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REVIEW

The Relationship Between Dermatologic Diseases and Dementia; A Review

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ABSTRACT

In some inflammatory skin diseases such as bullous pemphigoid, psoriasis and atopic dermatitis, inflammation is not limited to the skin but spreads systemically. Systemic inflammatory processes can activate proinflammatory cytokines, stimulate neurons and microglia in the brain, and trigger neuroinflammation. Ultimately, some skin diseases may affect the course of dementia and worsen the prognosis. Dementia, which progresses with a decrease in cognitive functions, can also trigger some skin diseases, accelerate their formation, and increase their severity. For example; bullous pemphigoid and dementia are two complex disease groups with multifaceted interactions. The strongest association among bullous pemphigoid comorbidities is with neurological diseases. The effect on cognitive impairment in patients with atopic dermatitis begins in infancy. The risk of cognitive impairment increases in the first year, especially in herpes zoster patients with trigeminal nerve involvement. In delusional parasitosis, which is a delusional disorder, patients first apply to dermatologists, and delusional parasitosis accompanies Lewy body dementia, one of the dementia types. This review aims to summarize dermatological diseases associated with dementia, such as bullous pemphigoid, psoriasis, atopic dermatitis, herpes zoster, crusted scabies, and delusional parasitosis, and to provide suggestions based on these relationships that may provide dermatologists, neurologists, and psychiatrists with a new perspective on the management of dermatological findings in patients with dementia.

Keywords: Dementia. Dermatitis. Atopic. Herpes Zoster. Pemphigoid. Bullous. Psoriasis.

Dermatolojik Hastaliklar ve Demans İlişkisi; Bir Gözden Geçirme

ÖZET

Büllöz pemfigoid, psoriasis, atopik dermatit gibi bazı inflamatuar deri hastalıklarında inflamasyon sadece deriye sınırlı kalmayıp sistemik yayılım gösterir. Sistemik inflamatuar süreç proinflamatuar sitokinleri aktive ederek, beyindeki nöronları ve mikrogliaları uyarabilir, nöroinflamasyonu tetikleyebilir. Sonuçta bazı deri hastalıkları demansın seyrini etkileyebilir, prognozu kötüleştirebilir. Bilişsel fonksiyonlarda azalma ile giden demans hastalığıda birtakım deri hastalıklarını tetikleyebilir, oluşumunu hızlandırabilir, şiddetini artırabilir. Örneğin; büllöz pemfigoid ve demans çok yönlü etkileşimi olan karmaşık iki hastalık grubudur. Büllöz pemfigoid komorbiditeleri arasında en güçlü ilişki nörolojik hastalıklarladır. Atopik dermatitli hastalarda bilişsel bozukluk üzerindeki etki bebeklik döneminden itibaren başlamaktadır. Özellikle trigeminal sinir tutulumu olan herpes zoster hastalarında ilk 1 yıl için bilişsel bozukluk riski artmaktadır. Sanrısal bir bozukluk olan delüzyonel parazitoz hastalığında ise hastalar ilk dermatologlara başvurmaktadır ve delüzyonel parazitoz demans tiplerinden Lewy cisimcikli demanslara eşlik etmektedir. Bu derleme demans ile ilişkili dermatolojik hastalıklardan büllöz pemfigoid, psoriasis, atopik dermatit, herpes zoster, krutlu uyuz, delüzyonel parazitoz hastalıklarını özetlemeyi ve bu ilişkilere dayanarak demans hastalarındaki dermatolojik bulguların yönetiminde dermatologlara, nörologlara ve psikiyatristlere yeni bir bakış açısı kazandırabilecek öneriler sunmayı amaçlamaktadır.

Anahtar Kelimeler: Demans. Dermatit. Atopik. Herpes Zoster. Pemfigoid. Büllöz. Psöriyazis.

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Dementia is an acquired disease characterized by a decline in cognition involving one or more cognitive domains (learning, memory, language, executive function, attention, perceptual-motor, social cognition). In recent years, the prevalence of dementia in aging populations has been increasing at an alarming rate. Approximately 50 million patients worldwide have dementia and dementias due to various causes are becoming more common in the elderly [1]. Alzheimer's disease is divided into different

groups including dementia with Lewy bodies, frontotemporal dementia, vascular dementia and mixed dementia. Although each of the dementia subtypes has its own pathological findings such as abnormal protein accumulation, the common etiology shared among all these conditions is cerebrovascular dysfunction that occurs at some point in the disease process[2].

