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ORIGINAL RESEARCH

Psoriasis Increases the Risk of ANCA Associated Vasculitis: Insights from A Propensity Score-Matched Study

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Purpose: This study aimed to investigate the risk of anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAVs) among patients with psoriasis in a large population.

Patients and Methods: In this population-based study using the collaborative electronic health record research network, the risk of developing AAVs (ie, eosinophilic granulomatosis with polyangiitis (EGPA), granulomatosis with polyangiitis (GPA), and microscopic polyangiitis (MPA)) was analyzed in a cohort of patients diagnosed with psoriasis between 2006 and 2024. Non-psoriasis controls were selected in a 1:1 ratio using propensity score matching. Patients who were diagnosed with AAVs before the index date were excluded. Univariate Cox proportional hazard model and subgroup analyses were used to estimate the hazard ratio (HR) with a 95% confidence interval (CI) for developing AAVs. The Kaplan-Meier method was used to plot the cumulative incidence curves. The risk of AAVs in psoriatic patients treated with biological agents was explored.

Results: After matching, 436,201 patients were included in each cohort. There were 281 incident cases of AAV during follow-up in the psoriasis cohort and 122 incident cases of AAV in the non-psoriasis cohort. The risk of developing AAVs in the psoriasis cohort was significantly higher than in the non-psoriasis cohort (HRs (95% CI) for AAVs, EGPA, and GPA were 2.01 (1.63 to 2.49), 1.84 (1.11 to 3.06), and 2.11 (1.65 to 2.71), respectively. Compared with psoriasis patients who did not receive biologics, those treated with biologics showed no statistically significant increase in AAVs risk (HR 1.31, 95% CI 0.65 to 2.66).

Conclusion: Patients with psoriasis have a higher risk of AAVs development. Treatment with biologic agents is not associated with an elevated risk of AAVs.

Keywords: psoriasis, anti-neutrophil cytoplasmic antibody, vasculitis, biological agents, incidence

Introduction

Psoriasis is a chronic immune-mediated inflammatory skin condition that affects a significant number of individuals globally, with an estimated 125 million people living with this disease. Psoriasis is associated with comorbidities and increased rates of psoriatic arthritis, cardiometabolic diseases, and mental health disorders.[1] The anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitides (AAVs) are a group of rare disorders characterized by inflammation of small blood vessels, endothelial injury, and subsequent tissue damage.[2] It contains three types of vasculitis, namely granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and eosinophilic GPA (EGPA). Historically, AAVs prevalence was estimated at 48–184 cases per million,[3] with improved survival and better case definition, this

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estimate has now risen to 300–421 cases per million.[4] GPA and MPA are, in most cases, defined by the presence of ANCAs targeting either leukocyte proteinase 3 (PR3) or myeloperoxidase (MPO).[2] Clinically, these antibodies are identified through indirect immunofluorescence (IIF) and high-sensitivity antigen-specific immunoassays—most notably ELISAs for PR3-ANCA and MPO-ANCA.[5]

Previous studies have suggested a potential link between psoriasis and AAVs. Kutukculer et al found that perinuclear ANCA (pANCA) positivity was present in 33.3% (12/33) of psoriatic patients, a rate significantly higher than the 5% (1/20) observed in the control group ($p<0.05$).[6] De Bandt et al reported that 4% (1/25) of patients with psoriatic arthritis (PsA) who had no vasculitic symptoms tested positive for pANCA.[7] Additionally, Quarenghi et al described a case of a PsA patient with pANCA-positive vasculitis.[8] Although these findings hint at a possible association between psoriasis and an increased risk of developing AAVs, the studies involved small sample sizes, and the exact impact of psoriasis on the incidence of AAVs remains uncertain.

To address this gap, the current comprehensive, population-based study aims to investigate the relationship between psoriasis and the incidence of AAVs in a larger and more representative population.

Methods

Data Sources

The data utilized in this analysis were sourced from collaborative electronic health records (EHR) database, a comprehensive health research network that offers real-time access to a vast array of electronic health record data.[9] These data encompass a wide spectrum of patient information including demographics, diagnoses, procedures, medications, laboratory results, and vital signs. The EHR database adheres to stringent regulations set forth by the Health Insurance Portability and Accountability Act (HIPAA) and the General Data Protection Regulation (GDPR), ensuring the privacy and security of patient information. The Western Institutional Review Board granted a waiver for informed consent due to the platform's handling of only aggregated data counts and statistical summaries, which are derived from de-identified information. This waiver is a testament to the platform's commitment to maintain patient confidentiality. The current study was approved by the Institutional Review Board of Chung Shan Medical University Hospital (Approval Number: CS2-21176), further ensuring the ethical conduct of the research.

