

# Treatment and Outcomes of Acute Myocardial Infarction in Patients With Polymyalgia Rheumatica With and Without Giant Cell Arteritis

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**This study analyzed the characteristics, management, and outcomes of patients with polymyalgia rheumatica (PMR) hospitalized with acute myocardial infarction (AMI), including sensitivity analysis for presence of giant cell arteritis (GCA). Using the National Inpatient Sample (January 2004 to September 2015) and International Classification of Diseases, Ninth Revision, all AMI hospitalizations were stratified into main groups: PMR and no-PMR; and subsequently, PMR, PMR with GCA, and GCA and no-PMR. Outcomes were all-cause mortality, major adverse cardiovascular/cerebrovascular events (MACCEs), major bleeding, and ischemic stroke as well as coronary angiography (CA) and percutaneous coronary intervention (PCI). Multivariable logistic regression was used to determine adjusted odds ratios with 95% confidence interval (95% CI). A total of 7,622,043 AMI hospitalizations were identified, including 22,597 patients with PMR (0.3%) and 5,405 patients with GCA (0.1%). Patients with PMR had higher rates of mortality (5.8% vs 5.4%,  $p = 0.013$ ), MACCEs (10.2% vs 9.2%,  $p < 0.001$ ), and stroke (4.6% vs 3.5%,  $p < 0.001$ ) and lower receipt of CA (48.9% vs 62.6%,  $p < 0.001$ ) and PCI (30.6% vs 41.0%,  $p < 0.001$ ) than the no-PMR group. After multivariable adjustment, patients with PMR had decreased odds of mortality (0.75, 95% CI 0.71 to 0.80), MACCEs (0.78, 95% CI 0.74 to 0.81), bleeding (0.79, 95% CI 0.73 to 0.86), and stroke (0.88, 95% CI 0.83 to 0.93); no difference in use of CA (1.01, 95% CI 0.98 to 1.04) and increased odds of PCI (1.07, 95% CI 1.03 to 1.10) compared with the no-PMR group. Similar results were observed for patients with concomitant PMR and GCA, whereas patients with GCA only showed increased odds of bleeding (1.51, 95% CI 1.32 to 1.72) and stroke (1.31, 95% CI 1.16 to 1.47). In conclusion, patients with AMI with PMR have an increased incidence of crude adverse in-hospital outcomes than those without PMR; however, these differences do not persist after adjusting for age and comorbidities. © 2022 Elsevier Inc. All rights reserved. (Am J Cardiol 2022;00:1–8)**

Inflammatory rheumatic diseases are associated with an increased risk of cardiovascular disease.<sup>1</sup> Polymyalgia rheumatica (PMR) is a chronic inflammatory condition characterized by bilateral stiffness and pain in the shoulder and hip

region<sup>2</sup> that is associated with an increased risk of vascular events.<sup>3</sup> The mainstay of treatment of PMR is long-term corticosteroids.<sup>2,4</sup> Chronic corticosteroid therapy increases the cardiovascular risk with complications such as hypertension, hyperlipidemia, type 2 myocardial infarction (T2MI) and obesity.<sup>5–8</sup> Furthermore, corticosteroid therapy increases the bleeding risk which could affect the provision of mainstay treatments for AMI, such as the use of potent antiplatelet agents or an invasive strategy.<sup>1,6</sup> PMR is closely associated with giant cell arteritis (GCA), which also increases the risk for the development of cardiovascular disorders.<sup>9,10</sup> There is little evidence focusing on AMI treatment and outcomes in patients with PMR specifically and whether GCA influences these outcomes.<sup>11</sup> This study aimed to describe the prevalence, characteristics, and clinical outcomes of AMI in patients with PMR with and without GCA using a national cohort of United States hospitalizations.

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See page 7 for disclosure information.

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## Methods

The National Inpatient Sample (NIS) is the largest available database of US hospitalizations developed for the Healthcare Cost and Utilization Project, sponsored by the

Agency for Healthcare Research and Quality.<sup>12</sup> The NIS contains anonymized data on diagnoses and procedures from over 7 million hospitalizations annually.<sup>12</sup> The NIS represents a 20% stratified sample of all discharges from United States community hospitals, excluding rehabilitation and long-term acute care hospitals, with the sample representing 97% of the population in the United States.<sup>12</sup>

All adult hospitalizations between January 2004 to September 2015 with a principal discharge diagnosis of AMI were identified and stratified by the presence of PMR. The International Classification of Diseases 9th revision (ICD-9) codes were used to extract data on patient characteristics, comorbidities, management strategies, and hospital outcomes (Supplementary Table 1). Cases were excluded due to missing data, which represented 0.8% of the study sample ( $n = 13,240$ ; Supplementary Figure 1). Analyses were weighted using discharge weights to estimate for national averages.

