

Identification of Acute Giant Cell Arteritis in Real-World Data Using Administrative Claims-Based Algorithms

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Objective. The objective of this study was to validate claims-based algorithms for identifying acute giant cell arteritis (GCA) that will help generate real-world evidence on comparative effectiveness research and epidemiologic studies. Among patients identified by the GCA algorithm, we further investigated whether GCA flares could be detected by using claims data.

Methods. We developed five claims-based algorithms based on a combination of *International Classification of Diseases, Ninth Revision* (ICD-9) diagnosis codes, specialist visits, and dispensed medications using Medicare Parts A, B, and D linked to electronic medical records (2006-2014). Acute cases of GCA were determined by chart review using the treating physician's diagnosis of GCA as the gold standard. Among the patients identified with acute GCA, we assessed if a GCA flare occurred during the year after initial diagnosis.

Results. The number of patients identified by each algorithm ranged from 220 to 896. Positive predictive values (PPVs) of the algorithms ranged from 60.7% to 84.8%. Requirement for disease-specific workups, multiple diagnosis codes, or specialist visits improved the PPVs. The highest PPV (84.8%) was noted in an algorithm that required two or more diagnosis codes of GCA from inpatient, emergency department, or outpatient rheumatology visits plus a prednisone-equivalent dose greater than or equal to 40 mg/day occurring 14 days before or after the second ICD-9 diagnosis date, with the cumulative days' supply greater than or equal to 14 days. Among patients identified as having GCA, 18.2% of patients had definite evidence of a flare and 25% had a potential flare.

Conclusion. A claims-based algorithm requiring two or more ICD-9 diagnosis codes from inpatient, emergency department, or outpatient rheumatology visits and high-dose glucocorticoid dispensing can be a useful tool to identify acute GCA cases in large administrative claims databases.

INTRODUCTION

Giant cell arteritis (GCA) is the most common systemic vasculitis in adults. Because the incidence of GCA rises dramatically with age, the number of individuals with GCA will increase as the population ages, and more than 3 million adults are projected to have GCA by the year 2050 (1). Our understanding of GCA incidence, prevalence, and outcomes is based on population-based cohort studies (2-14) and multisite vasculitis registries (15,16). Population-based studies of GCA have identified cases through a variety of methods, including diagnosis codes from general

practitioners, temporal artery biopsy pathology reports from regional hospitals, and medical record review of patients in a geographic region. GCA cohorts identified through these methods generally include several hundred patients.

An algorithm to identify GCA using administrative data would facilitate comparative effectiveness research and epidemiologic studies potentially among thousands of patients with GCA in the United States. Claims-based algorithms that incorporate diagnosis and procedure codes, medication data, and laboratory orders have demonstrated a range of accuracy for identifying rheumatoid arthritis, systemic lupus erythematosus, and antineutrophil

Presented in part at the 2020 European League Against Rheumatism e-Congress, June 3, 2020.

Supported by a research grant from F. Hoffmann-La Roche.

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Dr. S. C. Kim has received research grants from Pfizer, F. Hoffmann-La Roche, AbbVie, and Bristol-Myers Squibb to Brigham and Women's Hospital. No other disclosures relevant to this article were reported.

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Submitted for publication December 1, 2020; accepted in revised form December 3, 2020.

SIGNIFICANCE & INNOVATION

- A well-performing claims-based algorithm using a combination of diagnosis codes, procedure codes, and medication claims can be a useful and efficient tool to identify patients with giant cell arteritis (GCA) in large real-world electronic health care data sets.
- An algorithm using two or more *International Classification of Diseases, Ninth Revision* diagnosis codes from inpatient, emergency department, or outpatient rheumatology visits and a dispensing for high-dose oral glucocorticoid achieved a positive predictive value of 84.8%.
- Presence of a GCA flare can be identified by using claims linked to electronic medical records but requires adequate longitudinal information on the patient's symptoms, laboratory values, and medication dose in addition to medical and pharmacy claims.

cytoplasmic antibody-associated vasculitis (17-20). We hypothesized that a claims-based algorithm that included these elements would identify GCA with high accuracy. Among patients identified by the GCA algorithm, we investigated whether GCA flares (ie, relapse of disease after diagnosis and initially successful treatment) could be detected by using claims data.

