

Utility of follow-up cardiac magnetic resonance imaging in patients with eosinophilic granulomatosis with polyangiitis

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Abstract

Objective

Cardiac involvement in eosinophilic granulomatosis with polyangiitis (EGPA) occurs in approximately 20-40% of patients and has been shown to be a major predictor of mortality. Cardiac magnetic resonance imaging (CMRI) can detect late gadolinium enhancement (LGE), which can indicate myocardial inflammation and/or fibrosis. However, the role of CMRI at diagnosis and to monitor evolution of cardiac manifestations in EGPA remains unclear. Our aim was to describe the CMRI findings in our EGPA patient cohort, including the presence and persistence of LGE, and to report on the clinical outcomes observed during follow-up.

Methods

This monocentric retrospective descriptive cohort study included 75 patients diagnosed with EGPA, who were seen at least twice between 2015 and January 2023.

Results

Among these 75 patients (male 56%, mean age at diagnosis 45 years), 30 patients underwent CMRI at diagnosis, 9 (30%) of whom had cardiac symptoms. All 30 patients (100%) were treated with glucocorticoids, with 13 (43%) also receiving cyclophosphamide. 16/30 (53%) had LGE on first CMRI; 10/11 had persistent LGE on second CMRI, 4/4 had LGE on third CMRI, and 2/2 had LGE on fourth CMRI. Of these 30 patients, 3 (10%) had cardiovascular events at follow-up, 2 of whom had atrial fibrillation (neither had LGE on their CMRIs), and 1 of whom had coronary artery disease requiring stenting (with LGE on his 4 CMRIs).

Conclusion

The role of CMRIs to study and monitor cardiac involvement in EGPA remains unclear. LGE most often persists despite treatment, with no apparent association with adverse cardiac outcomes in this cohort.

Key words

eosinophilic granulomatosis with polyangiitis, antineutrophil cytoplasmic antibody-associated vasculitis, cardiomyopathy, cardiac magnetic resonance imaging, late gadolinium enhancement

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 Received on May 14, 2026; accepted in
 revised form on July 13, 2026.
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 EXPERIMENTAL RHEUMATOLOGY 2026.

Competing interests: M. Soowamber has received fees for serving on advisory boards from AbbVie, Otsuka, Kabicare and Celltrion; lecture fees from AbbVie, Otsuka and educational support from Abbvie and Kabicare. C. Pagnoux has received fees for serving on advisory boards from Otsuka, Glaxo-SmithKline, AstraZeneca, Amgen, NeoVii, CSL Vifor and NS Pharma; for lecture fees from GlaxoSmithKline and Otsuka; and educational grant support from Otsuka, Pfizer, and GlaxoSmithKline. All other authors have declared no competing interests.

Introduction

Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare multisystem disorder affecting small and medium blood vessels (1). As one of the antineutrophil cytoplasm antibody (ANCA)-associated vasculitides, approximately 30-40% of patients with EGPA have ANCA specific for myeloperoxidase (MPO) (2, 3). The characteristic clinical manifestations of EGPA include asthma, rhinosinusitis often with sinonasal polyposis, peripheral blood eosinophilia and necrotising vasculitis (3). Cardiac involvement in EGPA is variable, occurring in 16-60% (average 20-40%) of patients (4-7), is more frequent in ANCA-negative patients (4, 7-9), and is associated with higher eosinophil counts and greater disease activity (7, 8, 10-12). Cardiac manifestations are heterogeneous and may include myocarditis, pericarditis, cardiomyopathy, heart failure, coronary vasculitis, arrhythmias and cardiac thrombosis (5, 8, 12). Patients may present with symptoms such as chest pain, dyspnoea, and arrhythmias, but up to 45% may be asymptomatic (7, 8, 10, 11).

Cardiac involvement in EGPA has been identified as a major predictor of mortality (6, 7, 13). Even subclinical cardiac disease was found associated in some cohorts with a higher all-cause and cardiovascular mortality (8, 14, 15). Thus, screening for cardiac manifestations with echocardiography and electrocardiography has been suggested in all patients with EGPA (9). A few studies also showed that early detection and aggressive treatment may lead to improved outcomes and possibly even reverse myocardial thickening (10, 16, 17). Therefore, early diagnosis and treatment of cardiac lesions in EGPA is critical.

