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ASSOCIATION BETWEEN PSORIASIS AND DEMENTIA: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Psoriasis is a chronic immune-mediated skin disease characterized by systemic inflammation, while dementia, including Alzheimer's disease (AD) and vascular dementia, is increasingly recognized as an inflammatory neurodegenerative condition. Both disorders share common risk factors such as age, cardiovascular comorbidity, and metabolic disease. Whether psoriasis independently increases dementia risk remains unclear.
Objectives: To systematically evaluate the epidemiologic association between psoriasis and dementia, explore potential causality using genetic approaches, and assess the impact of systemic therapies, particularly biologics, on cognitive outcomes.
Methods: Following PRISMA guidelines, PubMed was searched for studies published between 2013 and 2025. Eligible studies included observational cohorts, case-control analyses, and Mendelian randomization studies examining dementia outcomes in patients with psoriasis. Data extraction was performed independently by two reviewers.
Results: Epidemiologic evidence from Taiwan, Korea, Denmark, and the UK suggests a modestly increased risk of dementia in psoriasis (odds ratios 1.10–1.25), with higher estimates in younger patients and for vascular subtypes. However, findings were inconsistent, with some cohorts reporting null or protective associations. Notably, systemic and biologic therapies were consistently associated with reduced dementia incidence. Mendelian randomization studies found no genetic evidence supporting psoriasis as a causal risk factor for AD, suggesting shared inflammatory and vascular pathways rather than direct causality.
Conclusions: Psoriasis may modestly increase dementia risk through systemic inflammation and comorbid vascular disease, rather than genetic liability. Systemic therapies, particularly biologics targeting TNF-α and IL-12/23, may confer neuroprotective benefits. Further prospective studies are warranted to clarify causality and therapeutic implications.

KEYWORDS

Psoriasis, Dementia, Alzheimer's Disease, Inflammation, Cognitive Impairment

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Background

The purpose of this systematic review is to comprehensively evaluate the epidemiologic relationship between psoriasis and dementia (Alzheimer's disease and vascular dementia), to assess potential causality using genetic instruments, and to examine the influence of psoriasis therapies, especially biologic agents, on cognitive outcomes.

Psoriasis is a chronic inflammatory skin disease characterized by skin scale, erythema and induration. Psoriasis prevalence estimates of around 2% of the population. Dementia is a general term for loss of cognitive function, including memory, thinking and judgement such that it interferes with daily living. Globally, the number of people living with dementia is projected to rise from 50 million in 2018 to 150 million by 2050. While around 45% of dementia cases are attributable to 14 modifiable risk factors, the cause of many cases is unknown.

There is a substantial evidence linking higher levels of inflammatory markers with Alzheimer's disease (AD), and vascular dementia. It is also possible that sleep, which is known to be disturbed in both psoriasis and dementia, suggest a common pathway relating to circadian dysregulation. Some observational studies show associations between chronic inflammatory conditions such as psoriasis and risk of Alzheimer's disease and other dementias, although evidence is conflicting [1].

Methods

The review was structured according to PRISMA guidelines. Articles identified via PubMed between 2013 and 2025 were included. Eligible studies reported on adults with psoriasis and subsequent risk of dementia or cognitive decline. Both observational and genetic causal-inference designs were included. Exclusions were case reports, commentaries without data, and dermatologic studies unrelated to neurocognition. Two reviewers independently extracted study design, sample size, comparator groups, outcome definitions, effect estimates, and adjustments.