Dementia is associated with many dermatologic diseases. Dementia leads to the triggering, progression and exacerbation of some skin diseases; on the other hand, skin diseases negatively affect the course and prognosis of dementia[3-6]. Chronic inflammation is involved in some forms of dementia such as Alzheimer's disease and dermatologic diseases such as psoriasis, atopic dermatitis and bullous pemphigoid[7,8]. Chronic inflammation in the central nervous system and periphery may participate in the pathogenesis of diseases both by permeability changes in the blood brain barrier and by causing microglial activation changes[9,10]. First-generation antihistamines including diphenhydramine, chlorpheniramine and hydroxyzine, which are frequently used in the treatment of pruritus in dermatology, easily cross the blood-brain barrier, leading to sedation, anticholinergic side effects and dementia in the elderly[11].

Several studies have shown the impact of dementia on dermatologic diseases and how these diseases may worsen the prognosis of dementia[1,4,5,12]. Although the relationship between Alzheimer's disease and skin diseases among dementia types has been summarized, there are not enough articles on dermatoses common in all dementia subtypes. This review aims to summarize the dermatological diseases associated with dementia such as bullous pemphigoid, psoriasis, atopic dermatitis, herpes zoster, crusted scabies, delusional parasitosis and to provide recommendations that may provide a new perspective to dermatologists, neurologists and psychiatrists in the management of dermatological findings in dementia patients based on these relationships.

This review was prepared by searching Turkish and English peer-reviewed original research articles and case reports published in Google Scholar and PubMed databases until March 2025. The key terms dementia and dermatologic diseases were used during the search. Unlike the reviews in this field, in this study, dermatologic diseases that are common in patients with dementia were included by examining their pathogenesis and multifaceted interactions separately.

The relationship between dementia and dermatological diseases is summarized in Table I.

Dementia and Bullous Pemphigoid

Like dementia, bullous pemphigoid is another disease with increasing prevalence in the elderly population. The relationship between bullous pemphigoid and dementia has been the subject of many studies. In this section, the multifaceted the interaction of dementia and BP in terms of pathogenesis, morbidity and mortality in recent studies will be is summarized.

Bullous pemphigoid (BP) is the most common vesiculobullous disease among subepidermal autoimmune bullous diseases. This disease typically affects the elderly and is characterized by bullous lesions on a localized or generalized erythematous background accompanied by pruritus[13]. It may show various clinical presentations. For example, only excoriation, prurigo-like lesions, urticarial and eczematous lesions may be observed without bullous lesions. BP is an autoimmune bullous disease associated with circulating autoantibodies against the basement membrane hemidesmosomal proteins BP180 (BP antigen 2 or type XVII collagen) and BP230 (BP antigen 1)[14].

Histopathology plays an important role in the diagnosis of BP. In biopsy material obtained from a newly formed intact bulla, subepidermal bullae dominated by eosinophils and neutrophils, accumulation of eosinophils and neutrophils in the dermis, and eosinophilic infiltration along the dermoepidermal junction may be observed. In the diagnosis of autoimmune bullous diseases, immunofluorescence, immunofluorescence in saline-separated skin, ELISA and immunoblotting methods are needed in addition to routine histopathologic examination[15].

First-line treatment of BP includes topical or systemic corticosteroids. Immunomodulatory (azathioprine, mycophenolate mofetil, methotrexate, dapsone) and anti-inflammatory (tetracycline, nicotinamide, dapsone) drugs are used in cases of resistant disease unresponsive to topical treatments or to minimize the adverse effects of chronic corticosteroid treatment[16-18]. Other treatment modalities include intravenous immunoglobulin, plasma exchange and immunadsorption. Since long-term administration of corticosteroids may cause serious side effects, recent studies support the use of new biological agents such as rituximab, omalizumab and dupilumab in treatment by utilizing their ability to selectively inhibit autoantibody formation and inflammatory cascade[19]. Studies examining the clinical and demographic characteristics of patients diagnosed with BP and the treatment process with comorbidities are important in understanding the etiopathogenesis of the disease, in the management of BP patients, in determining the appropriate treatment for the patient and in preventing comorbidities that may develop.