The primary outcome of this study was all-cause mortality and major acute cardiovascular and cerebrovascular events (MACCEs). Secondary outcomes were major bleeding and acute ischemic stroke as well as the receipt of invasive management for AMI (coronary angiography [CA] and percutaneous coronary intervention [PCI]). Sensitivity analyses were conducted investigating the effect of GCA on the outcomes of patients with PMR. This included 2 further groups: patients with PMR and concomitant GCA and patients with GCA only.

Continuous variables such as age, length of stay, and total charges (cost of the inpatient episode) were summarized using median and interquartile range (IQR). Categorical variables were compared using the chi-square test and summarized as percentages (%). Multivariable logistic

regression was performed to determine the adjusted odds ratio (aOR) for invasive management and adverse outcomes (Supplementary Appendix 1). Results are presented as aOR with 95% confidence intervals (CI). Statistical adjustments for multiple testing were not used due to the large sample size compared with the number of tested variables. Results were determined significant at the level of  $p < 0.05$ . All statistical analyses were performed using SPSS version 27 (IBM Corp, Armonk, New York).<sup>13</sup>

## Results

Following the exclusion of missing data and weighting, a total of 7,622,043 AMI admissions between January 2004 and September 2015 were identified (Supplementary Figure 1). In this cohort, 22,597 patients (0.3%) had a diagnosis of PMR. The prevalence of PMR increased from 0.23% (2004) to 0.34% (2015) (Figure 1). Patients with PMR were on average 15 years older (median age 82 vs 67 years,  $p < 0.001$ ), and more likely to be female (65.6% vs 40.1%) and white (92.6% vs 76.2%) compared with the no-PMR group. The PMR group had a higher prevalence of previous CVA, heart failure, hypertension, hypothyroidism, and chronic renal failure than the no-PMR group ( $p < 0.001$ ) (Table 1).

Patients with PMR had lower receipt of CA and PCI than the no-PMR group (41.0% vs 62.6%,  $p < 0.001$  and 30.6% vs 48.9%,  $p < 0.001$ , respectively) (Figure 2, Table 2). However, when accounting for the differences in baseline characteristics, there was no significant difference in the receipt of CA between the groups, whereas patients with PMR were more likely to receive PCI (aOR 1.07, 95% CI 1.03 to 1.10) (Figure 3, Table 3). Patients with PMR had higher

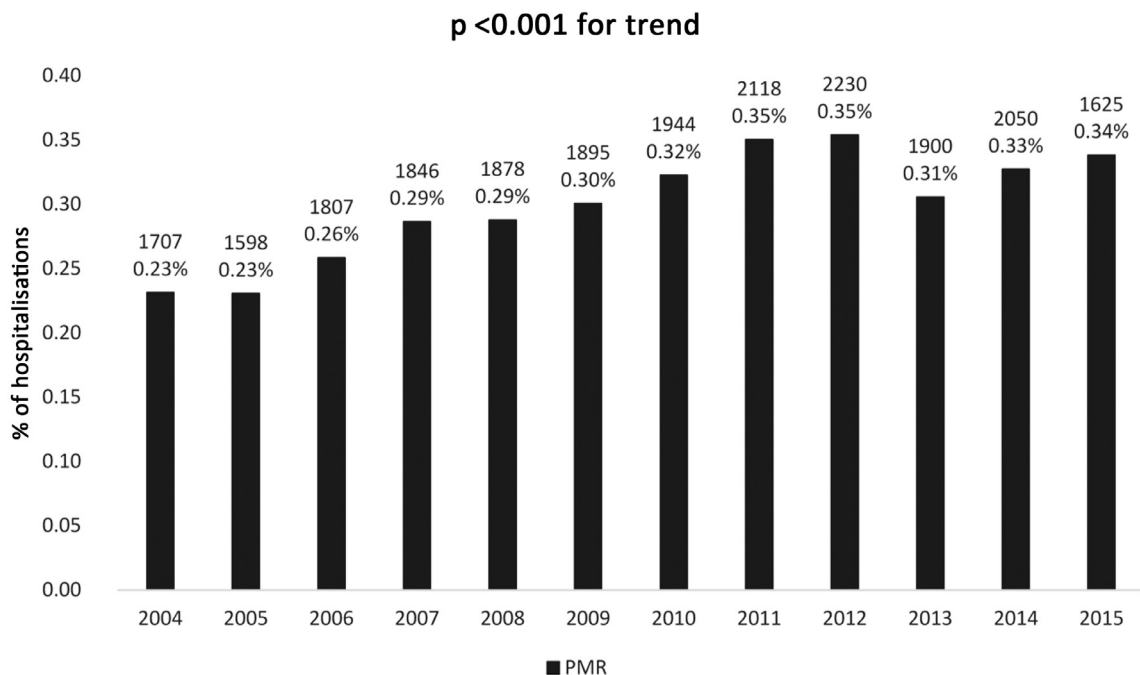


Figure 1. Prevalence of PMR during the study period.

