Original Article

Antidepressants and the Risk of Psoriasis Induction: A Case–Control Study

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Abstract

Background: Psoriasis (PSO) is a common chronic autoimmune skin disease with a significant psycho-socio-economic burden. Some antidepressants (ADs) such as fluoxetine and bupropion can induce or exacerbate PSO. This study aimed to investigate the correlation between ADs history before PSO onset, and the risk of PSO induction, in Isfahan province, Iran.

Materials and Methods: In this case–control study, 80 patients with PSO were selected by non-probability sampling method, and 80 healthy individuals were selected using simple random sampling. They were interviewed and medical information was recorded. Chi-square, Mann–Whitney, and Kruskal–Wallis tests for dichotomous or categorical data, and independent-sample t test for continuous data were used. Statistical significance was taken as $P \leq 0.05$.

Results: In this case–control study, a total of 160 individuals, 80 participants in each group, were included. The mean age of the total samples was 44.8 ± 16 years. Forty-three percent of the individuals were women. PSO familial history in the cases was significantly higher than the control group (OR = 11.94, $P = 0.001$). It was revealed that use of ADs by patients before PSO induction, was greater than the controls (OR = 2.78, $P = 0.058$).

Conclusions: Past history of ADs in the cases before PSO onset, was higher than the controls, indicating a possible association between ADs and the risk of PSO induction. This study can be effective to pay more attention to the possible complications of ADs and PSO risk factors. Accurate knowledge of PSO risk factors will be useful for better management and morbidity reduction.

Keywords: Antidepressants, autoimmune dermatologic disease, depression, psoriasis

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Introduction

Psoriasis (PSO) is one of the most common dermatologic disorders affecting 2% to 3% of the world's population. It is introduced by World Health Organization (WHO) as a serious health challenge due to a high psycho-socio-economic burden. Although PSO often presents with cutaneous manifestations, it is not confined to the skin and is considered a systemic inflammatory disorder. PSO has a genetic pattern, and 23% of the patients have a positive familial history. This disease can occur at any age; however, it often begins at adolescence (18- to 39-year-old) or adulthood (50- to 69-year-old). PSO has the same prevalence rate in the males and females. Normally, the immune cells, including dendritic cells, macrophages, regulatory, and helper T cells, can regulate the keratinocytes proliferation, maturation, and migration by secreting some cytokines. Due to genetic defects and external stimuli, these cells become over-activated and over-produce the cytokines making abnormal regulation of the keratinocytes. Recurrent periods of PSO are very annoying for the patients who have been affected throughout their lives, and no definitive

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lifelong treatment has been identified due to the lack of complete accurate identification of all involved factors in PSO pathogenesis.[1-11]

A wide spectrum of internal and external agents plays the important roles in PSO induction or progression. A range of external stimuli have been identified including stress, allergies, diet, infections, and skin injuries. Some medications are related to PSO pathogenesis, including antimalarial drugs (chloroquine), beta blockers, calcium channel blockers, captopril, lipid-lowering drugs, interferons alpha and beta, interleukins, anti-tumor necrosis factor alpha antagonists, CD20 monoclonal antibodies (rituximab), vascular endothelial growth factors (VEGFs) antagonists, granulocyte colony stimulating factors (GCSFs), anti-PD1 immune checkpoint inhibitors, acute withdrawal of systemic or potent topical corticosteroids, nicotine receptor antagonist, terbinafine, glyburide, penicillin, and lithium carbonate. Also, attention to rare reports of drugs cutaneous complications in connection with PSO is key. From this perspective, PSO development has been reported in several case reports during the course of tyrosine kinase inhibitors (TKIs) therapy like imatinib and nilotinib. Furthermore, some antidepressants (ADs) such as fluoxetine and bupropion can induce or exacerbate PSO.[12–33]