PATIENTS AND METHODS

Data source and study population. We used longitudinal Medicare claims data from Parts A (inpatient coverage), B (outpatient coverage), and D (prescription benefits) from January 1, 2006, to December 31, 2014. Only Medicare Fee-for-Service patients were included. Medicare claims were linked to the Partners Research Patient Data Registry (RPDR), which is a centralized electronic medical record (EMR) data warehouse for two large health care provider networks in the Boston, Massachusetts, area and consists of tertiary hospitals, community hospitals, and primary care centers. The details of the RPDR are described elsewhere (21).

We included patients who were aged 65 years or older and enrolled in Medicare Parts A, B, and D at the time of the index date (defined below). We also required all patients to have at least 365 days of continuous enrollment and claims data in Parts A, B, and D before and after the index date. This study was approved by the Institutional Review Board at Brigham and Women's Hospital (2019P001602).

Identification of GCA with algorithms. We developed five algorithms using combinations of specialist visits, *International Classification of Diseases, Ninth Revision* (ICD-9) diagnosis codes, procedural codes, and prescription claims related to the diagnosis or the management of GCA (Supplementary Table 1).

The first algorithm was defined by one or more ICD-9 codes for GCA (446.5) by any physician, high-dose oral steroid dispensing (defined as a prednisone-equivalent dose greater than or equal to 40 mg/day occurring 14 days before or after the first ICD-9 diagnosis date, with the cumulative days' supply greater than or equal to 14 days), and a *Current Procedural Terminology* (CPT) code for erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), or high-sensitivity CRP (hsCRP) testing occurring 14 days before or after the ICD-9 diagnosis date.

The second algorithm was similar to the first algorithm except that it required a rheumatologist visit. It required one or more ICD-9 codes for GCA by a rheumatologist, high-dose oral steroid dispensing as defined above, and one or more CPT code for ESR, CRP, or hsCRP testing. The third algorithm required two or more ICD-9 codes for GCA (limited to outpatient codes by a rheumatologist, inpatient diagnosis codes, and emergency department diagnosis codes) separated by 7 to 30 days and high-dose oral steroid dispensing (prednisone-equivalent dose greater than or equal to 40 mg/day occurring 14 days before or after the second ICD-9 diagnosis date, with the cumulative days' supply greater than or equal to 14 days). The fourth algorithm required two or more ICD-9 codes for GCA by in any setting (outpatient, inpatient, or emergency department) by any physician in addition to high-dose oral steroid dispensing.

The fifth algorithm incorporated biopsy and radiology imaging codes. It required one or more ICD-9 diagnosis codes for GCA by a rheumatologist, high-dose oral steroid dispensing, one or more CPT codes for ESR, CRP, or hsCRP testing, and a CPT code for a temporal artery biopsy or for imaging (temporal artery duplex ultrasound, computed tomography angiography, magnetic resonance angiography, or positron emission tomography and computed tomography) occurring 30 days before or after the first ICD-9 diagnosis code. The index date for all algorithms was defined as the date of the first oral steroid prescription.

Identification of flares among confirmed GCA cases.

Among the GCA cases identified from the five algorithms, we also attempted to assess the presence of GCA flares during a 1-year period. The gold standard for identifying a GCA flare was the treating rheumatologist's impression in clinical notes, discussed below. Because the algorithms were not mutually exclusive, we first identified a list of unique patients who were confirmed to have GCA from the chart reviews. On the basis of clinical experience, we hypothesized that a patient's oral steroid dose (eg, increase in daily dose) or the addition of a steroid-sparing agent (abatacept, cyclophosphamide, methotrexate, or tocilizumab) in claims data would be sensitive measures for identifying GCA flare. We captured the prednisone-equivalent mean oral steroid dose and prescription claims for steroid-sparing agents for each month following the GCA index date up until month 12. We screened out patients with no increase in the steroid dose assessed each month or with no dispensing of steroid-sparing agents and reviewed the

EMR in the remaining patients for the presence of a GCA flare, as detailed below.