Table 1  
Patient characteristics

Variable	PMR		p-value
	No (97.7%)	Yes (0.3%)	
Number of hospitalizations	7,599,445	22,597	
Age (years), median (interquartile range)	67 (57, 79)	82 (75, 87)	<0.001
Women	40.1%	65.6%	<0.001
White	76.2%	92.6%	<0.001
Black	10.1%	2.8%	<0.001
Hispanic	7.7%	2.2%	<0.001
Other	6.0%	2.4%	<0.001
ST elevation myocardial infarction	26.7%	18.7%	<0.001
Non-ST elevation myocardial infarction / Acute coronary syndrome	73.3%	81.3%	<0.001
Weekend admission	25.8%	2.5%	0.001
Primary expected payer			<0.001
Medicare	56.5%	88.7%	<0.001
Medicaid	6.5%	0.9%	<0.001
Private Insurance	27.9%	9.1%	<0.001
Self-pay	5.8%	0.6%	<0.001
No charge	0.7%	0.02%	<0.001
Other	2.8%	0.7%	<0.001
Median Household Income (percentile)			<0.001
0-25 <sup>th</sup>	28.3%	18.7%	
26 <sup>th</sup> -50 <sup>th</sup>	27.3%	27.0%	
51 <sup>st</sup> -75 <sup>th</sup>	23.4%	27.6%	
76 <sup>th</sup> -100 <sup>th</sup>	21.0%	26.7%	
Cardiogenic shock	4.7%	3.2%	<0.001
Cardiac arrest	2.9%	2.0%	<0.001
Ventricular tachycardia	5.7%	4.1%	<0.001
Ventricular fibrillation	2.6%	1.0%	<0.001
Atrial fibrillation	16.1%	26.8%	<0.001
Dyslipidaemia	56.4%	54.8%	<0.001
Thrombocytopenia	3.1%	3.3%	0.120
Smoker	35.0%	20.0%	<0.001
Previous Acute Myocardial Infarction	10.5%	12.3%	<0.001
History of Ischemic Heart Disease	74.5%	72.2%	<0.001
Previous Percutaneous Coronary Intervention	11.7%	12.2%	0.01
Previous Coronary Artery Bypass Grafting	7.5%	8.5%	<0.001
Previous Cerebrovascular Accident	3.9%	7.5%	<0.001
Anemia	15.2%	27.2%	<0.001
Heart failure	29.7%	38.6%	<0.001
Valvular disease	0.2%	0.2%	0.623
Hypertension	67.0%	73.1%	<0.001
Peripheral vascular disorders	10.6%	15.5%	<0.001
Diabetes mellitus	34.3%	29.5%	<0.001
Hypothyroidism	9.7%	23.3%	<0.001
Chronic pulmonary disease	20.6%	22.3%	<0.001
Pulmonary circulation disorders	0.1%	0.2%	0.007
Coagulopathy	4.2%	0.04%	0.881
Dementia	5.6%	9.9%	<0.001
Depression	6.5%	0.1%	<0.001
Psychoses	2.1%	1.9%	0.031
Paralysis	1.6%	1.2%	<0.001
Other neurological disorders	5.7%	7.3%	<0.001
Liver disease	1.2%	0.8%	<0.001
Peptic ulcer (without bleeding)	0.03%	0%	0.006
Chronic renal failure	16.2%	25.0%	<0.001
Acquired Immunodeficiency Syndrome	0.1%	0.0%	<0.001
Alcohol abuse	2.8%	0.6%	<0.001
Drug abuse	2.1%	0.3%	<0.001
Fluid and electrolyte disorders	18.8%	21.0%	<0.001
Obesity	12.1%	8.4%	<0.001
Weight loss	2.1%	2.8%	<0.001
Solid tumor without metastasis	1.4%	1.5%	0.239

(continued)

Table 1 (Continued)

Variable	PMR		p-value
	No (97.7%)	Yes (0.3%)	
Metastatic cancer	0.8%	0.7%	0.049
Lymphoma	0.5%	0.4%	0.004
Bed size of hospital			<0.001
Small	11.1%	13.9%	
Medium	25.0%	25.9%	
Large	63.8%	60.2%	
Hospital Region			<0.001
Northeast	19.5%	24.1%	
Midwest	22.9%	28.3%	
South	39.9%	26.6%	
West	17.7%	21.0%	
Location/teaching status of hospital			<0.001
Rural	11.1%	13.5%	
Urban non-teaching	41.1%	40.1%	
Urban teaching	47.8%	46.4%	

PMR = Polymyalgia rheumatica.

crude rates of MACCEs (10.2% vs 9.2%,  $p < 0.001$ ), mortality (5.8% vs 5.4%,  $p = 0.013$ ), and stroke (4.6% vs 3.5%,  $p < 0.001$ ) (Figure 2, Table 2). However, after multivariable adjustment, patients with PMR had decreased odds of MACCEs (aOR 0.78, 95% CI 0.74 to 0.81), mortality (aOR 0.75, 95% CI 0.71 to 0.80), major bleeding (aOR 0.79, 95% CI 0.73 to 0.86), and stroke (aOR 0.88, 95% CI 0.82 to 0.93) (Figure 3, Table 3).