Since PSO exacerbation or induction has been reported for some ADs in limited studies with contradictory results, more assessments are needed to be more certain of this correlation. ADs have been frequently used for psychological disorders treatment. On the other hand, we should pay special attention to the underlying causes of these drugs consumption, especially chronic stress and depression in which they have a known role in PSO pathogenesis. This study aimed to determine the effect of ADs medication history before PSO onset, on this disorder induction, to gain a better understanding of PSO risk factors. This better knowledge can lead to more successful management and will ultimately help in the reduction of PSO burden and morbidity.[10,12,17–38]

Materials and Methods

In this case–control study, 80 patients with PSO were selected by non-probability sampling method, and 80 healthy individuals were selected by simple random sampling. The inclusion criteria were designed to control confounding variables. The case group that included patients with PSO was referred to the Al-Zahra hospital dermatology clinic and a private dermatology clinic, in Isfahan, Iran. Their inclusion criteria included informed consent to participate in this research, newly diagnosed PSO by a dermatologist in the past year based on clinical examination, skin biopsy and pathologic report, and absence of other chronic diseases except underlying causes that have led to ADs therapy. The control group consisted of healthy companions of the patients referred to another clinic at Al-Zahra hospital. Their inclusion criteria included informed consent of participation, and negative history of chronic diseases except underlying causes that have led to ADs therapy. Exclusion criteria included the participants' unwillingness to complete information, forgetfulness of medication information, and a positive history of other PSO-related medications. The selection bias approach in the cases was that the case selection from two different locations as two comprehensive dermatology centers. The ways to deal with the recall bias were the selection of newly diagnosed patients in a recent year, and also the individuals in both groups were excluded from the study if they had forgotten medication information.

The samples were studied after justification and consenting through interviewing and history taking in an information form. Historical data included demographic characteristics (age, sex, occupation, and education level), PSO family history, past drug history focusing on ADs and considering this history before PSO onset in the case group, medication period and dosage of ADs. Furthermore, the reasons for ADs therapy and whether these drugs were prescribed by a physician were also reviewed. If people with positive ADs history who entered this study did not have a document of psychiatric history and the reason for drugs taking, a psychiatric interview was conducted with them by experts. Data analysis was performed using SPSS software. Data were group-matched for demographic characteristics. Analysis was performed in a descriptive and analytical approach. In the descriptive section, the qualitative variables were reported as the percentage, and as the mean for the quantitative variables (standard deviation). Chi-square, Mann–Whitney, Kruskal–Wallis test, logistic regression, and t-test were used to assess the associations and differences between parameters. Statistical significance was taken as a P value ≤ 0.05 level.

Results

In this case–control study, a total of 160 individuals, 80 participants in each group, were included in the analysis. The mean age of the samples was 44.8 ± 16 years. Approximately, 43% of the total samples were females. Positive PSO family history was statistically different in the two groups ($P = 0.001$); however, the other variables between the case and control groups were not statistically significant [Table 1].

We drew a comparison between the case and control groups. There was no statistically significant difference between the drugs type and category, medication period, prescriber (doctor's prescription or self-prescribed), and reason for drug usage [Table 2].

As depicted in Table 3, using binomial logistic regression model and control of PSO family history, it was found that past history of ADs in the cases before PSO onset, was greater than the controls (OR = 2.78, $P = 0.058$). Nevertheless, this relation was statistically borderline, which could be due to the low sample size. Furthermore, PSO familial history was significantly correlate with the risk of PSO induction, assuming that the medication was the same in both groups (OR = 11.94. $P = 0.001$).

