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COVID-19 PANDEMİSİNİN OTOİMMÜN BÜLLÖZ HASTALIKLAR ÜZERİNDEKİ ETKİSİ: RETROSPEKTİF BİR ANALİZ

THE IMPACT OF THE COVID-19 PANDEMIC ON AUTOIMMUNE BULLOUS DISEASES: A RETROSPECTIVE ANALYSIS

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ÖZET

AMAÇ: Otoimmün büllöz hastalıklar (OBH) cilt ve mukozanın yapısal proteinlerine karşı otoimmün bir yanıtla karakterize edilen nadir ancak potansiyel olarak ciddi durumlar olup önemli morbidite ve mortaliteye yol açar. COVID-19 (Koronavirüs Hastalığı 2019) pandemisi, bağışıklık sistemini etkileyebilen ve potansiyel olarak otoimmün hastalıkları tetikleyebilen veya kötüleştirebilen büyük bir küresel sağlık krizini temsil etmektedir. Bu çalışmada, pandemi öncesi ve sonrası dönemde OBH tanısı alan hastaların klinik özellikleri, insidansı ve tedavi yanıtları karşılaştırılarak, COVID-19'un OBH üzerindeki olası etkilerinin değerlendirilmesi amaçlanmıştır.

GEREÇ VE YÖNTEM: Mart 2016 - Mart 2024 tarihleri arasında bir üniversite hastanesinde OBH tanısı alan hastalar retrospektif olarak incelendi. Demografik veriler, hastalık alt tipleri, klinik şiddet, tedavi yanıtı ve hastaneye yatış gereksinimleri değerlendirildi.

BULGULAR: Çalışmaya toplam 134 hasta dahil edildi; bunlardan 62'si (%46,3) pandemi öncesi, 72'si (%53,7) pandemi sonrası teşhis edildi. Pandemi sonrası dönemde büllöz pemfigoid insidansında anlamlı bir artış gözlendi (%59,7, p=0,03). Otoimmün büllöz hastalıkların insidansı yaşla birlikte artış gösterdi. (p=0,02). Hastalığın şiddetinde anlamlı bir fark bulunmamasına rağmen, pandemi sonrası dönemde tedaviye dirençli vakalar daha sık görüldü.

SONUÇ: Çalışmamız, COVID-19 pandemisinin özellikle yaşlı bireylerde OBH insidansının artmasına ve tedavi direncinin gelişmesine sebep olabileceğini düşündürmektedir.

ANAHTAR KELİMELER: Otoimmün büllöz hastalıklar, Pemfigus vulgaris, Büllöz pemfigoid, COVID-19 pandemisi, Otoimmün hastalıklar

ABSTRACT

OBJECTIVE: Autoimmune bullous diseases (AIBDs) are rare but potentially severe conditions characterized by an autoimmune response against structural proteins of the skin and mucosa, leading to significant morbidity and mortality. The COVID-19 (Coronavirus Disease 2019) pandemic represents a major global health crisis that may affect the immune system, potentially triggering or exacerbating autoimmune diseases. This study aims to evaluate the potential impact of COVID-19 on AIBDs by comparing the clinical characteristics, incidence, and treatment responses of patients diagnosed before and after the pandemic.

MATERIAL AND METHODS: Patients diagnosed with AIBDs at a university hospital between March 2016 and March 2024 were retrospectively analyzed. Demographic data, disease subtypes, clinical severity, treatment response, and hospitalization requirements were evaluated.

RESULTS: A total of 134 patients were included in the study, of whom 62 (46.3%) were diagnosed pre-pandemic and 72 (53.7%) post-pandemic. A significant increase in the incidence of bullous pemphigoid was observed in the post-pandemic period (59.7%, p=0.03). The incidence of AIBDs increased with age (p=0.02). Although no significant difference was found in disease severity, treatment-resistant cases were more frequently observed in the post-pandemic period.

CONCLUSIONS: Our study suggests that the COVID-19 pandemic may have contributed to an increased incidence of AIBDs, particularly in elderly individuals, and to the development of treatment resistance.

KEYWORDS: Autoimmune bullous diseases, Pemphigus vulgaris, Bullous pemphigoid, COVID-19 pandemic, Autoimmune diseases.

