

Safety of Intra-articular Hip Corticosteroid Injections

A Matched-Pair Cohort Study

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Background: Recent studies have suggested there is an increased risk of avascular necrosis (AVN), subchondral insufficiency fracture (SIF), femoral head collapse, and osteoarthritis (OA) progression in the 12-month period after hip corticosteroid/anesthetic injection (CSI); however, these studies have failed to account for preinjection OA severity or preexisting AVN/SIF.

Purpose: To compare these complication rates in patients treated with versus without hip CSI, while minimizing the aforementioned forms of selection bias present in previous investigations.

Study Design: Cohort study; Level of evidence, 3.

Methods: For all patients who had undergone a single hip CSI and hip magnetic resonance imaging (MRI) within the preceding 12 months at a single institution (CSI cohort), 2 musculoskeletal radiologists retrospectively graded OA severity (modified Kellgren-Lawrence classification) and femoral head collapse on hip radiographs taken within 12 months before, and 1 to 12 months after, CSI. Using identical methodology, radiographs from a control cohort (composed of hips that had never undergone CSI and had undergone hip MRI with hip radiographs taken within 12 months before, and 1-12 months after, MRI) were also graded. The cohorts were matched for age, sex, body mass index, and OA severity. Readers were blinded to cohort and time point. OA progression was defined as an increase in modified Kellgren-Lawrence grade ≥ 1 between radiographs.

Results: Included were 141 matched pairs. After exclusion of 48 matched pairs with at least 1 incidence of preexisting AVN or SIF on index MRI, CSI (n = 93; mean time between CSI and final hip radiograph, 5.43 months) and control (n = 93; mean time between MRI and final hip radiograph, 4.87 months), groups did not significantly differ in rates of OA progression (3.2% vs 3.2%) or new femoral head collapse (3.2% vs 2.2%).

Conclusion: In contrast to the findings of recent retrospective investigations, we did not find that patients treated with hip CSI had significantly higher rates of short-term OA progression or femoral head articular surface collapse after controlling for baseline OA severity and preexisting AVN or SIF. Future randomized trials investigating safety of hip CSI are needed to determine its exact short-term risk profile.

Keywords: avascular necrosis; corticosteroid injection; hip osteoarthritis; osteonecrosis of the femoral head; subchondral insufficiency fracture

Intra-articular corticosteroid/anesthetic injection (CSI) is a common treatment for osteoarthritis (OA) of the hip. It has been shown to have significantly greater short-term pain and functional improvements over placebo in multiple randomized controlled trials.^{2,13,17,23} However, there are relatively few high-level studies that investigate complications of hip CSI. In vitro studies have shown that single doses of both local anesthetic⁵ and corticosteroids⁶ result in

significant chondrocyte cytotoxicity; however, this has not yet been shown clinically. Two placebo-controlled, randomized controlled trials have investigated the effect of knee CSI on articular cartilage and have demonstrated conflicting results: Raynauld et al¹⁸ found no difference in radiographic arthritis at 12 and 24 months between the CSI and placebo groups, whereas McAlindon et al¹⁴ found that the CSI group had 0.11 mm more cartilage thinning on magnetic resonance imaging (MRI) relative to the placebo group at 24 months. Although MRI better detects cartilage thinning than does radiography,^{9,11} no minimal clinically important difference in MRI-determined cartilage thinning

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has been established. Furthermore, the cartilage thinning observed by McAlindon et al is less than one-third the thinning represented by a progression from Kellgren-Lawrence (KL) grade 2 to grade 3,²⁴ suggesting that this observed difference between cohorts may lack clinical significance.

In the absence of definitive level 1 evidence regarding the safety of hip CSI, there have been an increasing number of retrospective case series in the radiology literature studying outcomes in the 12-month period after intra-articular hip injection. Two recent studies by Simeone et al²⁰ and Kompel et al¹² have suggested that intra-articular steroid injection into the hip joint may be associated with increased rates of avascular necrosis (AVN), femoral head collapse (up to 17% in 1 study²⁰), subchondral insufficiency fracture (SIF), accelerated progression of OA (up to 44% in 1 study²⁰), and rapidly progressive OA (RPOA) within 12 months of injection. However, these studies had several limitations, the most serious being the failure to compare against a control group matched for baseline OA severity.¹ Additionally, neither study excluded patients with preinjection AVN or SIF from analysis.

