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Original Article

Markers Of Autoimmunity In Sars-Cov-2 Infection: Antinuclear Antibodies, Antiphospholipid Antibodies, Anti-Celiac Antibodies, And Anti-Hepak7 Antibodies.

Oussama Aazzane[1,2*], Hasnaa Bazhar [2], Nabil Gaougaou [3], Hassan Fellah [1,2]

1. Laboratory of Cellular and Molecular Pathology, Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, Morocco.

2. Immunology Laboratory, Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, Morocco.

3. Laboratory of Immunology and Histology, Faculty of Medicine and Pharmacy, Ibn Zohr University, Laayoune, Morocco.

Abstract

It is well known that viral infections, especially chronic ones, are associated with the development of autoimmune diseases (AID) in certain patients. In the case of COVID-19, some patients have developed disorders similar to AID. This raises the question: is COVID-19 responsible for these immune system dysfunctions, or does it merely exacerbate an underlying autoimmune pathology? It is therefore just as important to prevent the onset of these AID in certain COVID-19 positive patients as it is to fight the infection itself. To address this question, the present study aims to assess the prevalence of specific autoantibodies in COVID-19 patients, with a particular focus on IgG anti-nuclear, anti-phospholipid, anti-tissue transglutaminase, anti-deamidated gliadin, and anti-HepAk7 autoantibodies. More specifically, it seeks to identify potential immunological markers specific to SARS-CoV-2-induced autoimmunity, particularly in advanced stages of the disease (moderate to severe). This is a retrospective study conducted at the Immunology Laboratory of the Faculty of Medicine and Pharmacy in Casablanca. The analysis included 45 unvaccinated patients with confirmed SARS-CoV-2 infection and 24 healthy controls. Detection of anti-nuclear, anti-phospholipid, anti-CeliAk, and anti-HepAk7 IgG autoantibodies was performed using immunodot tests. Statistical analyses were carried out using GraphPad Prism 8 software. IgG anti-nuclear antibodies were detected in 6.66% of patients and 8.33% of controls, with no significant difference between groups ($p = 0.799$). In contrast, IgG anti-phospholipid antibodies showed significantly higher prevalence in COVID-19 patients (80%) compared to controls (0%) ($p < 0.0001$). IgG anti-tissue transglutaminase (anti-tTG) antibodies were found in 13.3% of patients vs. 4.2% of controls, without significant difference ($p = 0.229$). IgG anti-deamidated gliadin (anti-GD) antibodies were detected in 4.4% of patients and none in controls ($p = 0.294$). No subjects, patients or controls, were positive for IgG anti-HepAk7 antibodies ($p = 1$). These results highlight a significant association between SARS-CoV-2 infection and high prevalence of anti-phospholipid antibodies, supporting the hypothesis of virus-induced immune dysregulation, while the other analyzed autoantibodies (anti-nuclear, anti-tTG, anti-GD, anti-HepAk7) showed no significant differences between patients and controls. Larger prospective studies, including a broader range of autoantibodies and longitudinal follow-up, are essential to confirm these findings, assess the persistence of these abnormalities, and better understand the underlying mechanisms of COVID-19-associated autoimmunity.

Keywords: Autoantibodies, Autoimmune Diseases, COVID-19.

INTRODUCTION

Since its emergence in December 2019 in Wuhan, China, SARS-CoV-2 (a coronavirus) responsible for COVID-19 has caused an unprecedented global health crisis. Initially considered an acute respiratory disease, it can evolve into a systemic disease capable of triggering an exacerbated inflammatory response and affecting multiple organs, including the skin, kidneys, respiratory system, cardiovascular system, digestive system, nervous system, and hematological system. This immune hyperactivation, particularly observed in severe cases of the disease, can not only worsen the viral infection but also promote the onset of autoimmune phenomena [1-2, 3-4].

In the effort to eliminate the virus, the immune system may paradoxically stimulate both immune and non-immune cells, leading to deleterious hyperstimulation. Although neutralizing antibodies may have a protective effect against SARS-CoV-2, humoral immunity is often disrupted in COVID-19. In susceptible individuals, antibodies targeting SARS-CoV-2 viral proteins could trigger autoimmune reactions through cross-reactions with autoantigenic targets. Reports of inflammatory/autoimmune symptoms and the detection

***Corresponding Author: Oussama Aazzane.** Immunology Laboratory, Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, Tarik Ibnou Ziad Street Casablanca, 20250, Morocco. **Orchid:** 0000-0002-9032-8848, **Email:** oussama.aazzane-etu@etu.univh2c.ma.