Study Design and Subject Selections

Diseases in this study were classified using the International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM) coding system. The psoriasis cohort was defined as patients aged ≥ 20 years who received at least two independent diagnoses of psoriasis (ICD-10-CM: L40) between 1 January 2006 and 31 December 2024. The control cohort was composed of non-psoriasis patients (age ≥ 20 years) who underwent a general examination without complaint, suspected, or reported diagnosis (ICD-10-CM code Z00) and who have never been diagnosed with psoriasis. The primary outcome measure for this investigation was the diagnosis of AAVs, which included eosinophilic granulomatosis with polyangiitis (EGPA, ICD-10-CM code M30.1), granulomatosis with polyangiitis (GPA, ICD-10-CM code M31.3), and microscopic polyangiitis (MPA, ICD-10-CM code M31.7). Patients with a pre-existing AAVs diagnosis as of the index date were excluded from the study. The index date for the psoriasis cohort was defined as the date of the initial diagnosis of psoriasis. For the control cohort, the index date was defined as the date of the first general examination. The comorbidities evaluated encompassed a range of conditions: hypertensive disease (ICD-10-CM codes I10-I16), ischemic heart disease (ICD-10-CM codes I20-I25), cerebrovascular diseases (ICD-10-CM codes I60-I69), diabetes mellitus (ICD-10-CM codes E08-E13), lipoprotein metabolism disorders (ICD-10-CM code E78), liver disease (ICD-10-CM codes K70-K77), and depressive disease (ICD-10-CM code F32). Comorbidity was defined as any condition diagnosed within the one-year period preceding the index date. Additional covariates analyzed included age, sex, smoking status (ICD-10-CM code Z87.891), body mass index (BMI) (laboratory code TXN9084), socioeconomic status (related to housing and economic circumstances, ICD-10-CM code Z59), medical utilization, and medication use, specifically hormones/synthetics/modifiers (medication code HS000) and anti-rheumatics (medication code MS100). Exposure to biological agents was defined as their use within the first six months following a psoriasis diagnosis. The

biological agents included tumor necrosis factor-α inhibitors (etanercept, adalimumab, and infliximab), interleukin-17 inhibitors (ixekizumab, secukinumab), and interleukin-12/23 p40 inhibitor (ustekinumab).

Statistical Analysis

Statistical analyses were conducted using the EHR database analytics platform. To mitigate the influence of confounding variables, we leveraged propensity score matching (PSM) feature. This was accomplished by employing a 1:1 ratio matching algorithm, specifically a greedy nearest neighbor method with a caliper set to 0.1 times the pooled standard deviations of the demographic characteristics, comorbidities, and medication profiles of the two cohorts. The balance between the cohorts was assessed before and after matching using the standardized mean difference (SMD), with an SMD of less than 0.1 indicating a good match. Kaplan-Meier curves were used to estimate the risk of developing AAVs. Hazard ratios (HRs) and the corresponding 95% confidence intervals (CI) were derived using a univariate Cox proportional hazards model. The Log rank test was applied to determine whether there were significant differences in the survival curves between the two cohorts. A *p*-value of less than 0.05 was established as the threshold for statistical significance. Subgroup analyses were conducted to further investigate whether the risk of AAVs development varied among patients with psoriasis based on each covariate. In accordance with the EHR database design, PSM was repeated for each subgroup to ensure robust analysis. This rigorous statistical approach ensured that the findings were as unbiased as possible, providing a solid foundation for the conclusions drawn from this research.

Results

Patient Characteristics

We identified 436,462 patients with psoriasis between January 1, 2006 and December 31, 2024 in the psoriasis cohort and 14,193,528 non-psoriasis patients in the control cohort. After excluding AAVs cases diagnosed before the index date and performing PSM, the psoriasis and non-psoriasis cohorts finally included 436,201 patients each (Figure 1).