Cardiac magnetic resonance imaging (CMRI) remains the most sensitive non-invasive modality to detect subtle cardiac abnormalities such as late gadolinium enhancement (LGE), even in asymptomatic patients with normal ECG and echocardiography (7, 15, 16, 18-20). LGE is a marker of myocardial fibrosis; however, it is unclear if there is a pattern of LGE that is specific of EGPA-related cardiomyopathy

(18, 19, 21). Hence, the exact role of CMRI at diagnosis and the utility of repeat CMRI to monitor evolution of cardiac manifestations in EGPA remain unclear.

Our aim was to report on the use and findings of CMRI in our EGPA patient cohort, determine if there is added value in repeating CMRI, and describe the clinical outcomes of persistent LGE observed during follow-up.

Methods

In this monocentric retrospective descriptive cohort study, we included 75 patients with a diagnosis of EGPA who were seen at least twice in our vasculitis clinic between 2015 and January 2023. Patients satisfied the Chapel Hill nomenclature for EGPA and/or the 2022 ACR/EULAR classification criteria (22).

Patient charts were reviewed, and clinical and extra-clinical data were collected, including age, sex, date of EGPA diagnosis, EGPA-related manifestations, cardiovascular risk factors, history of previous cardiovascular events, presence of cardiac symptoms, eosinophil counts at diagnosis, ANCA status, ECG, echocardiography, and CMRI results. Generally, a core CMRI protocol was used including black blood anatomic imaging, cine steady-state free precession, and multiplanar delayed post contrast enhancement, with more advanced protocol used in some cases that included T2-weighted SPAIR images and cardiac relaxometry. As this study was retrospective, with CMRIs being performed at various hospital sites, the CMRI protocols used were not standardised. 30 patients had undergone CMRI at diagnosis, with 11 patients undergoing repeat CMRI during follow-up.

This retrospective study utilised data from the global CanVasc Registry Database which was reviewed and approved by the Research Ethics Board through the CTO Stream platform (CTO-0803).

Results

Patient characteristics

At the time of diagnosis, manifestations of EGPA of the 75 included patients

(male 56%, mean age at diagnosis 46 years) (Table I) were asthma in 69/75 (92%); ear, nose, and throat (ENT) symptoms (nasal polyps, sinusitis, allergic rhinitis) in 57/75 (76%); other respiratory symptoms (cough, haemoptysis) in 65/75 (87%); peripheral neuropathy in 51/75 (68%); constitutional symptoms (fever, fatigue, lethargy) in 42/75 (56%); skin involvement (cutaneous nodules, purpura, urticarial rash) in 29/75 (39%); renal involvement in 24/75 (32%); and central nervous system involvement in 6/75 (8%). 17/75 (23%) had cardiac signs or symptoms including chest pain, peripheral oedema, abnormal cardiac rhythm, cardiac arrest, and myocardial infarction (Table II). More precisely, myocarditis was observed in 3/75 patients (4%), syncope in 1/75 (1%), venous thromboembolism in 1/75 (1%), coronary thrombi in 1/75 (1%), and coronary artery disease in 1/75 (1%). The overall average clinical follow-up duration was 9.8 years from time of diagnosis, though patients were still being followed beyond the conclusion of this study in January 2023. The average follow-up duration for the 30 patients who underwent at least one CMRI was 7.6 years, and for the 11 patients who underwent repeat CMRI was 7.0 years.

Cardiovascular risk factors

14/75 (19%) had pre-existing hypertension, 8/75 (11%) had dyslipidaemia, 3/75 (4%) were active cigarette smokers, 13/75 (17%) were previous cigarette smokers, and 2/75 (3%) had a body mass index of 30 or more.

Investigations

ANCA was positive in 29/73 patients (40%), with 26 of the 29 (90%) being anti-myeloperoxidase antibody (anti-MPO) or perinuclear ANCA (p-ANCA) positive. 64/64 (100%) had eosinophilia $\geq 1.0 \times 10^9$ /litre. Of the 27/75 (36%) who had a troponin level drawn at diagnosis, 20/27 (74%) were elevated. 11/75 (15%) had an abnormal ECG. 31/75 (41%) underwent a TTE at diagnosis, with 23/31 (74%) being abnormal with findings of low left ventricular ejection fraction, wall motion abnormalities including hypokinesis

Table I. Baseline characteristics of EGPA patients.