Patients with concomitant PMR and GCA were more likely to be older (median age 83 years) and female (75.3%) than the PMR-only, GCA-only, and no-PMR groups (median age 82 years; 65.3% female for the PMR-only group, median age 80 years; 68.5% female for the GCA-only group, and median age 67 years; 40.1% female for the no-PMR group) ( $p < 0.001$ ), with the highest proportion of atrial fibrillation, anemia; and depression and the lowest prevalence of dyslipidemia and peripheral vascular disorders (Supplementary Table 2).

Patients with GCA only were more likely to be younger (median age 80) than the PMR-only and PMR with GCA group (median age 82 for PMR-only group and median age 83 years for PMR with GCA group) but older than the no-PMR group (median age 67), with the highest proportion of chronic pulmonary disease, heart failure, diabetes mellitus, coagulopathy, and chronic renal failure ( $p < 0.05$ ) (Supplementary Table 2).

Looking at the effect of concomitant GCA, patients with PMR with GCA had lower rates of CA and PCI than patients with PMR without GCA (34.7% vs 49.3% for CA; 30.9% vs 20.5% for PCI) (Supplementary Table 3). The crude rates of MACCEs and mortality were generally lower in patients with PMR with GCA than their counterparts but higher for bleeding and stroke (Supplementary Table 3).

After multivariable analysis, patients with PMR and GCA were significantly less likely to receive CA or PCI (aOR 0.66, 95% CI 0.56 to 0.79 for CA; aOR 0.73, 95% CI 0.61 to 0.89 for PCI) (Supplementary Table 4) and less likely to develop MACCEs and mortality (aOR 0.51, 95% CI 0.38 to 0.68 for MACCE, aOR 0.44, 95% CI 0.30 to

0.64 for mortality) than patients without PMR (Supplementary Table 4). Patients with PMR with GCA did not differ with the no-PMR group in adjusted odds for bleeding and stroke (Supplementary Table 4). Patients with GCA only were more likely to suffer bleeding and stroke than patients without PMR (aOR 1.51, 95% CI 1.32 to 1.72 for bleeding; aOR 1.31, 95% CI 1.16 to 1.47 for mortality) (Supplementary Table 4).

The receipt of CA and PCI for patients with PMR steadily increased over the study period (42.9% in 2004 to 57.7% in 2015 for CA; 27.1% in 2004 to 34.7% in 2015 for PCI) (Supplementary Figure 2). Finally, when looking specifically at the STEMI subgroup, the findings for patients with PMR were consistent in both unadjusted and adjusted results (Supplementary Tables 5 and 6).

## Discussion

To the best of our knowledge, this is the first study to examine the prevalence, management, and in-hospital outcomes of patients with PMR admitted due to AMI on a nationwide scale. We report several important findings. First, our analysis suggests that the prevalence of PMR in patients admitted with AMI has increased from 2004 (0.23%) to 2015 (0.34%). Second, patients with PMR are on average, over 15 years older than patients without PMR and have a higher prevalence of cardiovascular risk factors and comorbidities, with overall lower invasive management and worse outcomes. Third, once adjustments for age and other underlying comorbidities are made, we do not observe an excess risk of adverse outcomes associated with a diagnosis of PMR, with patients with PMR being more likely to receive PCI. As expected, patients with PMR and concomitant GCA had similar characteristics and mortality to patients with PMR but were less likely to receive CA and PCI than the control group.

There are limited data investigating the associations between PMR and AMI management and in-hospital outcomes, with most previous studies focusing on risk of vascular events in patients with PMR, reporting disparate