The purpose of this study was to compare short-term radiographic OA progression, the development of AVN or SIF, and the development of new femoral head articular surface collapse in patients treated with CSI to a control group matched for OA severity; at the same time, patients with preexisting AVN/SIF were excluded from analysis. We hypothesized that these complication rates would not differ significantly between CSI and the control groups and that CSI complication rates would be lower than reported previously.^{12,20}

METHODS

This study was exempt from institutional review board approval. We performed a retrospective query of all fluoroscopic-guided hip CSIs performed at our institution between May 2007 and December 2019. The inclusion criteria were hip MRI within 12 months before CSI (to assess for preexisting AVN or SIF), preinjection hip radiographs (including weightbearing anterolateral pelvis and/or hip radiographs and lateral hip radiographs) within 12 months before CSI, and postinjection hip radiographs 1 to 12 months after CSI.

A control group was composed of hips that had never undergone CSI but that had undergone MRI at our institution, pre-MRI hip radiographs taken within 12 months before MRI, and post-MRI hip radiographs taken 1 to 12 months after MRI. In both groups, hips diagnosed with

septic arthritis before the index procedure (hip injection for the CSI group and MRI for the control group) were excluded. The control group was 1:1 propensity matched for age, sex, body mass index (BMI), and OA severity (mild vs moderate vs severe OA on pre-MRI hip radiograph reports) with exact matches for sex and OA severity. If a hip underwent more than 1 MRI or preinjection radiograph in the 12-month period before CSI, the MRI or radiograph taken closest to the date of CSI was selected for interpretation in this study. In contrast, if a hip underwent more than 1 postinjection radiograph within 12 months of CSI, the one taken closest to 12 months after CSI was selected for interpretation in this study. We chose to include MRIs up to 12 months before CSI in this study because AVN has been shown to be identifiable on MRI many months before the onset of symptoms in certain patients.^{21,22} In addition, we chose to interpret radiographs taken 1 to 12 months after CSI because the aforementioned case series by Simeone et al²⁰ and Kompel et al¹² have focused on short-term complications occurring in the 12-month period after hip CSI.

BMI and age at the time of index procedure were collected from the medical record. MRI reports were reviewed for each patient in both groups to record the presence or absence of preexisting AVN or SIF. Matched pairs with at least 1 incidence of preexisting AVN or SIF were excluded for subgroup analysis.

Radiographs before and after the index procedure in each group were reviewed on a picture archiving and communication system (PACS; Bernex) independently by 2 musculoskeletal radiologists with more than 10 years of experience each (K.M.S. and N.S.). A third reader with more than 10 years of experience (L.S.B.) arbitrated discrepancies. All readers were blinded to group (CSI vs control) and time point (before vs after). The severity of OA was assessed using a modified KL classification system used in a similar study by Simeone et al²⁰ to determine rates of OA progression after hip CSI:

- Grade 1: normal/mild OA (KL 0 or 1).
- Grade 2: moderate OA (KL 2 or 3).
- Grade 3: severe OA (KL 4).

Radiographic AVN or SIF was documented as a binary numerical value: 0 = no evidence and 1 = radiographic evidence. Femoral head collapse was assessed as on a 4-tiered scale: 0 = no femoral head collapse; 1 = femoral head collapse, likely secondary to AVN or SIF; 2 = femoral head remodeling, likely secondary to OA; and 3 = femoral head remodeling, unable to assess cause. As side-by-side comparison of pre- and postinjection radiographs may have biased the readers to report change, radiographs were instead