Characteristics	All patients n=75 (%)	Patients with ≥ 1 CMRI n=30 (%)
Demographics		
Male	42 (56)	19 (63)
Mean age at diagnosis in years (standard deviation)	46 (16)	45 (17)
EGPA features		
Asthma	69 (92)	28 (93)
ENT symptoms	57 (76)	25 (83)
Non-asthma respiratory	65 (87)	27 (90)
Peripheral neuropathy	51 (68)	20 (67)
Constitutional symptoms	42 (56)	16 (53)
Skin involvement	29 (39)	10 (33)
Renal involvement	24 (32)	11 (37)
Central nervous system involvement	6 (8)	4 (13)
Laboratory tests		
Eosinophilia $\geq 1.0 \times 10^9$ /litre	64/64 (100)*	28/28 (100)*
Mean eosinophil count at diagnosis	8.0×10^9 /litre	7.4×10^9 /litre
Elevated troponin	20/27 (74)*	12/16 (75)*
ANCA positive	29/73 (40)*	9/29 (31)*
Anti-MPO or p-ANCA positive	26/29 (90)	9/9 (100)
Treatments		
Glucocorticoids	75 (100)	30 (100)
Cyclophosphamide	26 (35)	13 (43)
Rituximab	0 (0)	0 (0)
Azathioprine	48 (64)	21 (70)
Methotrexate	33 (44)	9 (30)
Mycophenolate or mycophenolic acid	6 (8)	3 (10)
Leflunomide	3 (4)	1 (3)
Mepolizumab	17 (23)	9 (30)
Benralizumab	7 (9)	6 (20)
Omalizumab	4 (5)	1 (3)

* Missing data points.

Table II. Cardiac parameters.

Characteristics	All patients n=75 (%)	Patients with ≥ 1 CMRI n=30 (%)
Clinical features		
Presence of cardiac signs or symptoms	17 (23)	9 (30)
Cardiac manifestations at diagnosis		
Myocarditis	3 (4)	2 (7)
Syncope	1 (1)	1 (3)
Venous thromboembolism	1 (1)	1 (3)
Coronary thrombi	1 (1)	1 (3)
Coronary artery disease	1 (1)	1 (3)
Cardiovascular risk factors		
Hypertension	14 (19)	7 (23)
Dyslipidaemia	8 (11)	4 (13)
Active smoker	3 (4)	3 (10)
Previous smoker	13 (17)	8 (27)
Obesity	2 (3)	1 (3)
Abnormal initial cardiac tests		
Electrocardiogram	11 (15)	8 (27)
Transthoracic echocardiogram	23/31 (74)*	15/16 (94)*
Cardiac manifestations during follow-up		
Major cardiac events during follow-up	10 (13)	3 (10)
Arrhythmia	5 (7)	2 (7)
CAD or cardiac arrest	3 (4)	1 (3)

* Missing data points.

or akinesis, valvular regurgitation or prolapse, coronary artery or aortic dilation, pericardial effusion, myocarditis, or pericarditis.

CMRI findings

30/75 (40%) patients underwent at least one CMRI, with 11/75 (15%) undergoing two CMRI, 4/75 (5%) undergoing

three CMRI, and 2/75 (3%) undergoing four CMRI (Table III). 9/30 (30%) had cardiac symptoms. 8/30 (27%) who underwent CMRI had an abnormal ECG and 15/16 (94%) had an abnormal TTE. 16/30 (53%) had late gadolinium enhancement (LGE) on first CMRI (Fig. 1). 11/16 (69%) patients with LGE on first CMRI underwent repeat imaging, of which 10/11 (91%) had persistent LGE on second CMRI, 4/4 (100%) had LGE on third CMRI, and 2/2 (100%) had LGE on fourth CMRI. The average time between first and second CMRI was 22 months (range 4-81 months). The average time between first and third CMRI was 38 months (range 20-56 months), and between first and fourth CMRI was 68.5 months (range 57-80 months). Other findings on first CMRI included left ventricular dysfunction (40%), pericardial effusion (13%), pericardial gadolinium enhancement (3%), hypokinesia (13%), myocardial oedema (10%), and cardiac thrombus (3%).