The mean age of patients in the two cohorts was 51.6 ± 15.4 years. The two cohorts had similar demographics, comorbidities, and medication use (SMDs <0.1). The baseline patient characteristics are presented in Table 1.

Risk of AAVs in Psoriasis and Non-Psoriasis Cohorts and Subgroup Analyses

In the psoriasis cohort, 281 incident cases of AAVs occurred during the study period. In the non-psoriasis cohort, there were 122 AAVs cases. GPA accounts for the majority of AAVs cases, whereas EGPA and MPA have similar incidence rates. Before PSM, the risk of developing AAVs in the psoriasis cohort was found to be significantly higher than in the non-psoriasis cohort (HRs with 95% CI) for AAVs, EGPA, and GPA were 2.00 (1.77 to 2.26), 1.82 (1.36 to 2.45), and 2.08 (1.81 to 2.39), respectively. This finding aligns with the post-PSM risk profile (Table 2). Although the psoriasis cohort showed an elevated risk of developing MPA compared to the non-psoriasis cohort, this association did not reach statistical significance (HR 1.48, 95% CI 0.88–2.51). Kaplan-Meier curves revealed that the incidence of AAVs in the psoriasis cohort was significantly higher than that in the non-psoriasis cohort (*Log rank* test, p<0.001) (Figure 2). Subgroup analysis also confirmed a higher risk of AAVs in patients with psoriasis (Figure 3).

Risk of AAVs in Psoriasis Patients Treated with Biological Agents

We further explored the impact of biological agents use on AAVs risk. Compared to psoriasis treated without biological agents, patients with psoriasis treated with biological agents showed no statistically significant increase in AAVs risk (HR, 95% CI: 1.31, 0.65 to 2.66) (Table 3).

Discussion

In this propensity score-matched analysis, patients with psoriasis exhibited a significantly higher risk of developing AAVs than their non-psoriasis counterparts following adjustment for potential confounders. The consistency of this association was further validated through subgroup analyses. Furthermore, the use of biological agents was not associated