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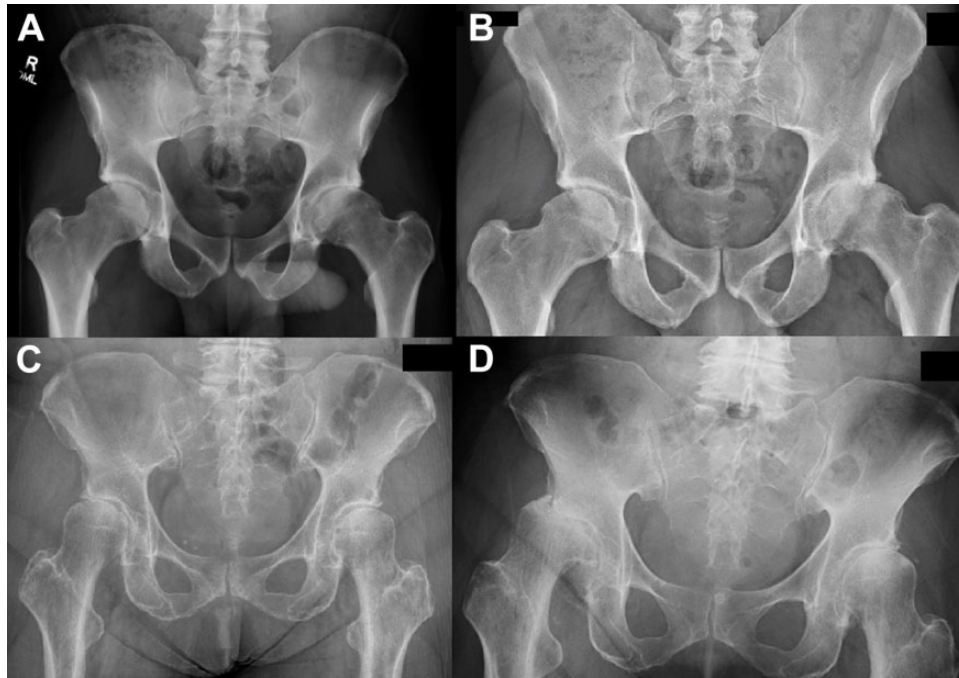


Figure 1. (A) Preinjection and (B) postinjection anteroposterior radiographs of a patient classified as having new left femoral head remodeling secondary to OA (postinjection radiographs taken 46 weeks after hip CSI). (C) Pre-MRI and (D) post-MRI anteroposterior radiographs of a control patient classified as having new right femoral head remodeling due to unknown etiology (post-MRI radiographs taken 13 weeks after hip MRI). Both patients were classified as having new femoral head collapse in our analysis and, as a result, potentially overestimating rates of femoral head collapse. CSI, intra-articular corticosteroid/anesthetic injection; MRI, magnetic resonance imaging; OA, osteoarthritis.

read in random order using software (Research Randomizer V 4.0; <http://www.randomizer.org>) to create a randomized list of all included hip radiographs (before and after radiographs of all hips in both groups).

OA progression was defined as an increase in the modified KL classification of ≥ 1 grade between arbitrated before and after radiograph reads. New AVN or SIF was defined as a preinjection radiographic AVN/SIF score of 0 and a postinjection score of 1. New femoral head collapse was defined as a preinjection femoral head collapse score of 0 and a postinjection score of 1, 2, or 3; as a result, new femoral head remodeling secondary to OA (ie, a change in score from 0 to 2) was treated as new femoral head collapse (Figure 1). As a secondary analysis, the medical record of each included patient was screened for a diagnosis of septic arthritis in the year after the index procedure. In addition, although this study was neither designed nor powered to assess the relative safety of different types and doses of hip CSI, we performed a subgroup analysis comparing these complication rates in hips injected with various corticosteroid types and doses.

We conducted a power calculation (power = 80%; $\alpha = .05$) using as a reference the study by Simeone et al,²⁰ which used a similar study methodology. From their incidence of 12-month modified KL grade progression in patients treated with and without hip CSI (44% vs 24%, respectively), we calculated that a sample size of 87 hips per group would be

necessary to redemonstrate a 20% difference in incidence of OA progression between groups.

Categorical variables were analyzed with chi-square or Fisher exact tests, as appropriate, and continuous variables were compared with Student *t* tests. Results with a *P* value of $P \leq .05$ were considered statistically significant. Interobserver reliability was assessed using the kappa coefficient (κ) for binary variables and weighted κ for ordinal variables. Statistical analysis was performed using SAS Version 9.4 (SAS Institute),

RESULTS

Included in this study were 141 hips in the CSI group (mean age, 55.3 years; mean BMI, 27.7 kg/m²; 53 [37.6%] male; 75 [53.2%] right hips). Regarding the control group of 565 hips that met inclusion criteria, 141 were included after propensity matching for age, sex, BMI, and baseline OA severity [mean age, 54.4 years, mean BMI, 28.2 kg/m²; 53 [37.6%] males; 68 (48.2%) right hips). There were no significant differences between groups in age, sex, BMI, laterality, baseline OA severity, or baseline AVN/SIF on index MRI (Table 1). There was also no significant difference in time between index procedure and final hip radiograph between CSI and control groups ($P = .18$) (Table 1). Table 2 details the corticosteroid type and dose of each CSI performed.

