Perhaps the most straightforward approach to minimizing the deleterious health impact of GDD in the population, both its frequency and severity, is the early recognition of the disease following a GBCA injection and avoiding further GBCAs in this group of individuals.

Acknowledgments

None

Conflicts of Interest

The authors have no conflicts of interest to declare.

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Citation: Semelka RC, Ramalho M. Characteristics of patients with self-described gadolinium deposition disease. *Int J Radiol Radiat Ther.* 2025;12(6):142–146. DOI: 10.15406/ijrrt.2025.12.00437