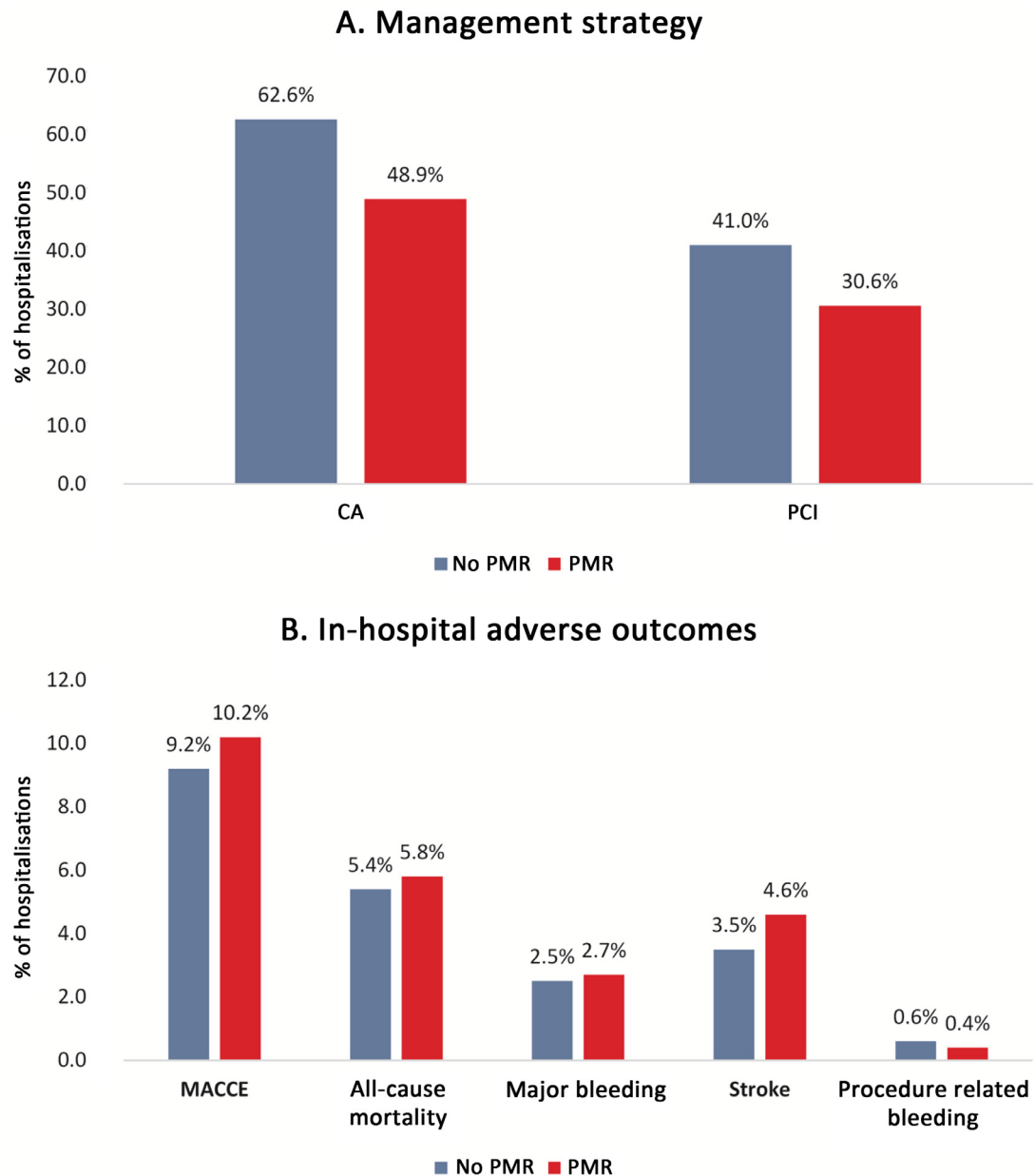


Figure 2. Comparison of management and in-hospital adverse outcomes. (A) Management strategies. (B) In-hospital adverse outcomes.

findings.<sup>3,11,14,15</sup> An older study reported that patients with PMR had an increased cardiovascular risk due to high inflammation levels in the early stages of PMR leading to plaque instability and increased risk of vascular events.<sup>3</sup> Likewise, a systematic review in 34,569 patients with PMR suggested that PMR was associated with an increased risk of coronary artery disease (OR 1.72 95% CI 1.21 to 2.45).<sup>15</sup> In contrast, a contemporary population-based study by Pujades-Rodriguez et al<sup>14</sup> found that the presence of PMR with and without GCA was associated with lower rates of coronary death (0.79, 95% CI 0.66 to 0.95), TIA (0.67, 95% CI 0.66 to 0.95), and coronary and death composite (24.17 vs 25.80/1,000 person-years; 0.90, 95% CI 0.82 to 0.98). Although no previous studies have investigated the management strategy and outcomes in GCA specifically,

Ray et al<sup>16</sup> have shown that older adults (>66 years of age) with GCA are more at risk of cardiovascular disease.

Our analysis suggests that the crude rate of PCI is lower for patients with PMR than patients without PMR. Interestingly the odds of receipt of PCI were higher after adjusting for age and comorbidities, suggesting that it is the presence of an adverse co-morbidity profile driving these conservative treatment choices rather than the presence of PMR. It has been demonstrated previously that patients with PMR, when age-matched to patients without PMR, are more likely to exhibit cardiovascular risk factors such as dyslipidemia, diabetes, hypertension and were at higher risk for vascular complications.<sup>3</sup> Additionally, patients with PMR, on average, are 15 years older and have more comorbidities such as chronic renal failure and pre-existing anemia, which

Table 2  
Comparison of management and in-hospital adverse outcomes

Variables	PMR		p-value
	No (97.7%)	Yes (0.3%)	
Management			
Coronary angiography	62.6%	48.9%	<0.001
Percutaneous coronary intervention	41.0%	30.6%	<0.001
Outcomes			
Major adverse cardiac and cerebrovascular events	9.2%	10.2%	<0.001
All-cause mortality	5.4%	5.8%	0.013
Major bleeding	2.5%	2.7%	0.152
Stroke	3.5%	4.6%	<0.001
Procedure-related bleeding	0.6%	0.4%	<0.001
Intra-aortic balloon pump or assist device	4.7%	2.9%	<0.001
Length of stay (days), median (interquartile range)	3 (2, 6)	3 (2, 6)	<0.001
Total charges (united states dollar), median (interquartile range)	41,883 (20,668, 74,431)	31,734 (15,041, 61,225)	<0.001