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INTRODUCTION

Autoimmune bullous diseases (AIBDs) are a group of dermatological disorders characterized by an autoimmune response against structural proteins of the skin and mucosa, leading to significant morbidity and mortality. These rare diseases result from dysregulated immune responses. Among AIBDs, pemphigus vulgaris and bullous pemphigoid (BP) are the most frequently encountered subtypes (1). In terms of etiopathogenesis, autoantibodies targeting desmosomal proteins, primarily Desmoglein 1 and Desmoglein 3, play a crucial role in the pemphigus group. In contrast, in the pemphigoid group, autoantibodies against BP180, BP230, and other components of the dermoepidermal junction are key pathogenic factors (2,3). Additionally, dermatitis herpetiformis is an AIBD associated with celiac disease and gluten intolerance, where IgA antibodies targeting tissue transglutaminase and epidermal transglutaminase are responsible for disease pathogenesis (4).

In March 2020, the World Health Organization (WHO) declared COVID-19 a global pandemic, affecting millions of individuals worldwide (5). During and after the pandemic, conditions that could arise as secondary consequences of infection have become an important area of research. It is hypothesized that viruses and other infectious agents may trigger autoimmune diseases or exacerbate pre-existing conditions, particularly in genetically predisposed individuals (6). The literature contains numerous reports of AIBD cases associated with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and COVID-19 vaccination (7,8). Although various pathophysiological mechanisms have been proposed to explain the role of SARS-CoV-2 in AIBD development, these processes remain incompletely understood. Furthermore, the long-term effects of the COVID-19 pandemic on autoimmune diseases have not yet been fully elucidated. Notably, there is growing concern that AIBDs may have increased incidence, a more severe clinical course, and higher treatment resistance in the post-pandemic period. This study aims to compare patients diagnosed with AIBDs before and after the pandemic to assess the impact of the COVID-19 pandemic on the incidence, clinical course, and treatment response of these diseases. The findings of this study are expected to contribute to a better understanding of the potential changes in AIBD pathogenesis and clinical management, thereby enriching the existing literature.

MATERIALS AND METHODS

Study Setting

The study was conducted at a university hospital that provides healthcare services to approximately 14 provinces, primarily in Northeastern Anatolia, Eastern Black Sea, and Western Black Sea regions, as well as partially to three neighboring countries (Georgia, Iran, Azerbaijan).

Patient Selection

This retrospective and observational study included patients diagnosed histopathologically with AIBDs between March 2016 and March 2024. During this period, biopsies were taken from 372 patients with suspected AIBDs. After excluding duplicate biopsies (10.8%), 332 patients remained. Among these, diagnostic evaluations confirmed AIBD in 46 of 69 patients with a preliminary diagnosis of pemphigus, 67 of 91 patients with a preliminary diagnosis of pemphigoid, and 21 of 172 patients with a preliminary diagnosis of dermatitis herpetiformis (DH). In the presence of appropriate clinical findings: Pemphigus was confirmed by epidermal intercellular DIF positivity or the detection of DSG3 or DSG1 autoantibodies in serum. Pemphigoid was confirmed by linear positivity along the basement membrane on DIF or the detection of BP180 and BP230 autoantibodies in serum. Dermatitis Herpetiformis (DH) was confirmed by granular IgA positivity on DIF or the detection of anti-tissue transglutaminase autoantibodies. Circulating autoantibodies in serum were detected using enzyme-linked immunosorbent assay (ELISA). As a result, the study population consisted of a total of 134 patients.

Data Collection

A database was first created by the dermatology faculty member conducting the study. Then, electronic patient records were reviewed, and data on age, sex, AIBD subtype, and treatment protocols were recorded. The severity

of the disease was assessed based on lesion extent, hospitalization requirements, and the treatment protocols applied, and patients were classified as having mild, moderate, or severe disease. Treatment resistance was defined as the failure to achieve disease control despite three months of systemic corticosteroid and immunosuppressive therapy. Patients whose diagnoses were not confirmed by histopathological or serological findings were excluded from the study. Patients who presented before March 11, 2020, the official date of the first confirmed COVID-19 case in Turkey, were classified as the pre-pandemic group, while those who presented on or after this date were classified as the post-pandemic group.

Ethical Committee

The study was approved by the Atatürk University Non-Interventional Clinical Research Ethics Committee (Approval No:07.06.2024/ B.30.2.ATA.0.01.00/381) and the hospital management.