Table I. The Relationship Between Dementia and Dermatologic Diseases

	Clinical presentation	Association with dementia	Pathogenetic interaction	Precautions
Bullous Pemphigoid	-Chronic itching -Erythematous urticarial plaques with bullae+/-	-BP patients are at high risk of dementia -The incidence of BP is high in patients with dementia. -The association of BP disease and dementia increases mortality risk	Cross-reaction of neuronal BP1 antigens with cutaneous BP1 -Blood brain barrier disruption and neuroinflammation	-BP patients should be screened for cognitive impairment at regular intervals for the development of dementia
Atopic Dermatitis	-Chronic, pruritic, recurrent erythematous lesions	-Individuals with atopic dermatitis are at increased risk of mild cognitive impairment and dementia	-Chronic inflammation, blood brain barrier disruption, astrogliosis and neurodegeneration, Increased expression of -IL-6-associated interleukin amyloid-beta precursor protein	- Elderly patients with atopic dermatitis should be evaluated for possible cognitive impairment
Herpes zoster	-Erythematous vesicular rashes accompanied by severe pain throughout the dermatome	-Involvement of the ophthalmic branch of the trigeminal nerve increases the risk of dementia in the first year -Early antiviral treatment is associated with a lower risk of dementia -Herpes zoster vaccine reduces the risk of dementia	-Reactivation of herpes zoster virus triggers the formation of misfolded oligomers, increased neuroinflammation with accumulation of amyloid plaques and neurofibrillary tangles composed of hyperphosphorylated tau protein	-To be vigilant for mild cognitive impairment in elderly and immunosuppressed patients that may develop in the first year -Initiation of antiviral therapy quickly and at effective doses
Psoriasis	- Sharply circumscribed, erythematous plaques with silvery squames	- A 1.2-fold increased risk of Alzheimer's disease in patients with psoriasis - Psoriasis patients receiving systemic treatment have a lower risk of Alzheimer's disease than healthy controls	- Systemic inflammation - Decreased permeability of the blood brain barrier and neuroinflammation Involvement of -IL-12/IL-23 signaling in the development of amyloid-induced neurodegeneration	- Psoriasis patients should be monitored for mild cognitive impairment
Scabiyez	-Itching that wakes you up at night and increases in hot environments - Excoriated papules and silion between the fingers, wrists, axillary folds, periumbilical area, penis in men, areolar areas in women	- Dementia patients at high risk of crusted scabies	-Different clinical presentation in the elderly -51% asymptomatic -Delays in diagnosis due to late recognition by the caregiver	-More than the nationalization - Treatment refusal due to stigmatization, - Community information should be provided to prevent outbreaks
Delusional parasitosis	-Tactile hallucinations such as itching or the feeling that parasites are crawling on you	-Especially accompanies dementia with Lewy bodies		-Symptomatic treatment and psychiatric consultation
Drugs			-Blocking the common p40 subunit of -IL-12 and IL-23 reduces the number of Aβ plaques in Alzheimer's disease -Acitretin increases metalloproteinase ADAM 10 gene expression, inhibits Aβ peptide production and prevents the formation of amyloid plaques	- Systemic anti-inflammatory drugs reduce neuronal inflammation and systemic inflammation and ultimately reduce the risk of Alzheimer's disease

BP:Bullous Pemphigoid
IL:Interleukin
Aβ: Amyloid-beta
ADAM-10: metalloproteinase domain-containing protein 10

Many comorbidities have been described in BP patients, but the strongest association has been found between BP and neurologic diseases[20,21] BP is significantly associated with neurologic disorders. Having a neurologic disease for more than twelve months increases the development of BP 3-fold, especially in patients with dementia[20]. In recent studies investigating the relationship between Alzheimer's dementia and bullous pemphigoid, the risk of dementia in female BP patients over 80 years of age was found to be 40%[22].

Degenerative neuronal diseases may activate autoimmune factors against neuronal BP1 which may cross-react with cutaneous BP1 involved in BP

pathogenesis[1,20]. The presence of shared proteins between the central nervous system and the skin recognizes antigens in the central nervous system. This results in neuroinflammation and impaired permeability of the blood-brain barrier[1]. A recent study in BP patients found that high serum antibody levels against BP1 correlated with the presence of neurologic diseases[10,23].

The risk of Alzheimer's disease in BP patients is approximately 39%[22]. Decreased cognitive abilities and a higher risk of cognitive impairment in BP patients have been shown in many studies[24,25]. Given that the onset of dementia in BP is relatively insidious, it is often overlooked in clinical practice. Since dementia in BP may start as mild cognitive impairment, patients should be evaluated periodically in terms of cognitive impairment.

Due to the diversity of clinical presentations in BP, patients may be followed for long periods without a diagnosis of BP. In individuals with neurological disease, skin symptoms such as itching, erosion or eczema may go unnoticed by caregivers or the duration of symptoms may be easily ignored. On the other hand, in patients with neurological diseases such as dementia, the constant urge to scratch and the associated involuntary scratching may lead to increased mechanical trauma and rapid progression of skin lesions, thus leading to early onset of bullous lesions. The wide clinical diversity may lead to delayed diagnosis and increased morbidity and mortality.