PMR = Polymyalgia rheumatica.

are known risk factors for periprocedural bleeding complications.<sup>17,18</sup> Furthermore, long-term treatment with steroids, increases the risk of vascular access site related bleeding and the potential for bleeding events in the setting of dual antiplatelet therapy after PCI.<sup>6,7,19,20</sup> Moreover, PMR is also associated with an increased risk of type 2 AMI, where PCI would not be indicated.<sup>7,10,20,21</sup>

We found that patients with PMR and concomitant GCA were less likely to be managed invasively but also less likely to suffer MACCEs and all-cause mortality than patients without PMR and GCA, whereas these differences

were not detected in the GCA-only subgroup. However, patients with GCA only had more ischemic stroke and major bleeding than patients without PMR and GCA. This could be explained by patients suffering from inflammatory vasculitis being prone to accelerated atherosclerosis, plaque rupture, and more friable vessels susceptible to bleeding events.<sup>22,23</sup> It is well established that patients with PMR and more so those with concomitant GCA have elevated levels of active inflammation.<sup>24</sup> Despite this, patients with AMI with PMR and more so those with concomitant GCA have lower incidence of MACCEs and all-cause mortality.

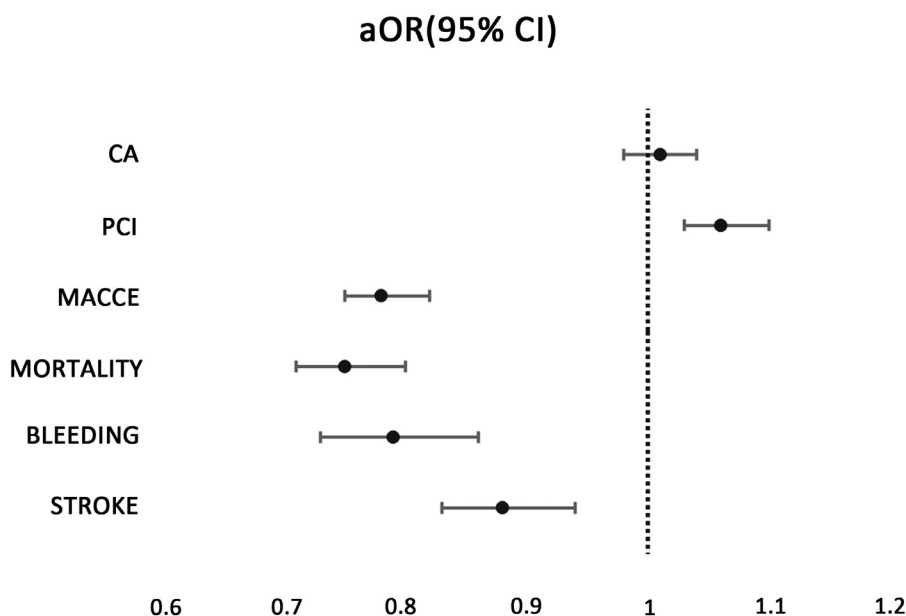


Figure 3. aOR for adverse in-hospital outcomes in patients with PMR.

Reference group is patients with no PMR, where a reference group is a point of comparison for the group being investigated. **Multivariable model:** Multivariable logistic regression model adjusted for: bed size of hospital, region of hospital, location/teaching status of hospital, age, sex, weekend admission, primary expected payer, smoking status, previous myocardial infarction, previous percutaneous coronary intervention, previous coronary artery bypass graft, dyslipidemia, heart failure, and Elixhauser comorbidities (anemias, chronic pulmonary disease, coagulopathy, depression, diabetes mellitus, hypertension, hypothyroidism, fluid and electrolyte disorders, liver disease, lymphoma, obesity, metastatic cancer, peripheral vascular disorders, renal failure, solid tumor without metastasis and valvular heart disease).

Table 3  
Adjusted odds ratios of in-hospital adverse outcomes

Variables	PMR	
	aOR [95% CI]	P-value
Management:		
Coronary angiography	1.01 [0.98, 1.04]	0.386
Percutaneous coronary intervention	1.07 [1.03, 1.10]	<0.001
Outcomes:		
Major adverse cardiac and cerebrovascular events	0.78 [0.74, 0.81]	<0.001
All-cause mortality	0.75 [0.71, 0.80]	<0.001
Major bleeding	0.79 [0.73, 0.86]	<0.001
Stroke	0.88 [0.82, 0.93]	<0.001

Reference group is group without PMR.

**Multivariable model:** Multivariable logistic regression model adjusted for: bed size of hospital, region of hospital, location/teaching status of hospital, age, sex, weekend admission, primary expected payer, smoking status, previous myocardial infarction, previous percutaneous coronary intervention, previous coronary artery bypass graft, previous CVA, dyslipidemia, heart failure, and Elixhauser comorbidities (anemias, chronic pulmonary disease, coagulopathy, depression, diabetes mellitus, hypertension, hypothyroidism, fluid and electrolyte disorders, liver disease, lymphoma, obesity, metastatic cancer, peripheral vascular disorders, renal failure, solid tumor without metastasis and valvular heart disease).