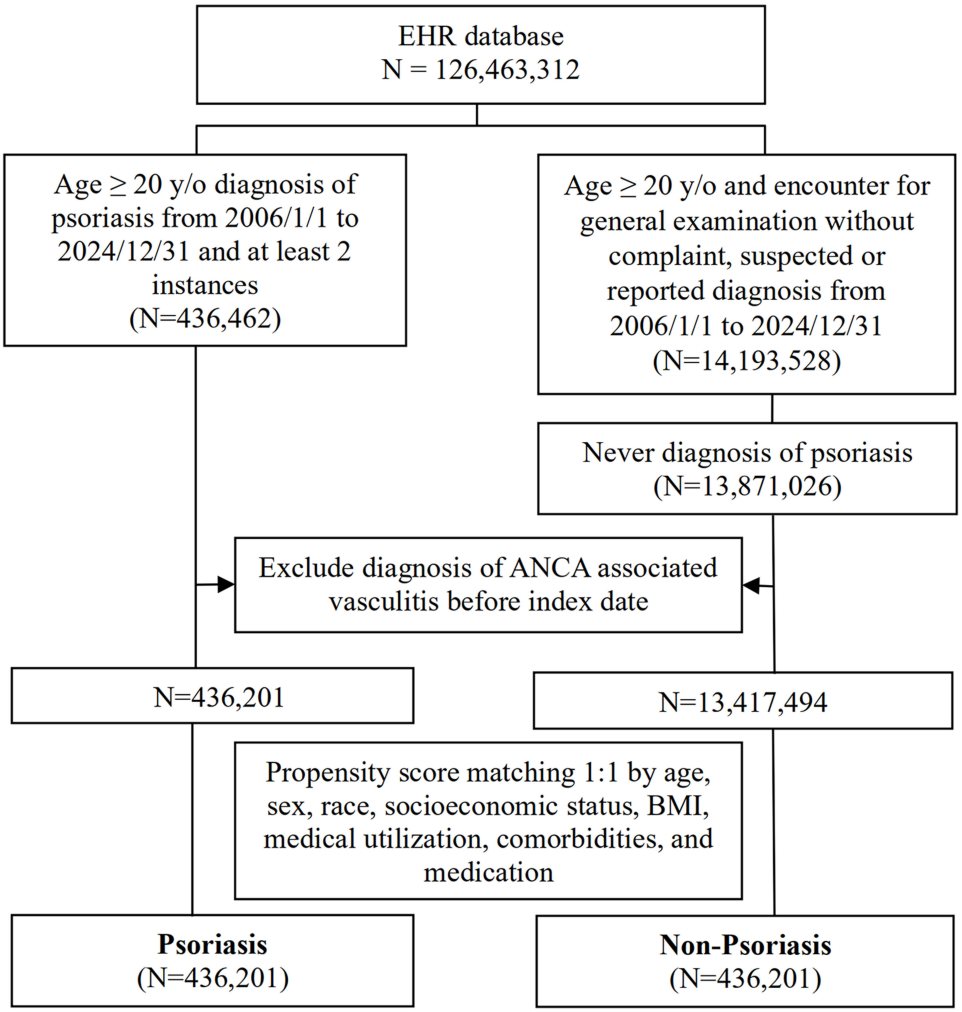

Figure 1 Flow chart of the study design.
Abbreviations: ANCA, anti-neutrophil cytoplasmic antibody; BMI, body mass index; EHR, electronic health records.

with an increased risk of AAVs. These findings significantly enhance our understanding of the relationship between psoriasis and the development of AAVs.

Psoriasis is increasingly recognized as being linked to a variety of other autoimmune disorders, underscoring its broader impact on the immune system. Chen et al demonstrated that patients with psoriasis have a significantly higher risk of developing dermatomyositis compared to those without psoriasis (HR: 2.41, 95% CI: 2.01–2.89).[10] Similarly, Kang et al found that psoriasis may increase the risk of Sjögren's syndrome (HR: 1.50, 95% CI: 1.42–1.58).[11] Furthermore, transcriptomic analyses have revealed potential shared pathogenic mechanisms between psoriasis and Sjögren's syndrome, involving cellular proliferation, immune cell recruitment, cytokine secretion, and the interferon response to viral infections.[11] Additionally, a cross-sectional study revealed that psoriasis was significantly associated with systemic lupus erythematosus (OR: 1.56, 95% CI: 1.30–1.85).[12] Psoriasis has also been linked to systemic sclerosis.[13] These findings underscore the multifaceted nature of psoriasis and its complex interplay with other autoimmune conditions.

Regarding our study, two underlying mechanisms may account for the risk of AAVs in patients with psoriasis. First, both psoriasis and AAVs can be triggered by the same bacterial infection.[14,15] Staphylococcus aureus (*S. aureus*), a Gram-positive bacterium, is known to colonize psoriatic skin and exacerbate skin lesions.[16,17] It has also been shown to induce psoriasis-related transcriptomes, which are further amplified by IL-17A and TNF-α.[18] In the context of AAVs,