TABLE 1
Baseline Characteristics in the CSI and Control Groups^a

	CSI (n = 141)	Control (n = 141)	P
Age (years)	55.3 (53.0-57.7)	54.4 (51.8-57.0)	.59
BMI (kg/m ²)	27.7 (26.8-28.6)	28.2 (27.2-29.2)	.41
Sex			≥.99
Male	53 (37.6)	53 (37.6)	
Female	88 (62.4)	88 (62.4)	
Laterality			.40
Left	66 (46.8)	73 (51.8)	
Right	75 (53.2)	68 (48.2)	
Baseline radiograph report OA severity			≥.99
No arthritis	44 (31.2)	44 (31.2)	
Mild	50 (35.5)	50 (35.5)	
Mild-to-moderate	3 (2.1)	3 (2.1)	
Moderate	25 (17.7)	25 (17.7)	
Moderate-to-severe	4 (2.8)	4 (2.8)	
Severe	4 (2.8)	4 (2.8)	
No OA read	11 (7.8)	11 (7.8)	
Modified KL grade			.66
No/mild OA	14 (9.9)	12 (8.5)	
Moderate OA	125 (88.7)	125 (88.7)	
Severe OA	2 (1.4)	4 (2.8)	
Preexisting AVN/SIF			.79
AVN	15 (10.6)	19 (13.4)	
SIF	6 (4.3)	7 (5.0)	
AVN and SIF	1 (0.7)	2 (1.4)	
Neither AVN nor SIF	119 (84.4)	113 (80.1)	
Time between index procedure and final hip radiograph, mo ^b	5.43 (4.87-5.98)	4.87 (4.26-5.48)	.18

^aContinuous variables are reported as mean (95% CI) and categorical variables reported as n (%). AVN, avascular necrosis; BMI, body mass index; CSI, corticosteroid injection, KL, Kellgren-Lawrence; OA, osteoarthritis, SIF, stress insufficiency fracture.

^b"Index procedure" was defined as CSI in the CSI group and as magnetic resonance imaging (MRI) in the control group.

An analysis of adjudicated radiographic outcomes was performed after exclusion of 48 matched pairs with at least 1 instance of preexisting AVN or SIF (Table 3). Rates of OA progression, new AVN or SIF, and new femoral head collapse were all similar between groups (Table 3). Of the 3 cases of new femoral head collapse in the CSI group, 2 were classified as femoral head remodeling secondary to OA, leaving only 1 (1.1%) definitive femoral head collapse secondary to AVN or SIF. Of the 2 cases of new femoral head collapse in the control group, both were classified as femoral head remodeling due to an unknown etiology, leaving no definitive femoral head collapses secondary to AVN or SIF. In addition, there were no instances of septic arthritis in the 12 months after the index procedure in either the CSI or control group (Table 3).

Table 4 compares the rates of OA progression, new AVN or SIF, new femoral head collapse, and septic arthritis in CSI patients treated with methylprednisolone and triamcinolone acetate. Table 5 details the rates of these complications in patients treated with various corticosteroid doses.