PMR = Polymyalgia rheumatica.

This is consistent with our finding of better MI outcomes in rheumatoid arthritis.<sup>25</sup> It may be hypothesized that this 'protective' effect may be due to chronic immunosuppressive corticosteroid therapy, which forms the mainstay of treatment for these patients.<sup>26</sup> In this context, it is also worth noting that the cumulative dose of corticosteroids used to treat GCA is significantly higher than that used to treat PMR alone, which could possibly mediate the difference in the groups.<sup>26</sup> The potential benefits of corticosteroid therapy are notwithstanding the other adverse effects such as weight gain, loss of glycemic control, dyslipidemia, and increase in blood pressure.<sup>2</sup> It is, however, possible that patients with PMR and GCA had more aggressive lipid treatment and primary prevention in view of their CV risk factor profile.<sup>27</sup> Closer patient monitoring in primary care settings and higher patient engagement due to chronic disease may also influence the outcomes of patients with PMR and GCA.

This study has several limitations that are inherent to the NIS database. NIS data are subject to potential selection bias due to coding inaccuracies and incomplete data.<sup>28</sup> Information on pharmacological management of PMR (e.g., prescription, dose, and duration of glucocorticoids) or laboratory findings (e.g., platelet and hemoglobin count) are not provided by the NIS and could have provided information to improve the analysis of risk and outcomes.<sup>6</sup> Furthermore, it was not possible to determine disease duration and disease activity which could have also influenced outcomes.<sup>14</sup> Also, this study is limited to in-hospital outcomes, and any long-term differences in the outcomes could not be defined. As this is an observational study, confounders that have not been factored into this study could contribute to adverse outcomes despite the broad scope of conditions covered by the NIS.

In conclusion, this analysis reveals that patients with AMI with PMR have an increased odds of invasive management and decreased odds of adverse in-hospital outcomes after adjusting for age and comorbidities compared with their counterparts without. Therefore, it is advanced age and comorbidities that contribute to overall worse outcomes. The presence of concomitant GCA modifies this effect so that patients with AMI with both PMR and GCA do not exhibit differences in stroke and major bleeding, whereas the GCA-only group was more likely to develop stroke and major bleeding, than patients without PMR.

## Disclosures

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## Supplementary materials

Supplementary material associated with this article can be found in the online version at <https://doi.org/10.1016/j.amjcard.2022.03.034>.

- Mohamed MO, Roddy E, Ya'qoub L, Myint PK, Al Alasnag M, Alraies C, Clarson L, Helliwell T, Mallen C, Fischman D, Al Shaibi K, Abhishek A, Mamas MA. Acute myocardial infarction in autoimmune rheumatologic disease: a nationwide analysis of clinical outcomes and predictors of management strategy. *Mayo Clin Proc* 2021;96:388–399.
- Kermani TA, Warrington KJ. Polymyalgia rheumatica. *Lancet* 2013;381:63–72.
- Hancock AT, Mallen CD, Muller S, Belcher J, Roddy E, Helliwell T, Hider SL. Risk of vascular events in patients with polymyalgia rheumatica. *CMAJ* 2014;186:E495–E501.
- Dasgupta B, Borg FA, Hassan N, Barraclough K, Bourke B, Fulcher J, Hollywood J, Hutchings A, Kyle V, Nott J, Power M, Samanta A, BSR and BHPR Standards. Guidelines and Audit Working Group. BSR and BHPR guidelines for the management of polymyalgia rheumatica. *Rheumatol (Oxf Engl)* 2010;49:186–190.
- Souverain PC, Berard A, Van Staa TP, Cooper C, Egberts AC, Leufkens HG, Walker BR. Use of oral glucocorticoids and risk of cardiovascular and cerebrovascular disease in a population based case-control study. *Heart* 2004;90:859–865.
- Pujades-Rodriguez M, Morgan AW, Cubbon RM, Wu J. Dose-dependent oral glucocorticoid cardiovascular risks in people with immune-mediated inflammatory diseases: a population-based cohort study. *PLoS Med* 2020;17:e1003432.
- Brotman DJ, Girod JP, Posch A, Jani JT, Patel JV, Gupta M, Lip GY, Reddy S, Kickler TS. Effects of short-term glucocorticoids on hemostatic factors in healthy volunteers. *Thromb Res* 2006;118:247–252.
- Yasir M, Goyal A, Bansal P, Sonthalia S. Corticosteroid adverse effects. In: *StatPearls. Treasure Island: StatPearls*; 2021:1–34. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK531462/>.
- Gonzalez-Gay MA, Vazquez-Rodriguez TR, Lopez-Diaz MJ, Miranda-Filloo JA, Gonzalez-Juanatey C, Martin J, Llorca J. Epidemiology of giant cell arteritis and polymyalgia rheumatica. *Arthritis Rheum* 2009;61:1454–1461.
- Smilowitz NR, Subramanyam P, Gianos E, Reynolds HR, Shah B, Sedlis SP. Treatment and outcomes of type 2 myocardial infarction and myocardial injury compared with type 1 myocardial infarction. *Coron Artery Dis* 2018;29:46–52.
- Hancock AT, Mallen CD, Belcher J, Hider SL. Association between polymyalgia rheumatica and vascular disease: a systematic review. *Arthritis Care Res (Hoboken)* 2012;64:1301–1305.