Table 1: Common characteristics of the cases and controls

Variable	All Cases, *n*=80	All Controls, *n*=80	*P*
Age (Year)	43.44±15.24	46.11±16.59	0.290
Gender (Male)	45 (43.8)	46 (57.5)	0.873
Positive PSO familial history	24 (30.0)	3 (3.8)	0.001
Positive ADs past history	14 (17.5)	6 (7.5)	0.056
Occupation			
Unemployed	29 (36.3)	30 (37.5)	0.797
Self-employed	34 (42.5)	29 (36.3)	
Government's employee	17 (21.3)	21 (26.3)	
Education level			
Primary school	4 (5.0)	8 (10.0)	0.707
High School, no degree	23 (28.8)	20 (25.0)	
High School degree	26 (32.5)	26 (32.5)	
University education	27 (33.8)	26 (32.5)	

Values are mean±SD or *n* (%). Chi-square, Mann-Whitney, and Kruskal-Wallis tests for dichotomous or Categorical data, and independent-sample t test for continuous data were used

Discussion

PSO is an inflammatory-mediated skin disorder following over-activation of the skin T cells and dendritic cells. These cells after migrating to the skin release pro-inflammatory cytokines such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor-α (TNF-α), which increases inflammation and the keratinocytes proliferation. Although not often overlooked, this disorder can lead to significant disability, and some estimate that its burden is comparable to other major diseases like chronic heart failure, chronic obstructive pulmonary disease, and cancers. According to the significant burden of PSO, accurate knowledge of its risk factors will be useful for better management. A wide range of the risk factors are correlated with PSO pathogenesis, and some medications such as ADs are associated with it. In this case–control study, our results showed that ADs history before PSO onset was higher in the cases ($P = 0.058$).[1–9,39,40]

In this study, the mean age of the patients was 43.44 ± 15.24 years. PSO can be seen in all ages from infancy to elderly. If PSO onset was in childhood, it will probably have a more severe progression. The mean age of the patients in a study by Farshchian *et al*., in Iran, was 34.6 years. Various studies around the world have shown that PSO is more common in people aged from 15 to 35 years.[1-4,7,8,41]

Our data revealed that there was no difference in the gender distribution. The findings of other studies suggested that both sexes are involved in the same proportion. Ashkevari *et al*. from Iran introduced that 53.1% of the patients were males and 46.9% were females. As a contrary, in the study by Hägg and the colleagues, in 5438 patients in Swedish centers, a higher PSO incidence was seen in men compared to women. Also, in a study by White O'Shea and Rogers, it was reported that PSO prevalence in men was higher than that in women.[1–4,7,8,42–44]

Our study did not show a significant difference between job and education level of the two groups. The previous data did not suggest that PSO is an occupational disease, although the occupational risk factors, for example, pollution, trauma, and irritants, may be correlated with PSO.[1-4,7,8,45]

Our findings revealed that positive PSO familial history was 30.0% among the patients with PSO ($P = 0.001$). Family history has been reported as a risk factor for PSO development in various studies, and the prevalence of positive PSO familial history has been seen in 4% to 91% of the patients.[1-4,6-8,46]

Our results suggest that ADs may be a risk factor for PSO induction (OR = 2.78, $P = 0.058$). This result was consistent with studies by Dowlatshahi *et al*., which showed that ADs consumption before PSO development was high in the patients, whereas Derme *et al*., in a study on the therapeutic outcomes in the treatment of patients with PSO with mental illness, by adding citalopram as a member of SSRIs (Selective Serotonin Reuptake Inhibitors) to TNF-α inhibitors, observed a significant decrease in PSO severity during follow-ups. Contrary to past researches, according to a recent published large cohort in Taiwan, low-dose SSRIs had protective effects on PSO development in the patients with MDD (major depressive disorder), but this study had no information about over-the-counter medications.[34,47,48]