Medical chart review and the gold standard. Three investigators (HL, SKC, and EK) conducted manual chart review for the assessment of GCA and flares in the EMR. Patients without medical records or visits assessing vasculitis or its related symptoms were excluded from the chart review. The gold standard was documentation of a GCA diagnosis (including probable GCA) by the treating physician or by a temporal artery biopsy report consistent with acute GCA or a chest imaging result consistent with GCA. Probable GCA cases were classified as part of the study definition of GCA because it reflects the real-world clinical uncertainty of diagnosing this systemic vasculitis and because these patients receive the same medical treatment for GCA.

The following three criteria, modified from a previous randomized clinical trial (22), were assessed while reviewing the presence of a GCA flare: 1) recurrence of signs or symptoms of GCA, including new-onset headache, scalp or temporal artery tenderness, visual symptoms, jaw pain, or polymyalgia rheumatica; 2) elevation of the ESR, CRP, or hsCRP value compared with the prior value; and 3) clinician's recommendation to increase the steroid dose or add a steroid-sparing agent. If a patient met two or three of these criteria, he or she was classified as having a definite flare. However, if only one of the three criteria was met, the patient was classified as having a potential flare. If none of the criteria were met, the patient was classified as having no evidence of a flare.

Statistical analysis. The positive predictive value (PPV) for each algorithm was calculated as the percentage of GCA cases confirmed by medical record review among the number of potential GCA cases with adequate EMR information as described above. The 95% confidence interval of the PPV for each algorithm was calculated by using the normal approximation of the binomial distribution. Among patients fulfilling the study's gold standard definition of GCA, the proportions of patients with GCA with flare, potential flare, and no evidence of flare were calculated. We also assessed the clinical characteristics of the patients with GCA identified by each of the five algorithms and further stratified the characteristics according to patients with flare versus no flare. All analyses were performed by using SAS 9.4 statistical software (SAS Institute Inc.).

RESULTS

GCA algorithm validation. The number of patients identified by the five algorithms ranged from 220 to 896 (Figure 1). All of the identified patients were assessed for adequacy of EMR information about GCA, except for patients identified by algorithms 1 and 4, who underwent random sampling to improve efficiency. Approximately half of the patients identified by one or more algorithms had medical records with adequate information about GCA; a total of 352 patients underwent a complete medical record review when all algorithms were combined. Algorithm 1, which identified the greatest number of patients ($n = 896$), yielded the lowest PPV of 60.7% (Table 1). Algorithm 5, which required disease-specific workups (such as a temporal artery biopsy or