Statistical Analysis

Statistical analysis was performed using SPSS Statistics 20. Descriptive statistical methods such as frequency and percentage were used, while Pearson's Chi-square test was applied for comparing categorical variables. A p-value <0.05 was considered statistically significant.

RESULTS

Among the 134 patients included in the study, 62 (46.3%) were diagnosed before COVID-19, while 72 (53.7%) were diagnosed after the onset of the pandemic. The study population comprised 57 males (42.5%) and 77 females (57.5%). Before the COVID-19 pandemic, the frequencies of pemphigus and pemphigoid diagnoses were equal (38.7%) among AIBD patients. However, after the pandemic, pemphigoid became the most frequently diagnosed AIBD (59.7%). In contrast, 66.7% of DH patients were diagnosed before the pandemic. A statistically significant difference was observed in the overall frequency of AIBDs before and after the COVID-19 pandemic (p=0.03). There was no significant difference between pre-pandemic and post-pandemic periods regarding gender distribution (p=0.36) or disease severity (p=0.90). However, the frequency of AIBDs increased with age in the post-pandemic period (p=0.02) **(Table 1)**.

Table 1: General Impact of the COVID-19 Pandemic on Autoimmune Bullous Diseases

		Pre-COVID-19 Pandemic	Post-COVID-19 Pandemic	Total	X2	p-value
		n (%)	n (%)	n(%)		
AIBDs Type	Pemphigus	24(38.7)	22(30.6)	46(34.3)	**7.102**	**0.03**
	Pemphigoid	24(38.7)	43(59.7)	67(50.0)		
	Dermatitis Herpetiformis	14(22.6)	7(9.7)	21(15.7)		
Gender	Female	33(53.2)	44(61.1)	77(57.5)	0,847	0.35
	Male	29(46.8)	28(38.9)	57(42.5)		
Severity	Mild	22(35.4)	24(33.3)	46(34.3)	0.192	0.90
	Moderate	19(30.7)	21(29.2)	40(29.9)		
	Severe	21(33.9)	27(37.5)	48(35.8)		
Age	0-29	10(16.0)	6(8.3)	16(11.9)	**6.166**	**0.02**
	30-59	28(45.2)	23(31.9)	51(38.1)		
	60 and above	24(38.7)	43(59.7)	67(50.0)		

When analyzing AIBD subtypes, no significant difference was found between pre-pandemic and post-pandemic periods in pemphigus and DH regarding gender (p=0.35, p=0.54), disease severity (p=0.70, p=0.26), or age distribution (p=0.85, p=0.09). However, in pemphigoid patients, the incidence increased with age (p=0.04), while no significant difference was found in gender (p=0.50) or disease severity (p=0.73) **(Table 2)**.

Table 2: Effects of the COVID-19 Pandemic on Autoimmune Bullous Diseases

AIBDs			Pre-COVID-19 Pandemic	Post-COVID-19 Pandemic	Total	X2	p-value
			n (%)	n(%)	n(%)		
Pemphigus	Gender	Female	12(50.0)	14(36.4)	26(56.5)	0.869	0.35
		Male	12(50.0)	8(63.6)	20(43.5)		
	Severity	Mild	9(37.5)	8(36.4)	17(37.0)	0,325	0.70
		Moderate	8(33.3)	6(27.3)	14(30.4)		
		Severe	7(29.2)	8(36.4)	15(32.6)		
	Age	0-29	4(16.2)	3(13.6)	7(15.2)	0.724	0.85
		30-59	15(62.5)	12(54.5)	27(58.7)		
		60 and above	5(20.8)	7(31.8)	12(26.1)		
Pemphigoid	Gender	Female	13(54.2)	27(62.8)	40(59.7)	0,469	0.49
		Male	11(45.8)	16(37.2)	27(40.3)		
	Severity	Mild	6(25.0)	14(32.6)	20(29.9)	0.117	0.73
		Moderate	8(33.3)	11(25.6)	19(28.4)		
		Severe	10(41.7)	18(41.9)	28(41.8)		
	Age	0-29	5(20.8)	3(7.0)	8(11.9)	3.983	0.04
		30-59	4(16.7)	4(9.3)	8(11.9)		
		60 and above	15(62.5)	36(83.7)	51(76.1)		
Dermatitis Herpetiformis	Gender	Female	8(57.1)	3(42.9)	11(52.4)	0.382	0.53
		Male	6(42.9)	4(57.1)	10(47.6)		
	Severity	Mild	7(50.0)	2(28.6)	9(42.9)	2.686	0.26
		Moderate	3(21.4)	4(57.1)	7(33.3)		
		Severe	4(28.6)	1(14.3)	5(23.8)		
	Age	0-29	1(7.1)	0(0.0)	1(4.8)	3.281	0,09
		30-59	9(64.3)	7(100.0)	16(76.2)		
		60 and above	4(28.6)	0(0.0)	4(19.0)		