In a meta-analysis examining the relationship between dementia and mortality in patients with BP, the accompaniment of dementia to BP was found to be associated with poor prognosis and increased the mortality rate almost 2-fold[27]. Therefore, caution should be exercised in patients with BP and concomitant dementia. Mortality may be increased by the neurologic diseases themselves and may be further increased by the autonomic dysfunction of patients with bullous pemphigoid; therefore, caution should be exercised in patients with bullous pemphigoid and concurrent dementia and stroke. When following both disease groups, it should be kept in mind that these diseases may present with atypical clinical forms in order to make an early diagnosis and prevent complications that may develop. Dermatologists should periodically screen BP patients for cognitive impairment in terms of dementia development. At the same time, patients with dementia presenting to the clinic should be evaluated in terms of atypical clinical presentations with pruritus and excoriation without bullae.

Dementia and Atopic Dermatitis

Atopic dermatitis is one of the most common chronic diseases in the world and is an inflammatory skin disease characterized by chronic, pruritic, recurrent erythematous lesions[8]. Although it is common in childhood, it may affect individuals of all ages. It shows a bimodal course, especially in early childhood and middle-aged individuals. Its association with systemic diseases recently suggests that it may be a systemic disease[27].

Although it has a very complex pathogenesis, it develops from a background of chronic inflammation triggered by genetic, immunologic and environmental factors. The mechanisms responsible for pathogenesis are microbiome alteration following barrier dysfunction, triggering immunologic changes as a result of allergen exposure and neuroinflammation involved in the formation of pruritus[28,29]. Th2 cells and related cytokines are predominant in atopic dermatitis[30].

The main symptom in the formation of the disease is pruritus, especially when flare-ups are not adequately controlled, cutaneous inflammation increases through mechanical stimulation, resulting in exacerbation of pruritus and disruption of the integrity of the skin barrier[27]. A vicious cycle of itching and scratching then develops[31]. It is reported that pruritus usually worsens at night and leads to severe sleep disturbances[31]. However, psychological distress and health problems are also increased, including anxiety, depression and attention deficit/hyperactivity disorder. However, it is not yet clear whether neuropsychiatric comorbidities in AD are caused by persistent and intense pruritus, poor sleep and stigmatization, or are direct consequences of proinflammatory cytokines produced by AD inflammation and accelerated neuroinflammation[32,33]. However, this condition has a significant impact on the quality of life of both patients and their families[34,35].

The main step in treatment is patient education, moisturizer application and repair of the barrier layer of the skin. Subsequently, itching is controlled and inflammation is suppressed. Various drugs, including topical and systemic therapies, can be used in treatment. Topical corticosteroids, calcineurin inhibitors and moisturizers are included[36]. Another topical drug that has completed a phase 3 study is the phosphodiesterase 4 inhibitor chrysabold[36]. In systemic treatment, antihistamines and immunomodulatory drugs (systemic steroid, cyclosporine, methotrexate, azathioprine, mycophenolate mofetil) are used[36]. Dupilumab, nemolizumab, mepolizumab, omalizumab are biological agents used in treatment[36].

Chronic inflammation, itching-scratching vicious cycle and resulting sleep disorders, stress and decreased quality of life are expected in atopic dermatitis. In a review of sleep and neurocognitive impairment, it was shown that quality sleep in infancy is important for neurocognitive development, and quality sleep in childhood and adolescence is important for the development of memory, language, sustained attention, encoding and consolidation of long-term memories, processing speed and executive functions[37-41].

Recent studies show that cognitive dysfunction, including all-cause dementia and mild cognitive impairment, is higher in the elderly population with atopic dermatitis than in the normal population[42,43]. In addition, a meta-analysis by Gwak et al. found an increased risk of all-cause dementia, especially Alzheimer's disease, in individuals with atopic dermatitis[44]. This relationship has been linked to many inflammatory hypotheses. Proinflammatory cytokines and chemokines are involved in the pathogenesis of both atopic dermatitis and Alzheimer's dementia[45,46]. Proinflammatory cytokines cross the blood-brain barrier and modulate neuroimmune pathways[47]. In addition, eotaxin, one of the chemokines, crosses the blood-brain barrier, stimulates microglia and generates reactive oxygen products in cells in the central nervous system and produces a cytotoxic effect on neurons[9]. As a result, cutaneous inflammation in atopic dermatitis may be thought to contribute to neurodegeneration in Alzheimer's disease by causing astrogliosis, microglia activation and release of inflammatory factors[48]. Furthermore, interleukin (IL)-6, a cytokine, is involved in the expression of amyloid-beta (Aβ) precursor protein[49]. The presence of circulating proinflammatory mediators and immune alterations may accelerate Aβ accumulation by impairing the Aβ clearance function of microglia in the brain[50,51]. However, prospective studies are needed to clarify the relationship between dementia and atopic dermatitis.