TABLE 2
Corticosteroid Type and Dose in CSI Cohort^a

	Full CSI Group (n = 141)	CSI Group Excluding Preexisting AVN/ SIF (n = 93)
Corticosteroid type and dosage		
Methylprednisolone 40 mg	34 (24.1)	21 (22.6)
Methylprednisolone 60 mg	18 (12.8)	14 (15.1)
Methylprednisolone 80 mg	26 (18.4)	17 (18.3)
Triamcinolone acetate 40 mg	43 (30.5)	24 (25.8)
Triamcinolone acetate 60 mg	4 (2.8)	4 (4.3)
Triamcinolone acetate 80 mg	15 (10.6)	12 (12.9)
Betamethasone 12 mg	1 (0.7)	1 (1.1)
Local anesthetic type and dosage		
1% lidocaine, 1-6 mL	55 (39.0)	36 (38.7)
0.5% ropivacaine, 2-6 mL	38 (27.0)	24 (25.8)
0.25% bupivacaine, 3-6 mL	23 (16.3)	14 (15.1)
0.2% ropivacaine, 1.5-6 mL	16 (11.3)	12 (12.9)
0.2% lidocaine, 5 ml	2 (1.4)	2 (2.2)
2% ropivacaine, 2.5-6 ml	2 (1.4)	2 (2.2)
2% lidocaine, 3-5 mL	2 (1.4)	1 (1.1)
2% chloroprocaine, 5 mL	1 (0.7)	1 (1.1)
1% lidocaine, 1-2 mL, and 0.5% ropivacaine, 3-6 mL	2 (1.4)	1 (1.1)

^aValues are reported as n (%). AVN, avascular necrosis; CSI, corticosteroid injection; SIF, stress insufficiency fracture.

TABLE 3
Complication Rates by Group, Excluding Preexisting Hip Pathology^a

	CSI Group Excluding Preexisting AVN/SIF (n = 93)	Control Group Excluding Preexisting AVN/SIF (n = 93)	P
Progression of OA	3 (3.2)	3 (3.2)	≥.99
New AVN or SIF	1 (1.1)	0 (0.0)	≥.99
New collapse of the femoral head	3 (3.2)	2 (2.2)	≥.99
New femoral head remodeling, secondary to OA	2 (2.2)	0 (0.0)	.50
Diagnosis of septic arthritis in the 12 months after index procedure	0 (0.0)	0 (0.0)	≥.99

^aData are reported as n (%). AVN, avascular necrosis; CSI, corticosteroid injection; OA, osteoarthritis; SIF, stress insufficiency fracture.

Excellent interrater agreement was found for modified KL classification, detection of AVN or SIF, and detection of femoral head collapse secondary to AVN/SIF (Table 6).

DISCUSSION

In contrast to the findings of recent retrospective investigations, our study did not find that patients treated with hip

CSI had significantly higher rates of OA progression or femoral head articular surface collapse in the 12-month period after injection after controlling for baseline OA severity and preexisting AVN or SIF. These findings support the hypothesis that short-term complication rates of hip CSI are lower than previously reported and do not differ greatly from the control. Although previous retrospective studies conducted by Kompel et al¹² and Simeone et al²⁰ reported rates of OA progression as high as 44% and rates of femoral head articular surface collapse as high as 17% in the 12-month period after hip CSI,²⁰ neither study controlled for baseline OA severity, causing potential selection bias. Those receiving injections were more likely to have more severe degenerative disease; as a result, they were more likely to have subsequent OA progression. In addition, these previous studies did not exclude patients with preexisting AVN or SIF. As femoral head collapse is the natural progression of untreated AVN in about 38% of cases¹⁵ and the majority of SIF cases result in significant

degenerative disease or subchondral collapse,¹⁹ failure to exclude patients with preexisting AVN or SIF in these prior studies may have inflated complication rates. In the present study, we repeated the study methodology of Simeone et al, but we created a control group matched for age, sex, BMI, and OA severity. We used preinjection MRI to exclude patients with preexisting AVN or SIF to limit these forms of selection bias as much as possible. After addressing both of these forms of bias, we found no difference in rates of OA progression, AVN or SIF, femoral head collapse, or septic arthritis between CSI and control groups.

Simeone et al²⁰ reported significantly higher short-term rates of OA progression in patients treated with hip CSI than without (44% vs 24%, respectively). In our study, which uses the same methodology to determine rates of short-term OA progression, there was no significant difference in rates of OA progression between groups (3.2% in both groups). In addition, previous studies investigating the natural history of hip OA have shown that about 15% of hips without AVN show accelerated joint space narrowing,^{3,8} suggesting that the 3.2% rate of OA progression in our CSI group is within expectation for natural disease progression. Similarly, Kompel et al¹² reported that 7.5% of their hip CSI cohort developed RPOA, a rate even higher than our CSI group's rate of OA progression after exclusion of those with preexisting, MRI-proven AVN or SIF. These findings suggest that preinjection AVN and SIF may confound the association between hip CSI and progression of hip OA shown in previous retrospective studies.