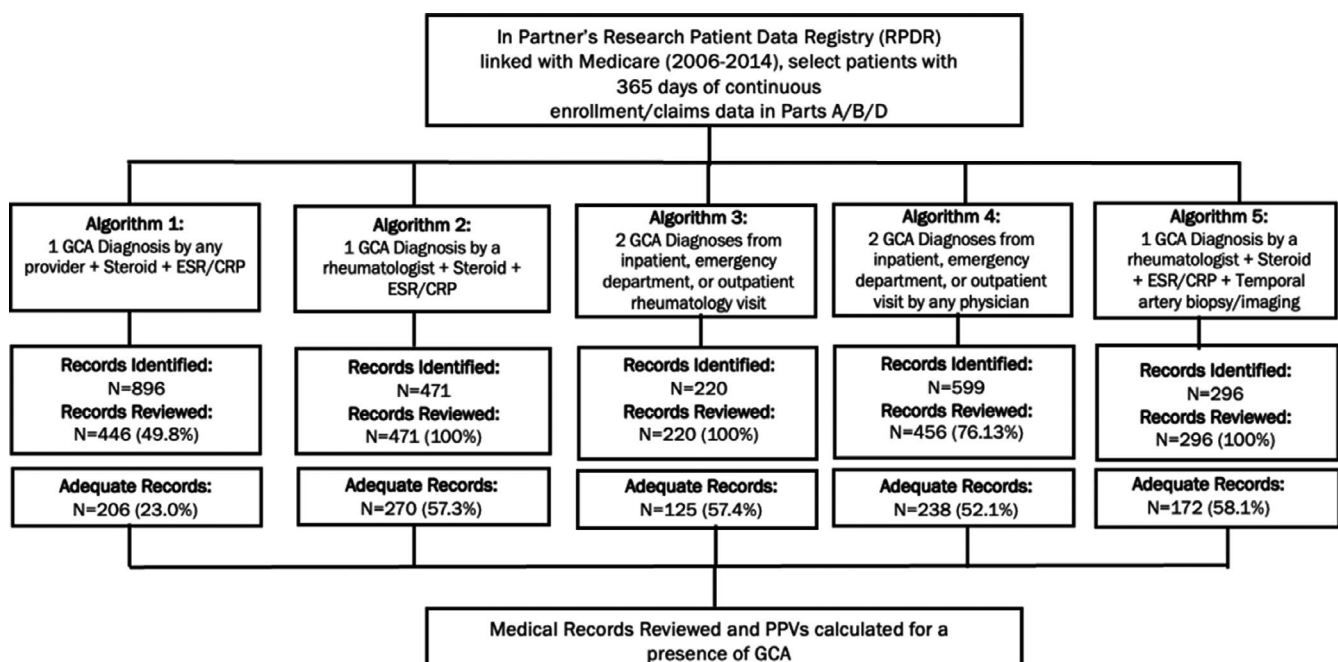


Figure 1. Flowchart of five claims-based algorithms for identifying patients with giant cell arteritis (GCA). CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; PPV, positive predictive value.

Table 1. PPVs of proposed algorithms for identifying GCA

	Algorithm	Records identified	Records reviewed	Adequate records	Definitive GCA/potential GCA	PPV ^a (95%CI)
1	≥1 ICD-9 code for GCA by any physician, high-dose steroid dispensing, and ≥1 CPT code for ESR/CRP/hsCRP testing	896	446 ^b	206 (46.2%)	108/17	60.7% (53.7-67.4)
2	≥1 ICD-9 code for GCA by a rheumatologist, high-dose steroid dispensing, and ≥1 CPT code for ESR/CRP/hsCRP testing	471	471	271 (57.5%)	183/30	78.6% (73.2-83.3)
3	≥2 ICD-9 codes for GCA by a rheumatologist or by any physician plus inpatient or ED setting, high-dose steroid dispensing	220	220	125 (57.4%)	90/16	84.8% (77.3-90.6)
4	≥2 ICD-9 codes for GCA by any physician in any setting, high-dose steroid dispensing	599	456 ^b	238 (52.2%)	167/21	78.99% (73.26-83.99)
5	≥1 ICD-9 codes for GCA by a rheumatologist, high-dose steroid dispensing, ≥1 CPT code for ESR/CRP/hsCRP testing, and CPT code for temporal artery biopsy or imaging	296	296	172 (58.1%)	113/18	76.2% (69.1-82.3)

Abbreviations: CI, confidence interval; CPT, *Current Procedural Terminology*; CRP, C-reactive protein; ED, emergency department; ESR, erythrocyte sedimentation rate; GCA, giant cell arteritis; hsCRP, high-sensitivity C-reactive protein; ICD-9, *International Classification of Diseases, Ninth Revision*; PPV, positive predictive value.

^a True-positive cases included patients with both definitive and probable GCA.

^b Four hundred forty-six records were randomly selected for chart review.

chest imaging), mildly improved the PPV to 76.2%. The PPV was highest in algorithm 3 (84.8%), which required two or more diagnosis codes of GCA from inpatient, emergency department, and outpatient rheumatology visits plus high-dose oral steroid dispensing.

GCA flare identification. GCA flares were assessed in the 214 unique patients confirmed as having GCA by medical record review in the five claims-based algorithms (Supplementary Figure 1). Ninety-six (44.86%) patients did not pass the initial screen for flare (ie, they had no increase in the steroid dose and no addition of a steroid-sparing agent after the index date) and so were excluded from further review. Among 118 patients who passed the initial screen for flare, 88 (41.1% of all GCA cases) had sufficient medical records and underwent a detailed record review for flare.

Among the 88 patients who passed the initial screen for flare and had sufficient data for chart review, 16 (18.18%) had definite evidence of a flare, meaning they displayed two of the following three: 1) signs or symptoms of GCA, 2) elevation of the ESR and/or CRP level, or 3) increased steroid dose and/or addition of a steroid-sparing agent (Tables 2 and 3). Twenty-two (25.00%) patients fulfilled just one of three flare criteria and were classified as having a potential flare.