Results

1. **Epidemiology**

In Ching-Chun Lin, Herng-Ching Lin and Hung-Wen Chiu study [2], among the 28,472 participants selected using the Taiwan Longitudinal Health Insurance Database 2000, psoriasis was diagnosed in 2.2% overall, 3.0% of dementia cases, and 1.5% of controls. After adjustment for monthly income, geographic region, urbanization level, diabetes, hyperlipidemia, hypertension, and coronary heart disease, the odds ratio (OR) for psoriasis in the dementia group was 1.46 (95% confidence interval (CI) 1.23–1.73; $p < 0.001$) compared with controls. Stratified analyses showed that the adjusted ORs for prior psoriasis with arthritis and without arthritis in the dementia group were 1.95 (95% CI 1.03–3.89) and 1.44 (95% CI 1.21–1.72), respectively. In contrast, no significant difference in the prevalence of prior bullous pemphigoid was observed between dementia cases (0.5%) and controls (0.4%).

According to J. Zhao, T. Li, J. Wang in a 17-year Taiwanese cohort study [1], psoriasis was associated with increased dementia risk, particularly for nonvascular dementia, although not for vascular dementia [3]. Risk of dementia among individuals with psoriasis: a nationwide population-based cohort study in Taiwan. Systemic therapy appeared protective, with reduced dementia risk observed among patients treated with systemic agents or biologics. In contrast, a larger Taiwanese cohort with over 100,000 psoriasis patients found no association between psoriasis and subsequent dementia. A Korean nationwide cohort including more than half a million psoriasis patients demonstrated a modest but significant increase in Alzheimer's disease risk. Again, systemic therapy was associated with lower risk, suggesting a potential protective effect [4]. Increased risk of Alzheimer's disease in patients with psoriasis: a nationwide population-based cohort study. A Danish hospital-based cohort also reported increased risk of vascular dementia in psoriasis patients. Findings from other populations were more variable. In the Rotterdam Study, psoriasis was associated with a reduced risk of dementia after long-term follow-up [5, 6]. UK hospital admission data indicated a significant association between psoriasis and subsequent dementia, consistent across both Alzheimer's and vascular subtypes. A Taiwanese case-control study found higher odds of prior psoriasis among dementia cases, even after excluding diagnoses made in the years immediately preceding dementia onset. Finally, a UK mortality study reported that patients with severe psoriasis were at increased risk of death from dementia, further supporting a possible link [7]. Associations between specific autoimmune diseases and subsequent dementia: retrospective record-linkage cohort study, UK. Overall, while the majority of studies suggest psoriasis may increase dementia risk, findings remain inconsistent across populations, and systemic therapy may attenuate or even reverse this association.

2. **Genetic theory**

In Yeung's et al. study [8], after excluding single-nucleotide polymorphisms (SNPs) in linkage disequilibrium and weak instruments (F < 10), 30 variants were retained for psoriasis, 11 for rheumatoid arthritis, 9 for multiple sclerosis, 9 for type 1 diabetes, 4 for sarcoidosis, 2 for Sjögren's syndrome, and 1 for giant cell arteritis. None of the instruments were located in the APOE locus or directly associated with Alzheimer's disease. Several variants were associated with selective survival, including 10 for psoriasis, 5 for rheumatoid arthritis, 1 for multiple sclerosis, 3 for type 1 diabetes, 2 for sarcoidosis, and 1 for giant cell arteritis. Conducted univariable Mendelian randomization analyses showed no clear associations between genetic liability to psoriasis, rheumatoid arthritis, type 1 diabetes, or sarcoidosis and Alzheimer's disease. Horizontal pleiotropy was detected for sarcoidosis with parental and maternal Alzheimer's disease. An inverse association was observed for rheumatoid arthritis with parental Alzheimer's disease after removal of survival-related SNPs, but this did not persist in multivariable models accounting for competing risk factors. Liability to type 1 diabetes showed a suggestive inverse association, which was attenuated after excluding survival-related variants. For multiple sclerosis, positive associations were observed with clinical and maternal Alzheimer's disease, consistent across analyses including MVMR and sensitivity models. In contrast, liability to Sjögren's syndrome showed a possible inverse association with maternal and sibling Alzheimer's disease, although analyses were limited by the small number of SNPs. Liability to giant cell arteritis was inversely associated with most Alzheimer's disease outcomes, with the exception of sibling Alzheimer's disease. Further sensitivity analyses were not possible due to the limited number of available SNPs.