Simeone et al²⁰ also reported short-term rates of new AVN and new femoral head collapse to be significantly higher in patients treated with hip CSI than without (AVN, 27% vs 4%; femoral head collapse, 17% vs 1%). In contrast, our study showed similar rates of AVN/SIF and femoral head collapse between groups, once again highlighting the ability of preexisting AVN and SIF to inflate complication rates and the importance of controlling for this confounder. Our reported rate of new femoral head collapse in the CSI group (3.2%) is likely an overestimate, because 2 hips graded as "femoral head remodeling, likely secondary to OA" were included in the calculation of this rate. In

TABLE 4
Complication Rates in CSI Group
by Steroid Type Administered^a

	Methylprednisolone (n = 52)	Triamcinolone Acetonide (n = 40)	P
Progression of OA	2 (3.8)	1 (2.5)	.72
New AVN or SIF	0 (0.0)	1 (2.5)	.43
New collapse of the femoral head	2 (3.8)	1 (2.5)	.72
New femoral head remodeling, secondary to OA	2 (3.8)	0 (0.0)	.50
Diagnosis of septic arthritis in the 12 months after index procedure	0 (0.0)	0 (0.0)	≥.99

^aValues are reported as n (%). AVN, avascular necrosis; CSI, corticosteroid injection; OA, osteoarthritis; SIF, stress insufficiency fracture.

TABLE 5
Complication Rates in Hips Injected With Methylprednisolone or Triamcinolone Acetonide by Dose Administered^a

	40 mg (n = 21)	60 mg (n = 14)	80 mg (n = 17)
Methylprednisolone			
Progression of OA	1 (4.8)	1 (7.1)	0 (0.0)
New AVN or SIF	0 (0.0)	0 (0.0)	0 (0.0)
New collapse of the femoral head	0 (0.0)	2 (14.3)	0 (0.0)
New femoral head remodeling, secondary to OA	0 (0.0)	2 (14.3)	0 (0.0)
Diagnosis of septic arthritis in the 12 months after index procedure	0 (0.0)	0 (0.0)	0 (0.0)
Triamcinolone acetonide			
Progression of OA	1 (4.2)	0 (0.0)	0 (0.0)
New AVN or SIF	1 (4.2)	0 (0.0)	0 (0.0)
New collapse of the femoral head	1 (4.2)	0 (0.0)	0 (0.0)
New femoral head remodeling, secondary to OA	0 (0.0)	0 (0.0)	0 (0.0)
Diagnosis of septic arthritis in the 12 months after index procedure	0 (0.0)	0 (0.0)	0 (0.0)

^aValues are reported as n (%). AVN, avascular necrosis; OA, osteoarthritis; SIF, stress insufficiency fracture.

TABLE 6
Interrater Reliability (κ) Between Reader 1 and Reader 2^a

	κ (95% CI)
Modified KL classification	0.86 (0.80-0.92)
AVN/SIF	0.90 (0.85-0.95)
AVN/SIF with femoral head collapse	0.88 (0.81-0.96)

^aAVN, avascular necrosis; KL, Kellgren-Lawrence; SIF, stress insufficiency fracture.

addition, our secondary analysis revealed that no hips in our CSI group were diagnosed with septic arthritis in the 12 months after injection, suggesting that septic arthritis is not a common complication of hip CSI.

As with any negative study, it is possible that our finding of no difference in short-term OA progression between study groups was the result of a type II error and that a difference may have been seen with a larger sample. However, this study is the largest ever to report on this issue, which is requisitely limited in sample size given the importance of preinjection MRI to rule out the confounding factor of preinjection AVN/SIF. In addition, it had sufficient power to detect a difference based on what has been seen in prior literature.²⁰ Furthermore, a post hoc power calculation using the baseline rate of OA progression observed in this study (3.2%) reveals that we had 80% power to detect a difference in OA progression incidence of less than 12% between groups. Therefore, while we cannot definitively rule out a small (<12%) increase in OA progression in patients treated with hip CSI, at a minimum, this study can confidently conclude that hip CSI does not appear to substantially increase OA progression in most patients. Moreover, even if a small significant difference in OA progression existed between groups, the risk-benefit tradeoff for CSIs may still be positive for select patients given the well-validated improvements in short-term pain and function that CSIs provide.^{2,7,13,17,25} Although a large, high-quality, randomized, placebo-controlled trial is needed to determine the exact effect of hip CSI on OA progression, this study offers the strongest data available that the risk associated with hip CSIs is not dramatically higher than control.