Treatment

All patients 75/75 (100%) were treated with glucocorticoids (Table I). 26/75 (35%) were also treated with cyclophosphamide, with none in this cohort receiving rituximab. Other immunosuppressive agents used during follow-up included azathioprine in 48/75 (64%), methotrexate in 33/75 (44%), mycophenolate or mycophenolic acid in 6/75 (8%), leflunomide in 3/75 (4%), mepolizumab in 17/75 (23%), benralizumab in 7/75 (9%), and omalizumab in 4/75 (5%). Of the 16 patients who had LGE on first CMRI, 9/16 (56%) received cyclophosphamide, 4/16 (25%) received benralizumab, and 5/16 (31%) received mepolizumab.

Cardiac events

Cardiac events, including arrhythmia, coronary event, or cardiac arrest, occurred in 10 of the 75 patients (13%) (Table IV). 5/10 (50%) were ANCA positive. 3/10 (30%) had undergone at least one CMRI, with 1/3 (33%) having coronary artery disease requiring stenting and late gadolinium enhancement on first and subsequent four CMRI. 2/3 (67%) had atrial fibrillation but neither

Table III. Cardiac MRI findings.

CMRI number	CMRI 1	CMRI 2	CMRI 3	CMRI 4
Abnormal CMRI	30/30	11/11	4/4	2/2
Findings on CMRI	n (%)	n (%)	n (%)	n (%)
Pericardial effusion	4 (13)	2 (18)	0 (0)	0 (0)
Pericardial gadolinium enhancement	1 (3)	0 (0)	0 (0)	0 (0)
Left ventricular dysfunction (EF<50%)	12 (40)	7 (64)	2 (50)	1 (50)
Hypokinesia	4 (13)	1 (9)	0 (0)	0 (0)
Myocardial oedema	3 (10)	0 (0)	0 (0)	0 (0)
Late gadolinium enhancement	16 (53)	10 (91)	4 (100)	2 (100)
Cardiac thrombus	1 (3)	0 (0)	0 (0)	0 (0)

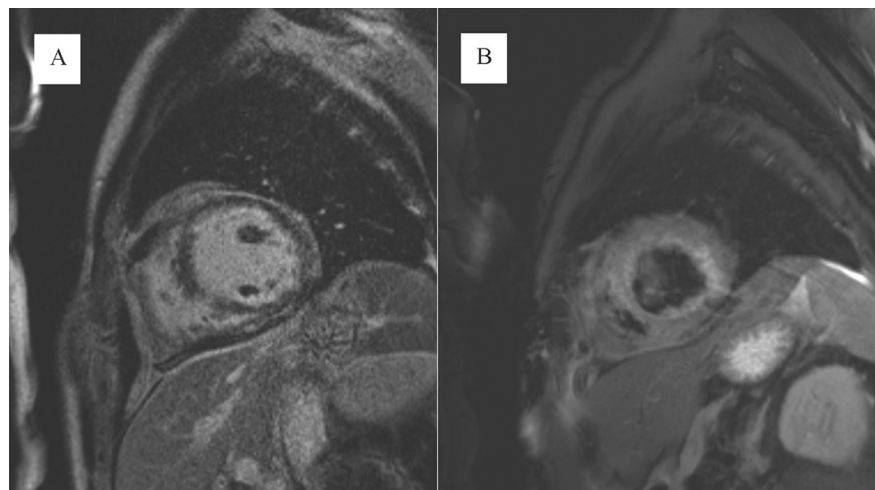


Fig. 1. CMRI of the same patient, without known coronary artery disease. **A.** T1 imaging showing nearly circumferential subendocardial LGE from base to apex with extension to the septum. **B.** T2/oedema imaging showing nearly circumferential increased signal at the apex corresponding to LGE on T1 imaging.

with LGE on their CMRI. 7/10 (70%) were treated with cyclophosphamide.

Discussion

In this retrospective cohort study, we evaluated the use and findings on CMRI in our patients with EGPA, specifically aiming to determine whether there is utility in repeating CMRI and the clinical impact of persistent LGE. We found that myocardial involvement, as indicated by LGE on CMRI, was common; LGE persisted despite adequate treatment; and cardiac events were rare despite the persistent presence of LGE. These findings support previous research that CMRI has value in identifying myocardial involvement (7, 20, 23, 24); however, cardiac adverse events were infrequent in our patients even with the presence of LGE on CMRI.