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of circulating autoantibodies in a subset of infected patients suggest that SARS-CoV-2 can induce autoimmune disorders in genetically predisposed individuals [5- 6, 7- 8, 9].

Indeed, numerous studies have highlighted the presence of autoantibodies in COVID-19 patients, particularly in severe cases. Among these are anti-nuclear antibodies (ANA), which are associated with generalized inflammation and tissue damage in autoimmune diseases. Additionally, an increased frequency of anti-phospholipid (APL) antibodies has been observed in patients with severe COVID-19, suggesting a link between these autoantibodies and a hyperinflammatory state [5, 10-11]. Furthermore, many newly diagnosed autoimmune diseases have been reported following SARS-CoV-2 infection, affecting various systems such as the nervous system (Guillain-Barré syndrome, transverse myelitis), musculoskeletal system (rheumatoid arthritis, spondyloarthritis), and endocrine system (Graves' disease, Hashimoto's thyroiditis) [1-12].

These observations raise a fundamental question: Is COVID-19 a triggering factor for autoimmune diseases, or does it merely exacerbate an underlying autoimmune pathology? Moreover, in certain COVID-19 positive patients, preventing this immune dysregulation is just as important as fighting the virus itself. To address this question, the present study aims to assess the prevalence of specific autoantibodies in COVID-19 patients, with a particular focus on IgG anti-nuclear, anti-phospholipid, anti-tissue transglutaminase, anti-deamidated gliadin, and anti-HepAk7 autoantibodies. More specifically, it seeks to identify potential immunological markers specific to SARS-CoV-2-induced autoimmunity, particularly in advanced stages of the disease (moderate to severe).

PATIENTS AND METHODS

Patients

This is a retrospective, monocentric study including 69 serum samples from 45 patients with COVID-19 and 24 healthy subjects (controls). These serum samples are part of the serotheque of the Immunology Laboratory at the Faculty of Medicine and Pharmacy of Casablanca (**Figure 1**).

Inclusion criteria:

The study included patients meeting the following criteria: individuals infected with SARS-CoV-2, unvaccinated, and whose positive diagnosis was confirmed by both a serological test and real-time RT-PCR. Regarding the control group, it consisted of healthy subjects who were not infected with SARS-CoV-2, and whose samples were collected before the COVID-19 pandemic.

Exclusion criteria:

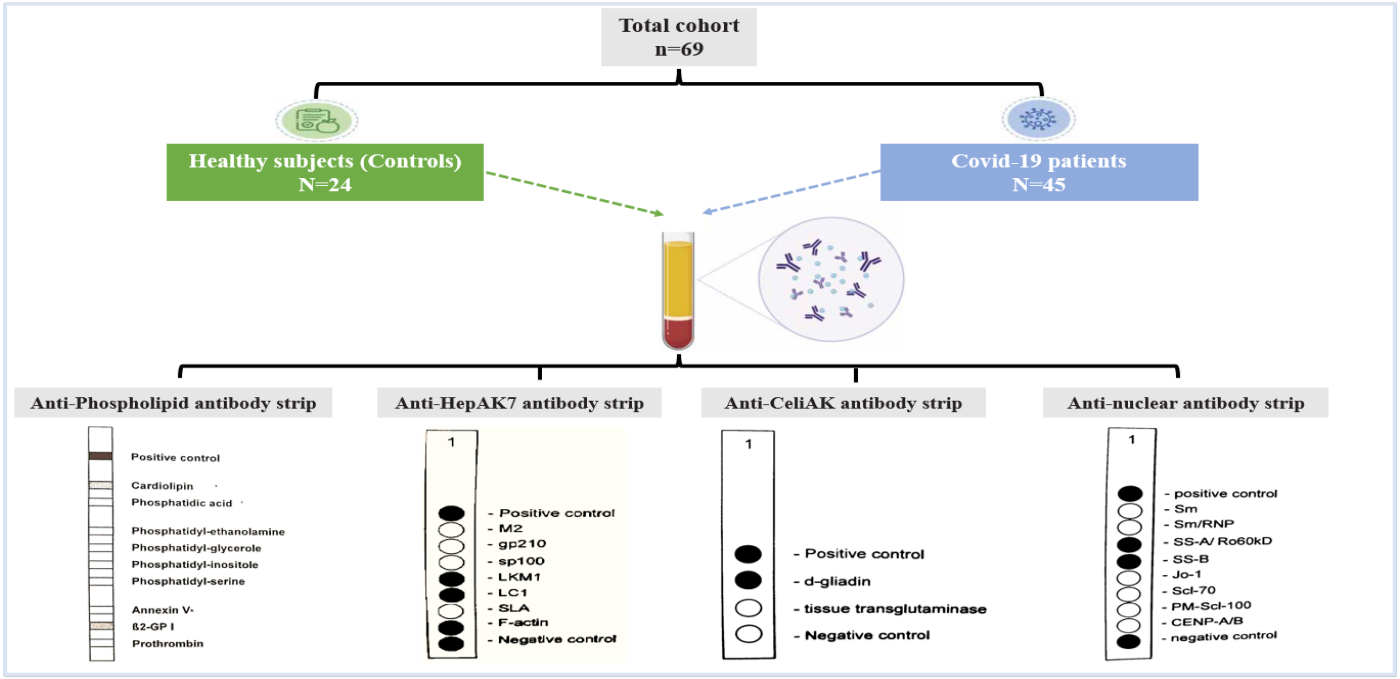
Patients and controls under the age of 18 were excluded from the study.