The finding that atopic dermatitis may be a potential risk factor for cognitive dysfunction and all-cause dementia in middle-aged and older adults should be a warning especially for dermatologists and patients with atopic dermatitis should be evaluated for possible cognitive impairment. When planning treatment, the effects of sleep disorders on long-term neurodevelopmental development should be taken into account, and it should be aimed to improve sleep quality and quality of life in addition to improving skin symptoms.

Dementia and Scabies

Scabies is an ectoparasitosis caused by Sarcoptes scabiei var. hominis. It can affect individuals of any age regardless of gender or race. Transmission from person to person can be direct (sexually, through close contact) or indirect (using personal belongings of scabies patients, using the same common living space). The diagnosis is easily made in most cases with anamnesis and typical clinical findings. The main complaint of the patients is itching that wakes them up at night and increases in warm environments. The presence of other itchy individuals in the family is another important finding supporting the diagnosis of scabies. The most common areas affected and excoriated papules are between the fingers, wrists, extensor faces of the extremities, axillary folds, sides of the trunk, periumbilical area, buttocks, penis in men and areolar areas in women[52]. Observation of gray-brown 0.5-1 cm comma-shaped tunnels caused by the mite is helpful in the diagnosis. Typical history and clinical findings constitute the main step in the diagnosis. Dermoscopy can be used to help the diagnosis. On dermoscopy, a linear segment corresponding to the cilium and characteristic small black triangular structures corresponding to the pigmented anterior part of the sarcopt at its end are seen and called jet sign or delta sign[53]. The most common form of scabies is classical scabies, which has two different clinical forms, classical scabies and crusty scabies, as well as forms with atypical clinical course. Scabies with crusty scabies is the most contagious rare form which is mostly seen in immunocompromised individuals and in patients with neurologic diseases that reduce scratching and itching, where the diagnosis can be easily missed[52]. It should be taken into consideration that the clinic may be atypical in patients with dementia and may progress to crusted scabies, which is highly contagious. Not delaying the diagnostic process and organizing an effective treatment in diagnosed patients constitutes an important step in preventing a large number of cases and preventing outbreaks. The increasing incidence of scabies, especially in recent years, constitutes an important public health problem.

In a study of elderly residents of nursing homes who were diagnosed with scabies by general practitioners and nursing home staff based on clinical symptoms, it was found that the time from the onset of clinical symptoms to the diagnosis of scabies was long and these patients were examined many times by general practitioners and misdiagnosed with other skin conditions such as eczema and received different treatments[12]. As a result, many of them were not diagnosed with scabies until other cases emerged, leading to an increased risk of transmission not only among patients but also among their caregivers and families, which can lead to outbreaks.

The signs and symptoms of scabies in the elderly differ from the classical symptoms. In a study by Cassel et al. 51% of patients diagnosed with scabies in

elderly people staying in a nursing home were asymptomatic and the majority of patients had a diagnosis of dementia[54]. In patients with dementia, itching-scratching action and expressive abilities decrease, it becomes very difficult to detect clinically and this situation brings with it an increase in the number of mites and progression to crusty scabies. Especially in dementia patients who require close contact care, the increase in the number of mites in crusty scabies and the very high level of contagiousness are effective in the formation of epidemics. Scabies outbreaks in healthcare facilities cause serious morbidity for other patients and staff as well as administrative burden[55]. Family physicians play an important role in the early diagnosis of patients in this group. In addition, the diagnosis of scabies brings stigmatization in society[56]. For this reason, especially family physicians may have difficulty in making a diagnosis in patients who do not show classical symptoms and whose relatives are asymptomatic. Relatives may not accept the diagnosis, refuse treatment and conceal it. Public information is also needed to prevent outbreaks and reduce cases.

Dementia and Herpes Zoster

Varicella zoster virus (VZV), a DNA virus, belongs to the alpha herpesviridiae family and is a highly contagious virus known as human herpes virus-3 (HHV-3). Primary infection occurs as varicella and is transmitted by direct lesion contact or droplet transmission. After primary infection, VZV settles in the cranial nerves and dorsal root ganglia and remains latent. In cases of advanced age, immunocompromised, stress and trauma, the virus reactivates and produces a clinical picture of herpes zoster characterized by erythematous, vesicular eruptions accompanied by severe pain along the dermatome innervated by the ganglion[57]. Thoracic dermatomes are most commonly involved, followed by cranial (most commonly the trigeminal nerve), lumbar and cervical involvement. The diagnosis is usually based on clinical findings. The diagnosis can be confirmed by Tzanck smear. Confirmatory tests include direct fluorescent antigen test, viral culture, polymerase chain reaction (PCR) to detect VZV DNA in skin lesion and organ samples[58]. It is recommended to start treatment within the first 72 hours after the onset of vesicular rash. Systemic acyclovir, valacyclovir, famciclovir and brivudine antiviral agents are used in treatment[57].