Table 1 Demographic Characteristics of Psoriasis Cohort and Non-Psoriasis Cohort

	Before PSM		P value	SMD	After PSM		P value	SMD
	Psoriasis (N = 436,201)	Non-Psoriasis (N = 13,417,494)			Psoriasis (N = 436,201)	Non-Psoriasis (N = 436,201)		
Age (yrs), mean ± SD	51.6 ± 15.4	47.7 ± 17.1	<0.001	0.242	51.6 ± 15.4	51.6 ± 15.4	0.999	<0.001
Sex, n (%)*								
Female	236327 (54.2)	7,234,795 (53.9)	0.001	0.005	236,327 (54.2)	236,327 (54.2)	0.999	<0.001
Male	182750 (41.9)	5,516,636 (41.1)	<0.001	0.016	182,750 (41.9)	180,085 (41.3)	<0.001	0.012
Race, n (%)*								
White	335953 (77.0)	8,720,321 (65.0)	<0.001	0.267	335,953 (77.0)	335,953 (77.0)	0.999	<0.001
Black or African American	23252 (5.3)	1,698,507 (12.7)	<0.001	0.258	23,252 (5.3)	23,252 (5.3)	0.999	<0.001
Asian	14898 (3.4)	570,288 (4.3)	<0.001	0.043	14,898 (3.4)	14,286 (3.3)	<0.001	0.008
Socioeconomic status, n (%)								
Problems related to housing and economic circumstances	907 (0.2)	40,267 (0.3)	<0.001	0.018	907 (0.2)	907 (0.2)	0.999	<0.001
BMI (kg/m^2), mean ± SD	31.2 ± 7.8	29.7 ± 7.3	<0.001	0.198	31.2 ± 7.8	29.9 ± 7.2	<0.001	0.168
BMI (kg/m^2), n (%)*								
<18.5	3338 (0.8)	121,931 (0.9)	<0.001	0.016	3338 (0.8)	4129 (0.9)	<0.001	0.020
18.5–24.9	36,583 (8.4)	1,361,752 (10.1)	<0.001	0.061	36,583 (8.4)	48,060 (11.0)	<0.001	0.089
25–29.9	53,982 (12.4)	1,709,145 (12.7)	<0.001	0.011	53,982 (12.4)	65,246 (15.0)	<0.001	0.075
≥30	85,222 (19.5)	2,121,891 (15.8)	<0.001	0.098	85,222 (19.5)	82,364 (18.9)	<0.001	0.017
Medical utilization, n (%)								
Outpatient	273159 (62.6)	7,813,431 (58.2)	<0.001	0.090	273,159 (62.6)	278,324 (63.8)	<0.001	0.025
Emergency	42278 (9.7)	1,256,043 (9.4)	<0.001	0.011	42,278 (9.7)	45,613 (10.5)	<0.001	0.025
Hospitalization	37150 (8.5)	1,043,095 (7.8)	<0.001	0.027	37,150 (8.5)	40,893 (9.4)	<0.001	0.030
Comorbidities, n (%)								
Hypertensive diseases	87386 (20.0)	2,431,171 (18.1)	<0.001	0.049	87,386 (20.0)	96,195 (22.1)	<0.001	0.050
Ischemic heart diseases	19943 (4.6)	542,547 (4.0)	<0.001	0.026	19,943 (4.6)	21,860 (5.0)	<0.001	0.021
Cerebrovascular diseases	8152 (1.9)	249,257 (1.9)	0.591	0.001	8152 (1.9)	9762 (2.2)	<0.001	0.026
Diabetes mellitus	40528 (9.3)	1,038,222 (7.7)	<0.001	0.056	40,528 (9.3)	42,810 (9.8)	<0.001	0.018
Disorders of lipoprotein metabolism	75280 (17.3)	2,010,569 (15.0)	<0.001	0.062	75,280 (17.3)	82,642 (18.9)	<0.001	0.044
Liver disease	13008 (3.0)	257,126 (1.9)	<0.001	0.069	13,008 (3.0)	11,119 (2.5)	<0.001	0.026
Depressive episode	25818 (5.9)	658,690 (4.9)	<0.001	0.045	25,818 (5.9)	26,461 (6.1)	0.004	0.006
Smoking	16244 (3.7)	423,772 (3.2)	<0.001	0.031	16,244 (3.7)	18,024 (4.1)	<0.001	0.021
Medication, n (%)								
Hormones/synthetics/modifiers	140225 (32.1)	3,102,169 (23.1)	<0.001	0.203	140,225 (32.1)	140,225 (32.1)	0.999	<0.001
Antirheumatics	72460 (16.6)	1,516,279 (11.3)	<0.001	0.154	72,460 (16.6)	72,460 (16.6)	0.999	<0.001

Notes: *The sum is not equal to 100% because of missing data in the collaborative electronic health record database.
Abbreviations: BMI, body mass index; PSM, propensity score matching; SD, standard deviation; SMD, standardized mean difference.

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Table 2 Risk of Anti-Neutrophil Cytoplasmic Antibody Associated Vasculitis Exposed to Psoriasis Compared to Non-Psoriasis

	Psoriasis	Non-Psoriasis	HR (95% CI)
Before PSM	N = 436,201	N = 13,417,494	
AAVs	281	3612	2.00 (1.77–2.26)
EGPA	47	656	1.82 (1.36–2.45)
GPA	212	2630	2.08 (1.81–2.39)
MPA	38	704	1.38 (0.99–1.91)
After PSM	N = 436,201	N = 436,201	
AAVs	281	122	2.01 (1.63–2.49)
EGPA	47	22	1.84 (1.11–3.06)
GPA	212	88	2.11 (1.65–2.71)
MPA	38	22	1.48 (0.88–2.51)

Notes: Propensity matching by age, sex, race, socioeconomic status, BMI, medical utilization, comorbidities, and medication.
Abbreviations: AAVs, anti-neutrophil cytoplasmic autoantibody-associated vasculitides; CI, confidence interval; EGPA, eosinophilic granulomatosis with polyangiitis; GPA, granulomatosis with polyangiitis; HR, hazard ratio; PSM, propensity score matching; MPA, microscopic polyangiitis.