Although there was no significant difference in disease severity, a higher rate of treatment resistance was recorded among patients presenting with severe disease in the post-pandemic period. Before the pandemic, these patients were successfully managed using first- and second-line therapies in accordance with national treatment guidelines. However, in the post-pandemic period, treatment failure rates increased, and three patients required third-line treatment agents (9). These patients ultimately achieved disease control with ri-

tuximab therapy. Among the AIBD subgroups, five patients were diagnosed with linear IgA dermatosis, all of whom were diagnosed before the COVID-19 pandemic. Additionally, four patients were diagnosed with pemphigus foliaceus (two pre-pandemic, two post-pandemic), two patients with pemphigus herpetiformis (one pre-pandemic, one post-pandemic), and one patient was diagnosed with gestational pemphigoid in the post-pandemic period.

DISCUSSION

The COVID-19 pandemic has significantly impacted healthcare systems worldwide, leading to changes in the course of many diseases. This study evaluates the effects of the pandemic on AIBDs and demonstrates a significant increase in AIBD diagnoses in the post-pandemic period ($p=0.03$). This increase is particularly pronounced in the pemphigoid group, correlating with older age. While no significant difference in disease severity was observed in our study group, treatment resistance was more frequently encountered, especially among pemphigoid patients, during clinical follow-ups.

Several studies in the literature support the role of COVID-19 infection in triggering autoimmune diseases. It has been reported that within 3 to 15 months following acute COVID-19 infection, the incidence of newly diagnosed autoimmune diseases increased by 43%in affected individuals. When compared to a control group, this corresponds to an absolute incidence difference of 4.50 per 1000 person-years (10). A large-scale, population-based study analyzing data from over 112 million patients demonstrated a significant increase in the risk of AIBDs, particularly pemphigus vulgaris, within three months after COVID-19 infection (7). Additionally, 19.3 percent of patients with autoimmune bullous dermatoses experienced disease exacerbations following COVID-19 infection (11).

The observed increase in AIBD risk is consistent with previous reports suggesting that viral infections can trigger or exacerbate autoimmune responses (6). Several mechanisms have been proposed for the development of autoimmune diseases following infections, including molecular mimicry, epitope spreading, prolonged survival of infected B cells, and viral persistence. All of these mechanisms can contribute to immune tolerance breakdown, thereby facilitating the emergence of autoimmune diseases (12). Moreover, the impact of viral infections on the immune system is not limited to an increased incidence of autoimmune diseases but may also lead to the development of treatment-resistant cases. The observation of treatment resistance particularly in BP patients in our study supports this hypothesis.

Most of the previous studies demonstrating the association between COVID-19 infection and AIBDs have been short-term, and data on the long-term effects of infection in this rare disease group remain limited. Our study makes a significant contribution to the literature by presenting data covering a four-year post-pandemic period specifically for AIBDs. A long-term study utilizing the Japanese database found that individuals with a history of COVID-19 had a 2.32-fold higher risk of developing any autoimmune disease compared to those without a history of COVID-19. This finding suggests that COVID-19 may have a long-term impact on the development of autoimmune diseases (13). Similarly, a recently conducted population-based study, which included long-term follow-up observations, reported an increased incidence of various autoimmune diseases. Specifically, for the AIBD group, in alignment with our study, a notable rise in the frequency of BP was identified (14).