Methods

The evaluation of IgG-type autoantibodies, including anti-Nuclear (REF: 5016), anti-Phospholipid (REF: 5040), anti-CeliAk (REF: 5015), and anti-HepAk7 (REF: 5021), was performed on serum samples from COVID-19 patients and healthy subjects (controls). The analysis was conducted using the immunodot technique with the DotDiver kit (Generic Assays, Germany).

The interpretation of results was based on comparing the intensity of the coloration obtained from the tested sample with that of positive and negative controls. A sample was considered positive for a given autoantibody if its coloration intensity exceeded that of the negative control (**Figure 1**).

Figure 1. Study Flow Chart.



Statistical Analysis

All statistical analyses were performed using GraphPad Prism 8 software. The Chi-square test was used to compare the presence of autoantibodies between patients and controls. A p-value < 0.05 was considered indicative of statistically significant differences.

RESULTS

Demographic Characteristics of Patients and Controls

In our study, the age of patients ranged from 21 to 90 years, with an average age of 50.77 years and a sex ratio of 0.87. Regarding the control group, the average age was 38.95 years [29–72 years], with a sex ratio of 0.41 (**Table 1)**.

Table 1. Demographic characteristics of patients and controls.

Variables	Patients	Controls
Age at diagnosis (years) Mean [Rank]	50.77 [21 - 90]	38.95 [29 - 72]
Gender, n (%) Men Women Sex ratio	21 (46.67) 24 (53.33) 0.875	07 (29.16) 17 (70.83) 0.41

Seroprevalence of Anti-Nuclear IgG Antibodies

The evaluation of anti-nuclear IgG antibodies in patients and controls revealed positivity in 6.66% of patients (N=3), exclusively for anti-SSA/Ro antibodies. Among the controls, 8.33% (N=2) were also positive for anti-nuclear IgG antibodies, with an equal distribution between anti-SSA/Ro antibodies (4.16%; N=1) and anti-Sm/RNP antibodies (4.16%; N=1). However, no significant difference was observed between the two groups (p=0.799) (**Table 2**).

Table 2. Results of Anti-Nuclear IgG Antibodies in Patients and Controls.

Anti-nuclear antibodies	Patients N=45 (%)	Controls N=24 (%)	P value
Anti-Sm	00 (00.00)	00 (00.00)	0.799
Anti-Sm/RNP	00 (00.00)	01 (04.16)	
Anti-SSA/RO	**03 (06.66)**	01 (04.16)	
Anti-SSB	00 (00.00)	00 (00.00)	
Anti-Jo1	00 (00.00)	00 (00.00)	
Anti-Scl70	00 (00.00)	00 (00.00)	

Seroprevalence of Anti-Phospholipid IgG Antibodies

The evaluation of anti-phospholipid IgG antibodies (aPLs) revealed that 80% of patients (36/45) were positive. Specifically, 75.56% (34/45) were double positive for IgG anti-phosphatidic acid and IgG anti-cardiolipin, while 4.44% (2/45) were positive for IgG anti-annexin V. In contrast, all controls tested negative for anti-phospholipid IgG antibodies. Statistical analysis showed a significant difference between patients and controls (p<0.0001) (**Table 3**).