This study attempted to address an important clinical question and provides

potentially useful real-world data on serial CMRI findings in EGPA, namely showing that LGE often persists over time. However, its retrospective design, selection bias of patients with high pretest suspicion of cardiac involvement (as evidenced by 94% of patients who underwent CMRI having an abnormal transthoracic echocardiogram), small number of patients who underwent repeat imaging, incomplete CMRI assessment among patients with cardiac events, and variable imaging sequences due to the multisite nature of CMRI imaging and non-centralised reading, preclude any final interpretation. This latter aspect also prevented us from conducting a more thorough analysis of changes in LGE extent or pattern over time.

Although only 23% of our patients had cardiac signs or symptoms at diagnosis, a much larger proportion were found to have cardiac abnormalities

Table IV. Cardiac events.

Sex	Age at diagnosis	MACE	CV risk factors	Eosinophil count (x10 ⁹ /L)	Troponin	ANCA	ECG	TTE	CMRI 1 findings	CMRI2 findings	CMRI3 findings	CMRI4 findings	Treatment
M	48	CAD with stenting	Previous smoker, hypertension	18.3	Elevated	Negative	Normal	Normal	LGE, LVEF 44%	LGE, LVEF 42%	LGE, LVEF 58%	LGE, LVEF 48%	GC, CYC, Mepo, MTX, AZA
M	45	Atrial fibrillation	None	2.4	*	p-ANCA	Normal	LVEF 29%	No enhancement, LVEF 47%	Not done	Not done	Not done	GC, CYC, MTX
M	53	Atrial fibrillation	None	*	*	Negative	Atrial fibrillation	Normal	Normal	Anteroseptal wall thickening	Not done	Not done	GC, Mepo, MMF, MTX, AZA
F	78	Atrial fibrillation	Previous smoker	8.6	Elevated	MPO	Atrial fibrillation	Dilated CM	Not done	Not done	Not done	Not done	GC, CYC, AZA
M	28	Ventricular tachycardia; severe dilated CM	None	6.5	*	MPO	Ventricular tachycardia	Myocarditis, pericarditis, LVEF 48%	Not done	Not done	Not done	Not done	GC, MTX, AZA
F	28	ICD insertion	None	30	Elevated	Negative	Normal	Mild LV impairment	Not done	Not done	Not done	Not done	GC, CYC, Mepo, AZA
M	58	Mitral valve repair	None	*	*	Negative	Normal	Normal	Not done	Not done	Not done	Not done	GC, CYC, AZA
M	38	Atrial fibrillation	None	*	*	Negative	Normal	Normal	Not done	Not done	Not done	Not done	GC
M	28	Cardiac arrest	None	5.8	Elevated	MPO	Normal	Moderate to severe hypokinesia of left ventricle with regional variability, LVEF 20-29%.	Not done	Not done	Not done	Not done	GC, CYC, Mepo, AZA
M	44	PEA arrest	None	*	*	MPO	Normal	Normal	Not done	Not done	Not done	Not done	GC, CYC, MTX, AZA

* Missing data points.

AZA: azathioprine; CAD: coronary artery disease; CM: cardiomyopathy; CV: cardiovascular; CYC: cyclophosphamide; GC: glucocorticoids; ICD: implantable cardioverter defibrillator; LGE: late gadolinium enhancement; LV: left ventricle; LVEF: left ventricular ejection fraction; Mepo: mepolizumab; MMF: mycophenolate; MTX: methotrexate.

on further investigation. This discrepancy re-emphasizes the difficulty of identifying cardiac involvement in EGPA based on clinical features alone (7, 8, 16, 20). Moreover, some of the events, such as coronary events, may be unrelated to EGPA, which makes it challenging to establish a causal relationship, particularly in a retrospective study. For instance, the sole patient with persistent LGE on all four CMRI and a cardiac event had two cardiovascular risk factors (previous smoker and hypertension), which may indicate a non-vasculitic aetiology. In our cohort, cardiac abnormalities were detected on ECG in 15% of patients, and on echocardiography in 74% of patients, with 40% of patients undergoing further evaluation with CMRI. These data support the use of advanced cardiac imaging in select patients with EGPA, particularly when there is biochemical, electrocardiographic, or echocardiographic evidence of possible myocardial involvement (7, 21, 24).