Clinical characteristics of patients with GCA. We reviewed clinical characteristics of unique patients with GCA identified in the five claims-based algorithms (n = 214) and subsets with subsequent flare (n = 38) and without flare (n = 50) (Table 4). In all three groups, the mean age was 76 years old, and a majority of patients were women and of White race. The mean (SD) baseline oral steroid dose ranged between 61.30 (18.31) mg and 64.34 (18.19) mg. The mean (SD) oral steroid dose at 6 months and 12 months post index GCA event did not differ greatly

between the three groups, ranging from 10.83 (13.55) mg to 13.95 (4.93) mg.

DISCUSSION

We demonstrated that a cohort of patients with acute GCA can be accurately identified in a large administrative claims data set. An algorithm that included at least two ICD-9 codes from inpatient, emergency department, and outpatient rheumatology visits, likely indicating the need for serious medical care to diagnose and treat GCA, and high-dose oral steroids dispensed from a pharmacy achieved a PPV of 85% for GCA, using the treating physician’s diagnosis of GCA as the gold standard. An algorithm that included ordered tests for ESR, CRP, or hsCRP; a temporal artery biopsy; and imaging achieved a lower PPV. Screening for GCA flare based on changes in the glucocorticoid dose or on newly prescribed steroid-sparing agents may improve the efficiency of manual chart review; however, it seems challenging to define GCA flares solely on the basis of claims-based algorithms.

Patients with GCA have been identified for prior epidemiologic studies by manually reviewing temporal artery biopsy

Table 2. Definition of GCA flare

GCA flare definition criteria	
1	Recurrence of signs or symptoms of GCA, including new-onset headache, scalp or temporal artery tenderness, vision loss or visual symptoms, jaw pain, and polymyalgia rheumatica (22)
2	Elevation of ESR and/or CRP level
3	Clinician’s recommendation to increase steroid dose or add a steroid-sparing agent ^a

Abbreviations: CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; GCA, giant cell arteritis.

^a Steroid-sparing agents include abatacept, cyclophosphamide, methotrexate, and tocilizumab.

Table 3. Proportion of flare, potential flare, and no flare identified

GCA flare identification	n (%)
Flare	16 (18.18)
Potential flare	22 (25.00)
No evidence of flare	50 (56.82)

Note. Definite flare: if two or three of criteria are present; potential flare: if one criterion is present; no evidence of flare: if none of the criteria are present.

Abbreviation: GCA, giant cell arteritis.

pathology reports and/or medical records from patients in a large geographic area (4,6,9,12,14). Although this method is likely highly specific for GCA, it is time consuming, requires infrastructure to enable medical record review of patients in the geographic area, and likely sacrifices sensitivity given that not all patients with GCA undergo a temporal artery biopsy. Our algorithm uses information that is readily available in commercial health insurance claims data sets and can readily identify a large GCA cohort in administrative data sets while achieving an excellent PPV of 85%.

Prior GCA studies using data from UK-based databases The Health Improvement Network (THIN) and General Practice Research Database (GPRD) used one diagnosis code for GCA plus a prescription for oral glucocorticoids to identify GCA cases (2,13). In a small validation sample, GCA was confirmed in medical records of 41 of 45 patients with a diagnosis code for “temporal arteritis” from a general practitioner (13). Our algorithm 1 was similar to this but specifically required high-dose steroids and also required testing of ESR and/or CRP. In the Medicare study population with sufficient EMR data for chart review, algorithm 1 achieved only a moderate PPV of 60.7% (lower than what was achieved in the UK data sets by using a similar algorithm and a similar gold standard GCA definition). It is possible that GCA prevalence is higher in the UK general population compared with the Medicare/tertiary health care study population, leading to a higher PPV for a similar algorithm. Differences in clinical diagnosis of GCA (the gold standard for both algorithms) could also affect the PPV; most

of the patients diagnosed with GCA in our study population were diagnosed by a rheumatologist, whereas the UK-based studies focused on patients who were diagnosed by general practitioners. It is also possible that the 45 patients for whom medical records were reviewed were not representative of the larger GCA population. In other studies, incident GCA cases were defined by requiring *International Classification of Diseases* codes for GCA in addition to at least four steroid prescriptions over a 6-month period at a specified dose (23,24) or by excluding individuals with diagnosis codes for other rheumatologic inflammatory diseases (25). However, no PPVs have been provided for direct comparison with our study, which allowed for identification of both incident and prevalent cases.