When evaluating the long-term effects of the pandemic, the potential impact of vaccines on AIBDs should also be considered. With the rapid development of vaccination policies during the pandemic, mRNA- and viral vector-based vaccines were introduced and widely administered worldwide. These vaccines include Moderna, Pfizer-BioNTech, AstraZeneca, Janssen, Sinovac, and Covaxin. Through case reports, registry-based studies, and adverse event reporting systems, a wide range of cutaneous reactions associated with COVID-19 vaccines have been documented (15). Among this broad spectrum of cutaneous reactions, AIBDs, a rare disease group, have also been reported. A 2024 review analyzed 62 studies that collectively reported a total of 142

AIBD cases following COVID-19 vaccination. Of these, 59 were pemphigus cases, while 83 were BP cases. In both groups, most cases occurred after vaccination with BNT162b2 (Pfizer-BioNTech), predominantly following the administration of the second dose (8).

Recent studies have challenged previous case reports and meta-analyses suggesting that vaccines act as triggers, instead proposing that vaccination does not induce autoimmune diseases and may, in fact, have a protective effect (7,14,16). In light of these findings, it has been suggested that vaccination contributes to maintaining immune homeostasis by priming the immune system in a controlled manner, thereby reducing the risk of abnormal immune responses that could lead to autoimmunity. This stands in stark contrast to the more robust but potentially dysregulated immune response triggered by natural infection (17-19).

The COVID-19 vaccination process in Turkey commenced on January 14, 2021, following the emergency use authorization of the CoronaVac vaccine, with healthcare workers and individuals aged 65 and above being prioritized. In April 2021, the BNT162b2 mRNA vaccine was also incorporated into the vaccination program. As of July 30, 2021, 45% of the adult population (more than 26 million individuals) had received two doses of the vaccine (20). Although the impact of vaccination on the development of BP remains unclear, our study assumes that the vaccination rates in the patient population are comparable to those in the general population. In our study, a significant increase in pemphigoid diagnoses was observed in elderly individuals during the post-pandemic period ($p=0.04$). Age-related alterations in the immune system are referred to as immunosenescence and are characterized by the functional decline of immune organs, changes in T cells, and weakened immune responses (21,22). This process leads to an increased production of autoantibodies and a reshaping of the immune system, thereby heightening susceptibility to autoimmune diseases. This risk becomes more pronounced in individuals with genetic predisposition (22,23). Notably, individuals over the age of 65 constitute the highest-risk group for developing autoimmune diseases following COVID-19, as immune system changes may further exacerbate autoimmune tendencies (24). Our study supports these findings. The most significant limitation of our study is the inability to definitively determine whether the observed increase in BP incidence is solely attributable to the COVID-19 pandemic. The overall rise in BP prevalence may be associated with various factors, including an aging population, pemphigoid cases linked to neurological disorders, an increase in drug-induced bullous pemphigoid incidence, and advancements in diagnostic techniques. Although a comparison was made between the pre-pandemic and post-pandemic periods, it is not possible to completely exclude the influence of these factors.

The retrospective design of our study may have led to data deficiencies or recording errors. Since disease severity was assessed based on clinical observations rather than a standardized scoring system, subjective variations may have occurred. Additionally, the altered healthcare-seeking behavior during the pandemic might have restricted access to medical services, particularly for mild cases, potentially resulting in a higher proportion of severe BP cases being recorded.

The impact of COVID-19 vaccinations on the development of BP could not be assessed, as vaccination data were not available in patient records. Since the vaccination status of individuals diagnosed with BPremains unknown, potential vaccine-related contributions could not be excluded when evaluating the effects of the pandemic on these diseases. Lastly, this study was conducted at a single center, and the sample size was limited.

Our study suggests that the COVID-19 pandemic may be associated with an increased incidence of autoimmune bullous diseases, particularly BP. However, establishing a definitive causal relationship is not possible. Prospective large-scale studies are needed to assess the long-term effects of the pandemic on autoimmune diseases. The findings underscore the necessity of adopting a more cautious and attentive approach in the management of autoimmune diseases in the post-pandemic period.

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REFERENCES

1. Rosi-Schumacher M, Baker J, Waris J, et al. Worldwide epidemiologic factors in pemphigus vulgaris and bullous pemphigoid. Front Immunol. 2023;14:1159351.

2. Schmidt E, Kasperkiewicz M, Joly P. Pemphigus. Lancet. 2019;394(10201):882-94.

3. D'Agostino GM, Rizzetto G, Marani A, et al.Bullous Pemphygoid and Novel Therapeutic Approaches. Biomedicines. 2022;10(11):2844.