There are views emphasizing that involvement of cranial nerves is associated with dementia. Hypotheses on the mechanisms by which VZV infection causes dementia suggest that reactivation of the virus triggers the formation of misfolded oligomers, increasing neuroinflammation through the accumulation of amyloid plaques and neurofibrillary tangles composed of hyperphosphorylated tau protein. VZV may also directly infect astrocytes and stimulate intracellular amyloid production and the aggregation of amyloid fibrils in the extracellular environment[5,59-61]. Another hypothesis is that herpes zoster causes nerve damage by causing cerebral vasculopathy and ischemia, especially when it involves the cranial nerves[62]. The results of studies investigating the relationship between herpes zoster and dementia vary[6]. Involvement of the ophthalmic branch of the trigeminal nerve increases the risk of dementia in the first year[63,64]. Recent studies emphasize that early initiation of antiviral treatment reduces the risk of dementia[65,66]. There are also studies emphasizing that the herpes zoster vaccine approved by the FDA in 2006 reduces the risk of dementia[67].

The results of studies examining the relationship between herpes zoster and dementia contradict each other. This may vary depending on the duration of herpes zoster diagnosis, the prevalence of skin symptoms, the time of initiation of antiviral treatment, the presence of concomitant immunosuppressive conditions, the severity of dissemination and inflammation. It needs to be supported by more comprehensive studies. Dermatologists should be especially careful about mild cognitive impairment that may develop within the first year in elderly and immunosuppressed patients diagnosed with herpes zoster and should emphasize the importance of rapid and effective initiation of antiviral treatment.

Dementia and Psoriasis

Psoriasis is one of the most common chronic autoinflammatory skin diseases. Clinically, the disease is usually characterized by sharply circumscribed, erythematous plaques with silvery scales. It is influenced by ethnicity, genetics and environmental factors; therefore, its prevalence varies between countries, with a prevalence of approximately 2-3% in the population. The most commonly affected areas are the knee, elbow, sacral region and extensor surfaces of the extremities. Scalp, joints and nails are also affected[68,69]. Psoriasis shows four different clinical patterns according to the morphologic appearance of the lesions; plaque, guttate, pustular and erythrodermic forms. According to their localization, they can be classified as scalp psoriasis, palmoplantar psoriasis, inverse psoriasis, nail psoriasis, etc.[70].

The pathogenesis of the disease is not known exactly. Genetic and environmental factors are thought to play a role together. It is a complex, immune-mediated disease in which T lymphocytes, especially Th1 lymphocytes, predominate and dendritic cells and cytokines (interleukin [IL] 23, IL-17 and tumor

necrosis factor [TNF]) play a central role. Typical clinical findings such as squamous, induration and erythema are the result of hyperproliferation and abnormal differentiation of the epidermis, inflammatory cell infiltrates and vascular dilatation[71,72]. The first step in treatment is the use of moisturizers, keratolytics and anti-inflammatory preparations. Topical treatment includes corticosteroids, vitamin D analogs, retinoids and calcineurin inhibitors. Another treatment option is phototherapy protocols such as narrowband UVB and PUVA. Advances in the pathogenesis of psoriasis have led to the use of targeted biologic agents. Systemic treatment includes methotrexate, psoretin, cyclosporine, fumarate, apremilast, immunomodulatory drugs and biological agents. Tumor necrosis factor alpha inhibitors (adalimumab, infliximab, etanercept), IL-17 inhibitors (secukinumab, ixekizumab), IL-23 inhibitors (ustekinumab, guselkumab, risankizumab) are biological therapies approved by the FDA in the treatment of psoriasis[73]. Improved pathogenesis and related treatment protocols have improved the quality of life of patients and reduced the development of comorbidities due to suppression of systemic inflammation. In recent studies, psoriasis patients receiving systemic treatment were found to have a lower risk of Alzheimer's disease compared to healthy controls[74].