S. aureus has been detected in the nasal carriage of patients with GPA experiencing relapses,[19] and molecular mimicry involving a plasmid-encoded sequence from *S. aureus* has been implicated in MPO-AAV.[20] Furthermore, *S. aureus* peptides exhibit a strong homology with PR3 peptides, potentially contributing to the development of neutrophil PR3-AAV.[21] Second, shared genetic factors, such as protein tyrosine phosphatase non-receptor 22 (*PTPN22*), drive both psoriasis and AAV. Polymorphisms in the *PTPN22* gene have been linked to the risk of psoriasis,[22–24] and are also common in both PR3-AAV and MPO-AAV.[25,26]

In our study, the biological agents use is not associated with an elevated risk of AAVs. However, this finding should be approached with caution because of the lack of strict definitions regarding the duration and dosage of biological agents and the absence of a separate analysis for TNFi agents. There have been isolated case reports suggesting that biological agents, particularly TNFi, could trigger AAVs.[27–30] The proposed mechanisms include the deposition of anti-TNF-α/TNF-α immunocomplexes on vessel walls,[31] or the induction of a T-cell cytokine profile that leads to inflammation.[32]

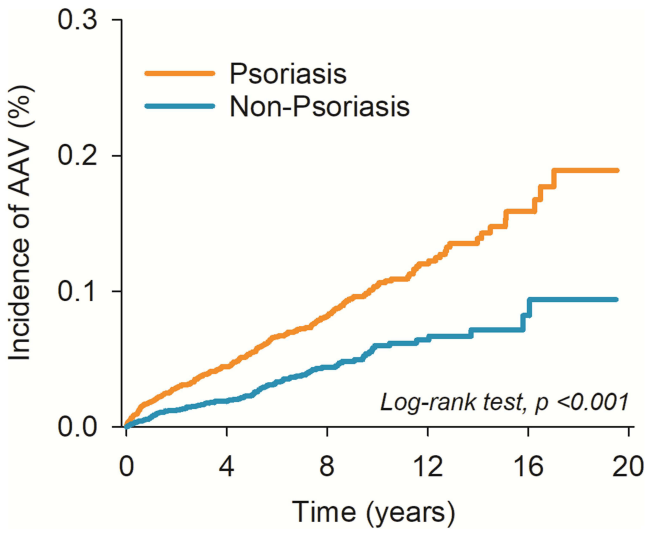

Figure 2 Kaplan-Meier analysis of the risk of ANCA-associated vasculitis.
Abbreviation: AAV, ANCA-associated vasculitis.