This present study suggested that it would be difficult to capture GCA flare correctly with claims-based algorithms only; more than half of patients meeting the screening criteria for flare (increased or no change in the glucocorticoid dose or the addition of a steroid-sparing agent) did not fulfill the gold standard definition of flare on medical record review. Flare identification by using claims data has also been shown to be challenging in gout, another chronic rheumatic disease with episodic flares (26). We did not review the medical record for all GCA cases to evaluate for possible flare (only those meeting screening criteria for flare) so were unable to estimate the sensitivity of claims data for flare identification.

Our study was limited by the lack of pertinent EMR data for approximately half of the patients fulfilling one or more algorithm; however, we were able to perform a chart review for the gold standard GCA definition for a large sample (N = 352), which improves on prior validation studies. Our study population was restricted to Medicare enrollees aged 65 years and older, an age range that includes most but not all patients with GCA, and our results may not generalize to patients with younger-onset GCA (eg, age 50-64). Furthermore, because our study is based on two large health care provider networks of tertiary academic medical

Table 4. Clinical characteristics of patients with acute GCA, stratified by flare status

	GCA cohort	GCA cohort with flares ^a	GCA cohort without flares ^a
No. of patients	214	38	50
Age at index date, mean (SD), year	76.44 (6.91)	76.29 (6.32)	76.96 (7.46)
Female sex, %	75.23	81.58	72.00
White, %	88.79	84.21	92.00
Initial oral steroid dose/day, ^b mean (SD), mg	61.55 (18.9)	64.34 (18.19)	61.30 (18.31)
6-month postindex date oral steroid dose/day, ^b mean (SD), mg	11.92 (14.80)	10.83 (13.55)	12.28 (14.59)
12-month postindex date oral steroid dose/day, ^b mean (SD), mg	11.35 (4.99)	13.95 (4.93)	11.38 (3.41)
Initiation of steroid-sparing agents, % ^c	14.02	31.58	16.00

Abbreviation: GCA, giant cell arteritis.

^a Among 88 patients with GCA fulfilling the study definition who had sufficient medical records to evaluate for the study definition of flare.

^b Mean oral steroid dose as a prednisone-equivalent dose.

^c Steroid-sparing agents include abatacept, cyclophosphamide, methotrexate, and tocilizumab, and the variable was measured 12 months post index date.

centers, community hospitals, and primary care centers, the performance of our algorithms may need to be tested externally. The gold standard GCA definition reflects a real-world definition of GCA and is similar to definitions used by prior population-based studies (2,13). Given the nature of insurance claims data, we were not able to include laboratory results in the algorithms. However, as a strength of using medical and pharmacy claims data, we were able to identify all the prescription claims, including name, dose, and days' supply, and were therefore able to identify high-dose steroid prescriptions and duration. Lastly, future studies may be needed to evaluate the performance of claims-based algorithms for GCA using *International Classification of Diseases 10th Revision* codes. In addition, exploration of the sensitivity and specificity of our algorithms will provide helpful information on future studies on the disease, health system, and economic burdens of GCA.

A claims-based algorithm that combined at least two ICD-9 diagnosis codes for GCA among inpatient, emergency department, and outpatient rheumatology visits and high-dose oral glucocorticoid dispensing achieved a PPV of 85% in identifying a cohort of patients with acute GCA. This algorithm can be applied in administrative claims data sets to identify a large cohort of patients with GCA for epidemiologic and comparative effectiveness research studies. However, it would be challenging to define GCA flares using only claims-based algorithms.

AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Dr. S. C. Kim had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study conception and design. Lee, Tedeschi, Chen, S. C. Kim.

Acquisition of data. S. C. Kim.

Analysis and interpretation of data. Lee, Tedeschi, Chen, S. C. Kim.

ROLE OF THE STUDY SPONSOR

F. Hoffmann-La Roche was given the opportunity to make nonbinding comments on a draft of the manuscript. The authors independently designed the study, collected the data, interpreted the results, and had the final decision to submit the manuscript for publication. Publication of this article was not contingent upon approval by F. Hoffmann-La Roche.

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