The fact that psoriasis is proven to be a systemic inflammatory disease in terms of its predominantly cutaneous involvement and accompanying comorbidities has brought its relationship with dementia, which is quite common, to the agenda and recent studies have been conducted for this purpose. In a meta-analysis conducted in 2022, retrospective cohort studies, most of which were conducted in Asia, the United States or Europe, were analyzed and a 1.2-fold increased risk of Alzheimer's disease was found in psoriasis patients[75]. The results of many studies in the literature support this relationship between psoriasis and Alzheimer's disease[76-78]. However, there are studies showing an inverse relationship in the literature[79,80]. Differences in study results may vary depending on the duration of the disease, treatment protocols and ethnicity of the patients. In order to fully elucidate the relationship between psoriasis and dementia, existing studies need to be supported by more comprehensive studies in which disease severity, socio-demographic data, treatment methods used, and the presence of comorbidities that trigger inflammation are also examined.

In the presence of systemic inflammation, proinflammatory cytokines are activated, neurons and microglia in the brain are stimulated, ultimately contributing to the pathogenesis of Alzheimer's disease[81]. As a result of the decrease in the permeability of the blood brain barrier with increasing age, activated cells and proinflammatory cytokines in the periphery cross the barrier and neuroinflammation is triggered in systemic inflammatory diseases[81]. Furthermore, IL-12/IL-23 signaling, which plays a key role in the pathogenesis of psoriasis, has been suggested to play a role in the development of amyloid-induced neurodegeneration[82]. Suppression of systemic inflammation is important for neurocognitive functions. The use of systemic therapy in psoriasis patients has been associated with a lower risk of dementia[83,84]. Therefore, it is important to apply early inflammation suppressive treatments in psoriasis patients in order to prevent dementia.

Psoriasis patients may have early deterioration in verbal memory, executive functions and attention in the long term[85]. In recent studies, patients with psoriatic arthritis were investigated for mild cognitive impairment and a higher risk was found compared to healthy controls[86]. Similarly, a large-scale study found that problems related to the cognitive domain concerned 20% of patients[87]. Gisondi et al. reported that the incidence of mild cognitive impairment was higher in patients with chronic plaque psoriasis than in controls, suggesting an increased risk of developing Alzheimer's disease in patients with psoriasis[85]. Mild cognitive impairment is higher in patients with chronic plaque psoriasis than in the general population[85]. The relationship between skin and brain has implications at multiple levels (immunologic, psychological and endocrinologic). Therefore, dermatologists should be aware of mild cognitive impairment that may develop early in patients with psoriasis.

Conditions Associated with Dementia and Dermatologic Symptoms

Dementia is a highly prevalent condition in society, affecting an estimated 50 million people worldwide[1]. Although 70% FH is the most common type of dementia, vascular dementia, dementia with Lewy bodies, Parkinson's disease dementia and frontotemporal dementia are among the types of dementia[3]. Dementia-related psychosis, including delusions and hallucinations, tends to increase as the duration and severity of the disease increases, contributing to increased hospitalization, cognitive decline and caregiver burden. Although a variety of symptoms can occur in all types of dementia, visual hallucinations are particularly common in dementias with Lewy bodies (dementia with Lewy bodies and Parkinson's disease dementia).

Ekbom syndrome is a delusional disorder in which the body is infested with parasites or insects, which may be accompanied by tactile hallucinations, such as

itching or the sensation of parasites crawling on it. It usually occurs in women in the fifth decade[88]. Typically, symptoms include peeling and scarring of the skin as a result of efforts to eradicate the parasites[88]. The most commonly affected areas are the scalp, face, mouth, eyes, arms, breasts and genitalia[88]. To prove the presence of parasites, patients may bring a matchbox or other container into the doctor's office in which they place various dust particles, skin fragments, fibers, etc. This behavior is often referred to as "matchbox sign" and "specimen sign". It may occur due to primary, secondary and organic causes and may also accompany neurodegenerative diseases, especially dementia with Lewy bodies.

The relationship between dementia and delusional parasitosis is mostly in the form of case reports. In a case report in 2020, a 72-year-old patient who presented with the complaint of insects in his head and a patient with no previous personal or familial history of neuropsychiatric disorder was presented. In the detailed questioning of the patient, it was found that 2 years ago, he had difficulty finding an address, money management was impaired and he had difficulty using a smartphone. Two years later, he started to complain of "ants" crawling on his head. He claimed that an "anthill" was covering his scalp and started scraping and picking it off. This belief persisted despite being denied by his family. He also began to hear his name being called or the doorbell ringing several times a day, as well as sometimes hearing sounds that others could not hear, such as the whistling and humming of insects. He also had sleep and movement disturbances at night and depressive symptoms. The patient was diagnosed with dementia with Lewy bodies as a result of investigations[89]. Similarly, an 89-year-old female patient initially complained of oral senestopathy, followed by a belief that she had filaria infection in her nose and eyes, and was diagnosed with dementia with Lewy bodies after the onset of parkinsonism and cognitive impairment[90]. These cases emphasize to dermatologists that resistant parasitosis delusions are one of the findings of dementia and cognitive functions of patients with parasitosis should be evaluated.