12. HCUP National Inpatient Sample (NIS). Healthcare Cost and Utilization Project (HCUP). 2012. Agency for Healthcare Research and Quality, Rockville, MD. [www.hcup-us.ahrq.gov/nisoverview.jsp](http://www.hcup-us.ahrq.gov/nisoverview.jsp)
13. SPSS IBM. *Statistics for Macintosh, version 27.0*. Armonk, N.Y., USA: IBM Corp.
14. Pujades-Rodriguez M, Duyx B, Thomas SL, Stogiannis D, Smeeth L, Hemingway H. Associations between polymyalgia rheumatica and giant cell arteritis and 12 cardiovascular diseases. *Heart* 2016;102:383–389.
15. Ungprasert P, Koster MJ, Warrington KJ, Matteson EL. Polymyalgia rheumatica and risk of coronary artery disease: a systematic review and meta-analysis of observational studies. *Rheumatol Int* 2017;37:143–149.
16. Ray JG, Mamdani MM, Geerts WH. Giant cell arteritis and cardiovascular disease in older adults. *Heart* 2005;91:324–328.
17. Fach A, Bünger S, Zabrocki R, Schmucker J, Conradi P, Garstka D, Fiehn E, Hambrecht R, Wienbergen H. Comparison of outcomes of patients With ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention analyzed by age groups (<75, 75 to 85, and >85 years): (results from the Bremen STEMI registry). *Am J Cardiol* 2015;116:1802–1809.
18. Gharacholou SM, Lopes RD, Alexander KP, Mehta RH, Stebbins AL, Pieper KS, James SK, Armstrong PW, Granger CB. Age and outcomes in ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention: findings from the APEX-AMI trial. *Arch Intern Med* 2011;171:559–567.
19. Ellis SG, Semeneck T, Lander K, Franco I, Raymond R, Whitlow PL. Effects of long-term prednisone (>or =5 mg) use on outcomes and complications of percutaneous coronary intervention. *Am J Cardiol* 2004;93:1389–1390. A6.
20. López-Cuenca A, Gómez-Molina M, Flores-Blanco PJ, Sánchez-Martínez M, García-Narbon A, De Las Heras-Gómez I, Sánchez-Galian MJ, Guerrero-Pérez E, Valdés M, Manzano-Fernández S. Comparison between type-2 and type-1 myocardial infarction: clinical features, treatment strategies and outcomes. *J Geriatr Cardiol* 2016;13:15–22.
21. Greigert H, Zeller M, Putot A, Steinmetz E, Terriat B, Maza M, Falvo N, Muller G, Arnould L, Creuzot-Garcher C, Ramon A, Martin L, Tarris G, Ponnelle T, Audia S, Bonnotte B, Cottin Y, Samson M. Myocardial infarction during giant cell arteritis: a cohort study. *Eur J Intern Med* 2021;89:30–38.
22. Amiri N, De Vera M, Choi HK, Sayre EC, Avina-Zubieta JA. Increased risk of cardiovascular disease in giant cell arteritis: a general population-based study. *Rheumatology (Oxford)* 2016;55:33–40.
23. Cohen Tervaert JW. Cardiovascular disease due to accelerated atherosclerosis in systemic vasculitides. *Best Pract Res Clin Rheumatol* 2013;27:33–44.
24. Weyand CM, Goronzy JJ. Clinical practice. Giant-cell arteritis and polymyalgia rheumatica. *N Engl J Med* 2014;371:50–57.
25. Martínez SC, Mohamed M, Potts J, Abhishek A, Roddy E, Savage M, Bharadwaj A, Kwok CS, Bagur R, Mamas MA. Percutaneous coronary intervention outcomes in patients with rheumatoid arthritis, systemic lupus erythematosus and systemic sclerosis. *Rheumatology (Oxford)* 2020;59:2512–2522.
26. González-Gay MÁ, Pina T, Prieto-Peña D, Calderon-Goercke M, Guallillo O, Castañeda S. Treatment of giant cell arteritis. *Biochem Pharmacol* 2019;165:230–239.
27. Neshet G, Berkun Y, Mates M, Baras M, Rubinow A, Sonnenblick M. Low-dose aspirin and prevention of cranial ischemic complications in giant cell arteritis. *Arthritis Rheum* 2004;50:1332–1337.
28. van Walraven C, Austin P. Administrative database research has unique characteristics that can risk biased results. *J Clin Epidemiol* 2012;65:126–131.