This research manifested that the underlying causes of ADs consumption were not confined to psychological disorders, and the other reasons included sleep disturbances and headache. It is noteworthy that ADs usage is not limited to depression, stress, and anxiety controlling. SSRIs category, as the most common ADs in the clinics, has been mostly suggested to first-line treatment of depression, but it is also used in many other disorders. The popularity of SSRIs class is generally because of its easiness to use, high-dose safety, relative tolerance, reasonable cost, and wide range of the indications. In this study, fluoxetine was the most common ADs, and SSRIs was the most frequent ADs category in the patients before PSO onset. Our data introduced that some ADs users took these drugs without a doctor's prescription, so it is important to inform the public about the possible complications and arbitrary usage of ADs. Skin complications have not been introduced as common side effects for ADs. In some reports, PSO development or exacerbation following fluoxetine and bupropion has been observed. Fluoxetine is a member of SSRIs, and bupropion is a member of NDRIs (norepinephrine and dopamine reuptake inhibitors). The mechanism of most ADs is the increase of certain neurotransmitters such as serotonin and dopamine. The importance of serotonin in assisting macrophages to activate T cells has been demonstrated. Dopamine is a neurotransmitter playing a role in Th2 cell differentiation and the pro-inflammatory cytokines release from the mast cells. The role of dopamine in controlling Th2 cells and modulating the immune system is crucial in PSO. In addition to these drug-mediated mechanisms, if ADs were used due to depression, stress, or anxiety, these underlying disorders can

Table 2: Comparison of drug use status in the case and control groups

Variable	All Cases, *n*=80	All Controls, *n*=80	*P*
Medication period			
<1 year	4 (5.0)	3 (3.8)	0.618
1-5 years	8 (10.0)	2 (2.5)	
>5 years	2 (2.5)	1 (1.3)	
Drug dosage			
Low dose	10 (12.5)	4 (5.0)	0.904
High dose	4 (5.0)	2 (2.5)	
Drug type			
Fluoxetine	4 (5.0)	1 (1.3)	0.849
Citalopram	1 (1.3)	1 (1.3)	
Amitriptyline	1 (1.3)	1 (1.3)	
Nortriptyline	2 (2.5)	1 (1.3)	
Fluoxetine + Clomipramine	1 (1.30)	1 (1.3)	
Fluoxetine + Clomipramine + Sertraline	1 (1.3)	1 (1.3)	
Paroxetine + Clomipramine + Amitriptyline	1 (1.3)	0	
Fluoxetine + Amitriptyline + Sertraline	1 (1.3)	0	
Drug category			
SSRIs (Selective Serotonin Reuptake Inhibitors)	7 (8.8)	3 (3.8)	0.979
TCAs (Tricyclic Antidepressants)	3 (3.8)	2 (2.5)	
MAOIs (Monoamine oxidase inhibitors)	2 (2.5)	0	
TCAs + SSRIs	2 (2.5)	1 (1.3)	
Physician prescription			
Yes	9 (11.3)	5 (6.3)	0.613
No	5 (6.3)	1 (1.3)	
Underlying reasons for taking ADs			
Depression	6 (7.5)	1 (1.3)	0.254
Sleep disorder	2 (2.5)	1 (1.3)	
Headache	0	2 (2.5)	
Obsession	0	1 (1.3)	
Depression + Anxiety	3 (3.8)	1 (1.3)	
Post-traumatic stress disorder (PTSD)	2 (2.5)	0	
Bipolar disorder	1 (1.3)	0	

Table 3: Correlation between ADs past history and PSO induction using binomial logistic regression

Variable	Odds ratio	Std. Err	*Z*	*P*	95% conf. interval	
					Lower	Upper
ADs past history	2.78	1.502	1.89	0.058	0.964	8.015
PSO family history	11.94	7.634	3.88	0.001	3.411	41.804

synergistically play a role by the other pathophysiological mechanisms in PSO induction.[21,28–33,49–55]