3. **Immunopathogenic theory**

Both psoriasis and dementia are driven by inflammatory pathways, and their potential association has been the focus of recent investigation. Several studies have reported an elevated risk of dementia among patients with psoriasis, with risk ratios ranging from 1.10 to 1.25.64–67 [9]. Conversely, dementia has also been associated with an increased likelihood of developing psoriasis. Interestingly, one study published in 2018 reported a paradoxical protective effect, with psoriasis associated with a reduced dementia risk (HR = 0.54). In cases of severe psoriasis, the risk of dementia-related mortality was more than tripled (HR = 3.64), suggesting dementia constitutes an important cause of death in this population. Cross-sectional studies of prodromal dementia markers have also suggested that psoriasis patients are at higher risk of mild cognitive impairment, particularly affecting visuospatial function, verbal memory, and executive function although one study did not confirm this association. MRI-based studies have shown trends toward reduced hippocampal volume and greater white matter lesion burden in psoriasis patients, but these differences were not statistically significant. The mechanistic links between psoriasis and AD remain incompletely understood, but accumulating evidence suggests shared immunopathogenic pathways. Psoriasis lesions are characterized by infiltration of activated T cells and dendritic cells, which release pro-inflammatory cytokines including TNF-α and IL-23. TNF-α, a key cytokine in psoriasis pathogenesis, is also implicated in AD, where it exacerbates amyloid-β and tau pathology in vivo. Notably, TNF-α antagonists such as etanercept, adalimumab, and infliximab have been associated with improved cognitive function in AD patients and with reduced dementia risk in psoriasis cohorts (OR = 0.47). Similarly, the IL-12/23 axis, which is critical in psoriasis pathogenesis and targeted by monoclonal antibody therapies, has been implicated in age-related neuroinflammation. In mouse models of AD, increased cerebrospinal fluid levels of the IL-12/23 p40 subunit have been reported, and blockade of p40 reduced amyloid burden and improved cognition. Genetic studies also support a potential overlap between the two conditions. Apolipoprotein E (APOE), the strongest genetic risk factor for AD, influences amyloid deposition, tau phosphorylation, and cardiovascular risk. Variants of APOE have also been implicated in psoriasis susceptibility and severity. A meta-analysis of seven studies (966 psoriasis patients and 1,086 controls) showed that carriers of the ε3 allele or ε3/ε3 genotype had reduced psoriasis risk, whereas the ε2 allele increased susceptibility. Furthermore, a 2016 genetic study identified eight polymorphisms and two pleiotropic loci shared between AD and several immune-mediated diseases, including psoriasis, highlighting the role of inflammation in AD pathogenesis. These converging lines of evidence emphasize the importance of treating psoriasis as a systemic inflammatory disorder. Observational data indicate that psoriasis patients receiving systemic anti-inflammatory therapy for at least three months have a reduced risk of developing dementia. Similarly, a Korean cohort study stratified patients by treatment and found that those receiving systemic therapy had significantly lower incidence rates of AD compared not only with untreated psoriasis patients but also with the general population.

Discussion

The possible association between psoriasis and dementia has garnered increasing attention due to shared immunological and metabolic pathways. Psoriasis is a chronic immune-mediated disorder characterized by systemic inflammation, while dementia, particularly Alzheimer's disease (AD), is increasingly recognized as an inflammatory and neurodegenerative condition. Both entities share common risk factors, including age, cardiovascular comorbidities, obesity, diabetes, hypertension, and dyslipidemia, raising the question of whether psoriasis itself constitutes an independent risk factor for dementia or whether the observed associations are largely attributable to confounding.