4. Gjersvik PJ, Rønnevig JR. Dermatitis herpetiformis [Dermatitis herpetiformis]. Tidsskr Nor Laegeforen. 2003;123(22):3234-6.

5. Onyeaka H, Anumudu CK, Al-Sharify ZT, et al. COVID-19 pandemic: A review of the global lockdown and its far-reaching effects. Sci Prog. 2021;104(2):368504211019854.

6. Hussein HM, Rahal EA. The role of viral infections in the development of autoimmune diseases. Crit Rev Microbiol. 2019;45(4):394-412.

7. Curman P, Kridin K, Zirpel H, et al. COVID-19 infection is associated with an elevated risk for autoimmune blistering diseases while COVID-19 vaccination decreases the risk: A large-scale population-based cohort study of 112 million individuals. J Am Acad Dermatol. 2025;92(3):452-63.

8. Martora F, Battista T, Potestio L, et al. Pemphigus and Bullous Pemphigoid Following COVID-19 Vaccination: A Systematic Review. Viruses. 2024;16(12):1896.

9. Uzun S, Durdu M, Kaya Tİ, Aktan Ş, editors. Otoimmün Büllöz Hastalıklar. 1st ed. İstanbul: Türk Dermatoloji Derneği Yayınları. 2017.

10. Tesch F, Ehm F, Vivirito A, et al. Incident autoimmune diseases in association with SARS-CoV-2 infection: a matched cohort study. Clin Rheumatol. 2023;42(10):2905-14.

11. Moghadam FS, Kianfar N, Dasdar S, et al. Adverse outcome and severity of COVID-19 in patients with autoimmune bullous diseases: A historical cohort study. Dermatol Ther. 2022;35(9):e15672.

12. Smatti MK, Cyprian FS, Nasrallah GK, et al. Viruses and Autoimmunity: A Review on the Potential Interaction and Molecular Mechanisms. Viruses. 2019;11(8):762.

13. Inokuchi S, Shimamoto K. Persistent Risk of Developing Autoimmune Diseases Associated With COVID-19: An Observational Study Using an Electronic Medical Record Database in Japan. J Clin Rheumatol. 2024;30(2):65-72.

14. Heo YW, Jeon JJ, Ha MC, et al. Long-Term Risk of Autoimmune and Autoinflammatory Connective Tissue Disorders Following COVID-19. JAMA Dermatol. 2024;160(12):1278-87.

15. Mahmood F, Cyr J, Li A, et al. Vesiculobullous and Other Cutaneous Manifestations of COVID-19 Vaccines: A Scoping and Narrative Review. J Cutan Med Surg. 2023;27(3):260-70.

16. Birabaharan M, Kaelber DC, Orme CM, et al. Evaluating risk of bullous pemphigoid after mRNA COVID-19 vaccination. Br J Dermatol. 2022;187(2):271-3.

17. De Martino M, Chiappini E, Galli L. Vaccines and autoimmunity. Int J Immunopathol Pharmacol. 2013;26(2):283-90.

18. Olivieri B, Betterle C, Zanoni G. Vaccinations and autoimmune diseases. Vaccines (Basel). 2021;9(8):815.

19. Krammer F. The human antibody response to influenza A virüs infection and vaccination. Nat Rev Immunol. 2019;19(6):383-97.

20. Sonmezer MC, Sahin TK, Erul E, et al. Knowledge, Attitudes, and Perception towards COVID-19 Vaccination among the Adult Population: A Cross-Sectional Study in Turkey. Vaccines (Basel). 2022;10(2):278.

21. Wang Y, Dong C, Han Y, et al. Immunosenescence, aging and successful aging. Front Immunol. 2022;13:942796.

22. Rosato E, Salsano F. Immunity, autoimmunity and autoimmune diseases in older people. J Biol Regul Homeost Agents. 2008;22(4):217-24.

23. Mishra N, Kammer GM. Clinical expression of autoimmune diseases in older adults. Clin Geriatr Med. 1998;14(3):515-42.

24. Müller L, Di Benedetto S. From aging to long COVID: exploring the convergence of immunosenescence, inflammaging, and autoimmunity. Front Immunol. 2023;14:1298004.