Serotonergic function of SSRIs focusing on the pharmaco-genetic differences in the drugs metabolism may be effective on PSO pathogenesis. In a fantastic systematic review and meta-analysis study, Kato and the colleagues examined the pharmaco-genetic impact on the response to ADs therapy and the side effects incidence. This review revealed a significant association between some genotypes or polymorphisms and the better efficacy of these drugs including TPH 1 218C/C genotype, tandem repeats polymorphism within intron 2 (STin2) 12/12 genotype in Asians, and the Met variant within the BDNF 66Val/Met polymorphism. Regarding the side effects, pooled ORs of serotonin transporter gene promoter polymorphism (5-HTTLPR) l and HTR2A − 1438G/G were associated with a significant risk modulation (OR = 0.64, $P = 0.0005$) and (OR = 1.91, $P = 0.0006$). This significance became more robust when analyzed with side effects induced by SSRIs only (5-HTTLPR: $P = 0.0001$, HTR2A: $P < 0.0001$). Also, the participants' race was discovered as an important factor in response to treatment. In a recent study published by Ramesh *et al*., the relationship between genes involved in serotonin neurotransmission and the outcome of SSRIs therapy was investigated. They concluded that the 5HTTLPR polymorphism and the SLC6A4 intron 2 polymorphism were associated with treatment response, with the l/l genotype and 12-copy allele showing a tendency towards better outcomes.[56,57]

In order to clarify the correlation between ADs and PSO, most studies have focused on the development of anxiety, stress, and depression in patients with PSO due to the reduced quality of life after PSO onset. Fewer studies have looked at

the role of depression in PSO development or exacerbation, so more studies are needed to clarify this correlation. Psychiatric disorders such as depression and anxiety are usually diagnosed in more than 30% of patients with PSO, which is much more than the normal population. Dominguez *et al*. in a big prospective cohort of American female nurses found that depression was an independent risk factor for PSO induction. Besides, during PSO compared to other skin disorders, psychiatric complications are higher and quality of life is lower. Some previous studies have placed great emphasis on the need to further investigate the common pathogenesis mechanisms of psychiatric disorders and PSO, because the exact causal physiological correlations between these disorders have not yet been elucidated. Zafiriou and her colleagues in a recent publication point to the pivotal immune-pathological roles of IL-7 and IL-23 in the development or progression of PSO, depression, anxiety, and obesity, which suggests a biological link between these disorders. In a new systematic review, inflammation was introduced as a key factor in understanding the mechanism of depression effect on pro-inflammatory cytokines induction, including IL-1β, IL-6, and TNF-α and subsequent inflammatory diseases. It has also been hypothesized that fluoxetine may be involved in reducing inflammatory cytokines. In support of this finding, several recent studies have published the anti-inflammatory effects of fluoxetine and fluvoxamine in reducing systemic inflammation during Coronavirus Disease 2019 (COVID-19).[9,10,12,17–38,58–60]

Conclusions

This study can be effective to pay more attention to the possible side effects of ADs and PSO risk factors. It is recommended that those involved in prescribing ADs become more aware of cutaneous complications and be more careful in choosing the best choice for treatment. Sometimes counseling, empowerment, and family support can be considered as good alternatives. Our data introduced that some AD users took this treatment without a doctor's prescription. It is important to inform the public about the possible complications and arbitrary consumption of ADs. The limitations of this study include the recall and selection biases control as general limitations of case–control studies, and also the lack of a big database of PSO patients. As regards the effects on PSO induction or progression have been reported for limited ADs in few studies with paradoxical results, further studies are needed to fully understand this correlation. As for anti-inflammatory mechanisms of some ADs, in reducing inflammation and inflammatory diseases risk, it seems that PSO development is in the field of systemic inflammation of their usage causes such as chronic depression, stress, and anxiety. Due to the limitations of this study and the contradictory results of past researches, wider studies in animal models and clinics are proposed to discover the effects and mechanisms of ADs impacts on PSO. A better knowledge about PSO risk factors can lead to more effective management and will ultimately help in the reduction of its significant morbidity.

Ethics approval

The study protocol was approved by the Research Ethics Committees of Vice-Chancellor in Research, Medical University of Isfahan (Approval ID: IR.MUI.REC.1395.1.152).

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Conflicts of interest

There are no conflicts of interest.

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