Epidemiological data provide mixed evidence. Several large cohort studies from Taiwan, Korea, Denmark, and the UK have reported an elevated risk of dementia among patients with psoriasis, with odd ratios (OR) generally in the range of 1.10–1.25. Stratified analyses consistently suggest that the risk may be higher in younger or middle-aged patients, and that vascular dementia may be more strongly linked to psoriasis than nonvascular subtypes in some populations. In contrast, other cohorts, notably the large Taiwanese database study by Wu et al., did not confirm an association, while the Rotterdam population-based study even suggested a reduced risk. Such discrepancies may reflect differences in study design, population characteristics, diagnostic coding practices, and the presence of competing risk factors.

A consistent observation across multiple datasets is that systemic anti-inflammatory therapy—including both biologic and nonbiologic agents—appears to mitigate or even reverse the increased dementia risk. Patients with psoriasis who received systemic therapy demonstrated lower incidence of Alzheimer's disease, vascular dementia, and nonvascular dementia compared not only to untreated psoriasis patients but also, in some studies, to non-psoriatic controls. These findings underscore the potential neuroprotective role of immunomodulatory treatment, although the possibility of healthy-user and selection biases must be considered.

Pathogenetically, several mechanisms could link psoriasis with dementia. Both conditions involve dysregulated T-cell responses, activation of dendritic cells, and overproduction of proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-17, and IL-23. TNF-α is known to exacerbate amyloid-beta and tau pathology in experimental AD models, while IL-12/23 signaling contributes to age-related neuroinflammation. The observation that TNF-α inhibitors and IL-12/23 blockers not only ameliorate psoriasis but also may reduce dementia risk strengthens the hypothesis of shared inflammatory pathways. Moreover, epidemiological studies have shown increased dementia-related mortality in severe psoriasis, reinforcing the clinical significance of systemic inflammation.

Genetic studies, however, provide a more nuanced perspective. Mendelian randomization analyses, including the largest effort to date by Yeong et al. (2024), found no causal relationship between genetic liability to psoriasis and Alzheimer's disease. After exclusion of weak instruments and survival-related variants, liability to psoriasis was not significantly associated with Alzheimer's risk, in contrast to conditions such as multiple sclerosis, which showed positive associations. This suggests that psoriasis per se may not be a direct genetic driver of dementia, but rather that shared inflammatory and vascular risk pathways, together with environmental and lifestyle factors, mediate the observed epidemiological associations. ApoE polymorphisms, strongly linked to Alzheimer's disease, have also been implicated in psoriasis susceptibility and severity, but these overlaps may reflect pleiotropic inflammatory mechanisms rather than direct causality.

Taken together, the evidence suggests that psoriasis is associated with an increased risk of dementia in many, though not all, populations. The strength of association is modest and likely influenced by confounding factors. Importantly, the attenuation of dementia risk with systemic therapy points toward systemic inflammation as a key mediator. Genetic data argue against psoriasis being a direct causal risk factor, instead supporting the notion of shared immunopathogenic pathways that predispose to both conditions.

Conclusions

Psoriasis and dementia share common risk factors and inflammatory pathways, and epidemiological evidence generally supports a modestly elevated risk of dementia among patients with psoriasis. However, inconsistencies across studies and the absence of strong genetic evidence for causality indicate that psoriasis should not be considered an independent risk factor for dementia in isolation. Instead, the association is most plausibly explained by systemic inflammation, vascular comorbidity, and overlapping immunopathogenic mechanisms.

Systemic therapy for psoriasis, particularly biologic agents targeting TNF-α and IL-12/23, appears to reduce the incidence of dementia, highlighting the importance of controlling systemic inflammation. While current data are promising, causality cannot be firmly established due to the limitations of observational

designs and residual confounding. Prospective longitudinal studies and research are warranted to clarify the neurocognitive impact of psoriasis and its treatments. Until then, clinicians should remain vigilant regarding cognitive health in psoriasis patients, especially those with additional vascular risk factors, and consider systemic therapy not only for dermatological control but also for its potential broader benefits.

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