Last, although this study was not designed or powered to assess the relative safety of different types and doses of hip CSI, we performed a subgroup analysis comparing rates of OA progression, new AVN or SIF, new femoral head collapse, and septic arthritis in hips injected with methylprednisolone and triamcinolone acetonide of various doses. Although we did not find these complications to be more frequent in hips treated with 1 corticosteroid type or dose, further studies are needed to conclusively determine whether single injections of certain CSI types and doses are associated with higher short-term complication rates. Furthermore, in vivo and in vitro studies have shown even single doses of local anesthetics to be chondrotoxic,^{4,5} suggesting that the recently reported complications of hip CSI may be due, at least in part, to the type or dose of local anesthetic injected. Although it is possible that the CSIs

reported in the studies by Simeone et al²⁰ and Kompel et al¹² used more chondrotoxic types and/or doses of local anesthetic than our study, this is unlikely, given that our CSI cohort was treated with very similar types and doses of local anesthetic to the hip CSI cohorts in these previous studies. The hip CSIs in the study by Simeone et al contained 4 mL 0.5% ropivacaine, whereas those in the study by Kompel et al contained 2 mL 1% lidocaine and 2 mL 0.25% bupivacaine, the 3 local anesthetics most represented in our CSI cohort. Despite this, there is a great need for future research comparing clinical outcomes in hips injected with various types and doses of both local anesthetic and corticosteroid.

A particular strength of this study was its control group, which was matched for age, sex, BMI, and radiographic OA severity. In addition, all patients had baseline MRIs to rule out preexisting AVN/SIF. Furthermore, the main radiographic outcomes of this study were assessed by multiple experienced musculoskeletal radiologists, whose readings showed high interrater agreement.

However, this study was subject to several limitations. First, because of its retrospective design, this study was unable to prove causality between CSI and the clinical outcomes on which we reported. Second, as with previous case series with similar study designs,^{12,20} this study focused on outcomes of single corticosteroid injections; the results may therefore not be generalizable to patients receiving serial injections. Third, because the purpose of this study was to improve upon the case series conducted by Simeone et al²⁰ by repeating its study methodology while minimizing selection bias, we limited our follow-up to 12 months after hip CSI, thereby limiting our ability to characterize long-term complications of hip CSI. Fourth, despite similar demographics between the CSI and control groups and exact matching of OA severity, these groups may still differ clinically from one another because the hip pain of patients in the CSI group was severe enough to lead to hip CSI. Therefore, it is possible that radiographically occult differences in the severity of patients' degenerative pathology may still confound the outcomes of this study, despite our attempts to match for disease severity. It should be noted, however, this would bias our results *against* CSIs, suggesting that the observed differences may still be upper bounds. Fifth, radiography is not an ideal tool for tracking OA over time because of its reliance on consistent patient positioning and the insensitivity of various OA classification systems (such as KL and Tönnis) to change.^{9-11,16} However, we minimized this limitation by having multiple highly experienced dedicated musculoskeletal radiologists review all films in a randomized fashion. Sixth, because RPOA diagnosis requires side-by-side comparison of before and after radiographs, and our study design limited readers from comparing radiographs in this fashion, we were unable to compare rates of RPOA.

CONCLUSION

Although previous retrospective studies investigating outcomes of hip corticosteroid injection reported rates of OA

progression as high as 44%, rates of RPOA as high as 7.5%, and rates of AVN with femoral head collapse as high as 17% in the 12-month period after hip CSI, they did not control for baseline OA severity or preexisting AVN or SIF. When controlling for these potential confounders, patients treated with CSI in our study showed OA progression in only 3% of cases and new femoral head collapse in only 3% of cases, which was not significantly greater than control and similar to the expected progression of natural disease. There is a great need for an adequately powered, multicenter, randomized, double-blind, placebo-controlled trial investigating the outcomes of steroid injections into the hip joint. In the interim, accurate data on the risks of CSIs are critical to enabling clinicians to carefully weigh the risks and benefits of CSIs for their patients.

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