In the two cases reported by Taomato et al. visual and tactile delusions were present with cognitive impairment. In both cases, mild cognitive impairment accompanied the onset of delusions. In case 1, the patient used insecticide to get rid of insects. In case 2, the patient had a history of self-immolation to get rid of insects. In addition, he had taken actions such as using insecticides and consulting many dermatologists[91].

In a 2020 case report from Turkey, a patient with delusional parasitosis who was followed up for 2 years with a diagnosis of dementia was presented. The patient was diagnosed with dementia 2 years ago and the presence of skin picking with the belief that he was infected with insects 1 year later was evaluated as secondary delusional parasitosis[92].

Patients self-mutilate in different ways, such as erosions, peeling, cuts, obsessive cleaning and even burning to remove parasites[93]. Due to the prevalence of skin manifestations, these patients consult a dermatologist rather than a psychiatrist[93]. Since patients do not accept the view that their delusions are not real, there is a significant delay in presentation to a psychiatrist and initiation of psychopharmacologic treatment[94]. In the approach to patients with delusional parasitosis admitted to the dermatology unit, it should not be tried to make them believe that insects do not exist, and after symptomatic treatment is organized for the skin lesions, a safe bond should be established with the patient and the patient should be appropriately referred to psychiatry.

Drugs Used in Dementia and Dermatologic Diseases

H1-antihistamine drugs are classified as first or second-generation antihistamines. First generation H1-antihistamines have anticholinergic effects[95]. Anticholinergic drugs block acetylcholine, a neurotransmitter in the central or peripheral nervous system. First generation antihistamines cross the blood brain barrier and cause drowsiness, sedation, somnolence and fatigue[95]. Drugs with anticholinergic activity include oxybutynin (urinary incontinence drugs), antidepressants, antiparkinsonian drugs and antihistamines. Although many studies have argued that antihistamines used to suppress allergic diseases are associated with the risk of dementia, a large-scale study conducted in 2018 found that antidepressant, urological and antiparkinsonian drugs with anticholinergic effects were associated with an increased incidence of dementia up to 20 years after exposure[96]. First generation H1-antihistamine drugs should be avoided especially in elderly people with dementia due to their side effects.

In particular, a significant reduction in the incidence of Alzheimer's disease has been found with systemic drugs used in the treatment of psoriasis (acitretin, methotrexate, cyclosporine and biological agents[74]. Anti-IL-12/23 p40 monoclonal antibody is a biological agent targeting the IL-23/T helper 17 axis used in the treatment of psoriasis. In recent studies, blockade of the common p40 subunit of IL-12 and IL-23 reduced the number of Aβ plaques in Alzheimer's disease and resulted in improvement in cognitive impairment in a mouse model[82,97]. In addition, the use of systemic anti-inflammatory drugs reduces the incidence of cardiovascular diseases in psoriasis patients. In a mouse model of Alzheimer's disease,

acitretin has been reported to increase gene expression of the metalloproteinase ADAM 10 (metalloproteinase domain-containing protein 10), inhibit Aβ peptide production and prevent the formation of amyloid plaques[98-100]. Systemic anti-inflammatory drugs reduce neuronal inflammation and systemic inflammation and ultimately reduce the risk of Alzheimer's disease[74]. However, the effects and mechanisms of these drugs on inflammation need to be supported by studies. Prospective studies in which diseases and comorbidities are considered separately are needed.

Conclusion

The fact that the skin and nervous system originate from a common germ and that the relationship between the skin and the brain is multifaceted with endocrinologic and immunologic interactions may explain why dermatologists are the first health professionals to encounter psychiatric and neurologic diseases. Many skin symptoms can be observed in patients with dementia. These symptoms may include nonspecific findings such as pruritus, xerosis, pressure sores, oral hygiene disorders, which increase with aging, as well as association with common dermatologic diseases such as psoriasis, BP, and atopic dermatitis associated with chronic inflammation and autoimmunity. Cognitive function assessments by dermatologists can help diagnose dementia at the level of mild cognitive impairment and treatment of skin diseases associated with systemic inflammation may prevent the development of dementia.

This review summarizes dermatologic manifestations and diseases in patients with dementia and provides guidance to dermatologists on the care of these patients. It also emphasizes the importance of a multidisciplinary approach to the management of dementia and suggests that skin diseases should be considered as part of this approach.

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Idea and design: Ç.H.;Data collection and processing:Ç.H., H.B.Y.; Analysis and interpretation of data: H.B.Y.; Writing of significant parts of the article: H.B.Y.
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