More than half of our patients who underwent CMRI had LGE. Additional CMRI abnormalities seen in our cohort included left ventricular dysfunction, pericardial effusion, regional wall motion abnormalities, and less commonly, cardiac thrombus, which highlight the heterogeneity of cardiac pathology associated with EGPA (7, 16, 21). Left ventricular dysfunction, which can occur as a result of myocardial damage, may also reflect concurrent ischemic or other cardiac disease and may not solely be attributable to EGPA (15, 25). The presence of myocardial oedema in a subset of patients suggests active inflammation, while LGE likely represents a combination of inflammation and irreversible myocardial damage (16, 19, 21, 24). CMRI identified cardiac abnormalities even in patients without previously known cardiac involvement from EGPA at diagnosis, supporting its use in detecting subclinical myocardial involvement (20, 24). How-

ever, the therapeutic management of patients with such subclinical cardiac findings and only the CMRI being abnormal remains unclear. Prior studies also described such a high prevalence of occult cardiac involvement only detected by CMRI (20, 24), and suggested a potential prognostic value. In a French retrospective study with 176 patients (7), LGE on CMRI was associated with more than double the risk of major adverse cardiac events, and similarly, in another retrospective study by Dunogué *et al.* (21), patients with persistent CMRI-detected cardiac lesions had significantly more cardiac events during follow-up. Although our study had a similar incidence of cardiac events as Sartorelli *et al.* (7) at 13%, we had fewer than Comarmond *et al.* who reported cardiac involvement in 27.4%, though of which only 16.4% had severe cardiac manifestations such as cardiomyopathy (4). Of our patients with cardiac events, only 33% (one of the three who underwent CMRI) had

LGE, and most patients with persistent LGE did not have subsequent cardiac events. The greater awareness, monitoring, and global management (beyond immunosuppressive therapy) of patients with EGPA and cardiac manifestations may also have contributed to this better outcome.

This study is one of the largest to date to describe the use of serial CMRI in patients diagnosed with EGPA. Among patients who underwent serial CMRI, LGE was present on subsequent scans in the vast majority of patients, with persistence observed in almost all patients who underwent second, third, or fourth CMRI. The average time interval of 22 months between first and second CMRI signifies that LGE often represents chronic myocardial injury rather than transient acute inflammatory change. While repeat CMRI may be valuable for monitoring ventricular function, resolution of myocardial oedema, or detection of new pathology, our findings suggest that LGE frequently persists despite time and adequate treatment. Similarly, other studies have also shown persistence of LGE in patients in clinical remission (20, 24). This persistence of LGE brings into question the prognostic significance of LGE in EGPA. Although our study was not powered or designed in a way to definitively assess outcomes, the consistent presence of LGE on serial imaging without major adverse cardiac events in most patients suggests that it may serve as a marker of cumulative myocardial damage rather than active disease alone. However, the persistence of LGE seen in our cohort is described only among a highly selected subgroup and may not reflect the natural history of cardiac lesions in EGPA more broadly.

Limitations of this study are its retrospective nature, including the non-standardised decision to repeat CMRI or not. All patients were managed and followed in the same tertiary vasculitis clinic, thereby also minimising variability in clinical management.

In conclusion, CMRI is highly sensitive at identifying myocardial abnormalities, including LGE which is commonly seen in EGPA-associated cardi-

omyopathy. However, LGE most often persists despite treatment, suggesting chronic myocardial injury and fibrosis. Even in the presence of LGE, there was an absence of apparent association with adverse cardiac outcomes in this cohort. Therefore, the role of serial CMRIs to study and monitor cardiac involvement in EGPA remains unclear. Although LGE most often persists and events were infrequent in our EGPA cohort, repeat CMRI may still be useful for assessing ventricular function recovery or deterioration, resolution of myocardial oedema, changes in pericardial involvement, detection of thrombus, and evolution of non-LGE markers of inflammation. Further prospective studies are needed to clarify its clinical utility.

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