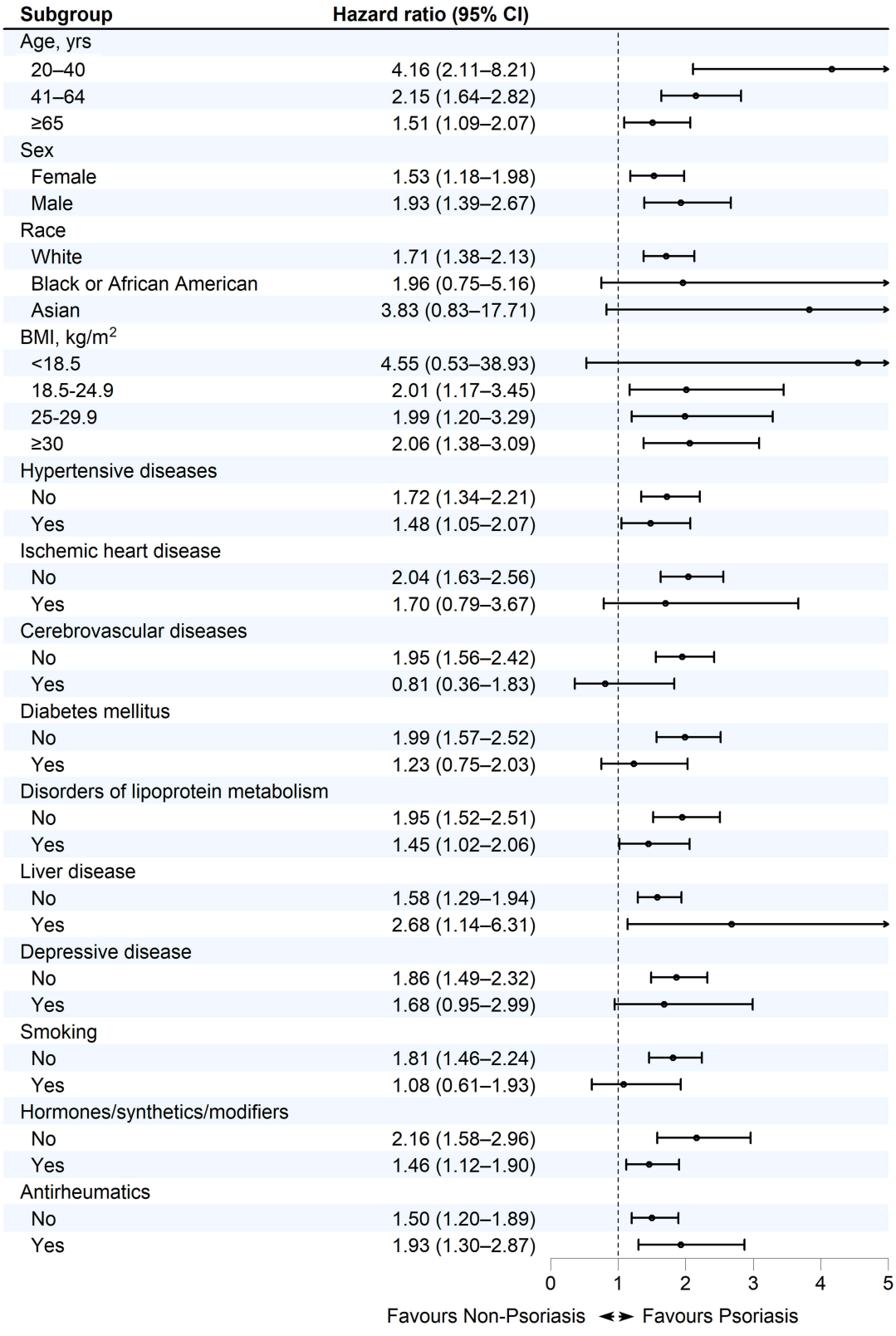

Figure 3 Forest plot for subgroup analysis of the risk of ANCA-associated vasculitis exposed to psoriasis compared to non-psoriasis.
Abbreviations: BMI, body mass index; CI, confidence interval.

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Table 3 Risk of Anti-Neutrophil Cytoplasmic Antibody Associated Vasculitis in Psoriatic Patients Treated with Biological Agents

	N	No. of AAVs Events	HR (95% CI)
Psoriasis without biological agents	21,677	14	Reference
Psoriasis with biological agents	21,677	17	1.31 (0.65–2.66)

Abbreviations: AAVs, anti-neutrophil cytoplasmic autoantibody-associated vasculitides; CI, confidence interval; HR, hazard ratio.

The primary strength of our study is its large sample size, which enabled us to conduct matching and subgroup analyses to account for a multitude of potential confounders. However, this study has some limitations. First, the possibility of over- or under-diagnosis of psoriasis, or AAVs cannot be entirely ruled out because of the inherent design of the EHR database. Second, the severity of psoriasis in our patient cohort was not described or compared because of the absence of relevant data. Third, the cause-effect relationship between psoriasis and AAVs was not explored because the GWAS data of AAVs were unavailable.[33,34]

Conclusions

Our propensity score-matched study demonstrated a significant association between psoriasis and an increased risk of AAVs. Biological agents use did not increase the risk of AAVs. These findings warrant further investigation in clinical practice.

Data and Computing Code Availability

The data that support the findings of this study are available from the EHR database but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available.

Ethics Approval

This study was approved by the Institutional Review Board of Chung Shan Medical University Hospital (Approval Number: CS2-21176).

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Disclosure

The authors report no conflicts of interest in this work.

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