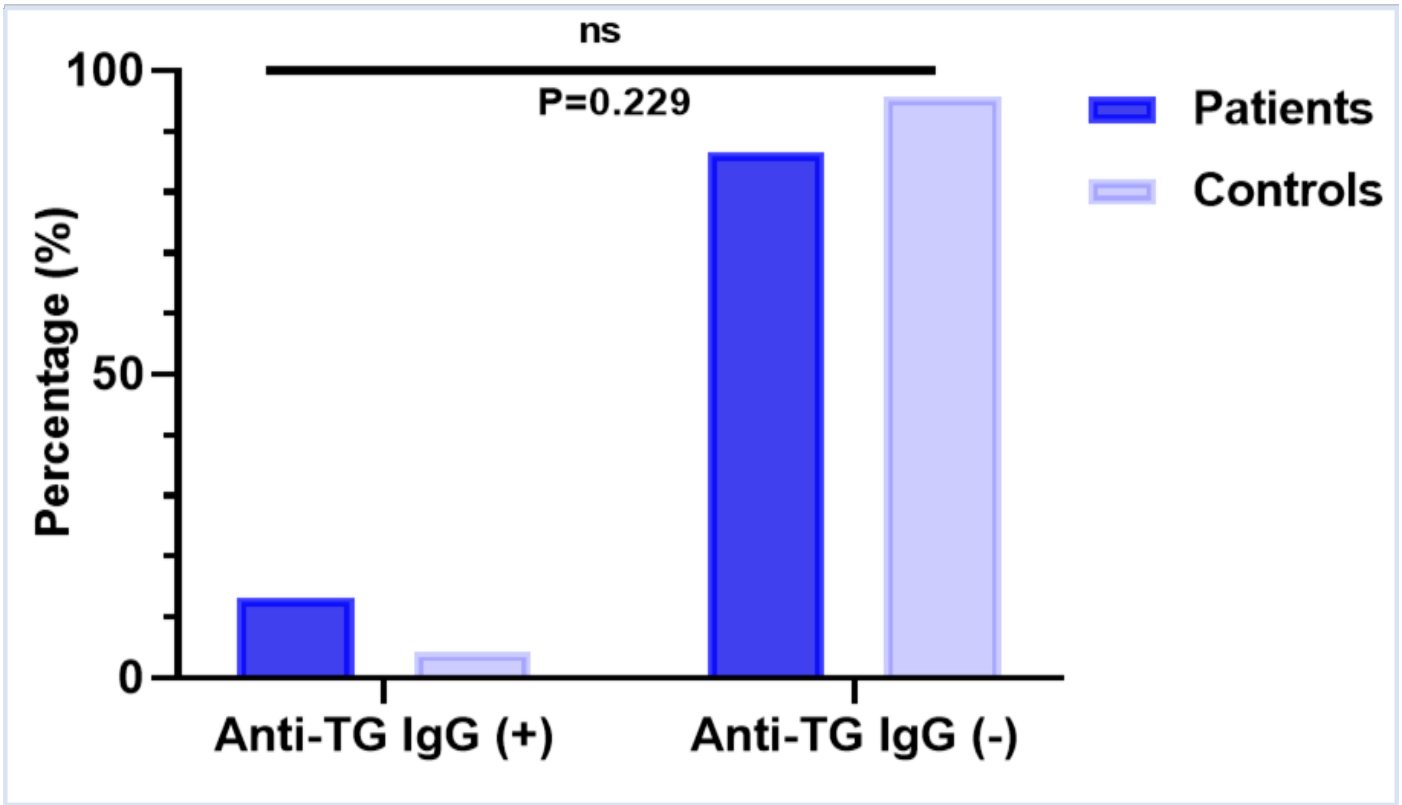
Table 3. Results of Anti-Phospholipid IgG Antibodies in Patients and Controls.

Anti- phospholipid antibodies	Patients N=45 (%)	Controls N=24 (%)	P value
Anti-Cardiolipin et Anti-Phosphatidic acid	34 (75.56)	00 (00.00)	<0.0001
Anti-Phosphatidyl-ethanolamine	00 (00.00)	00 (00.00)	
Anti- Phosphatidyl-glycerole	00 (00.00)	00 (00.00)	
Anti- Phosphatidyl-inositole	00 (00.00)	00 (00.00)	
Anti- Phosphatidyl-serine	00 (00.00)	00 (00.00)	
Anti-Annexin V	**02 (04.44)**	00 (00.00)	
Anti-B2-GP I	00 (00.00)	00 (00.00)	
Anti-Prothrombin	00 (00.00)	00 (00.00)	

Seroprevalence of Anti-Tissue Transglutaminase IgG Antibodies

The evaluation of anti-tissue transglutaminase IgG antibodies showed a higher prevalence in patients compared to controls, with respective rates of 13.33% and 4.17%. However, this difference was not statistically significant (p=0.229) (**Figure 2**).

Figure 2. Results of Anti-TG IgG Antibodies in Patients and Controls.

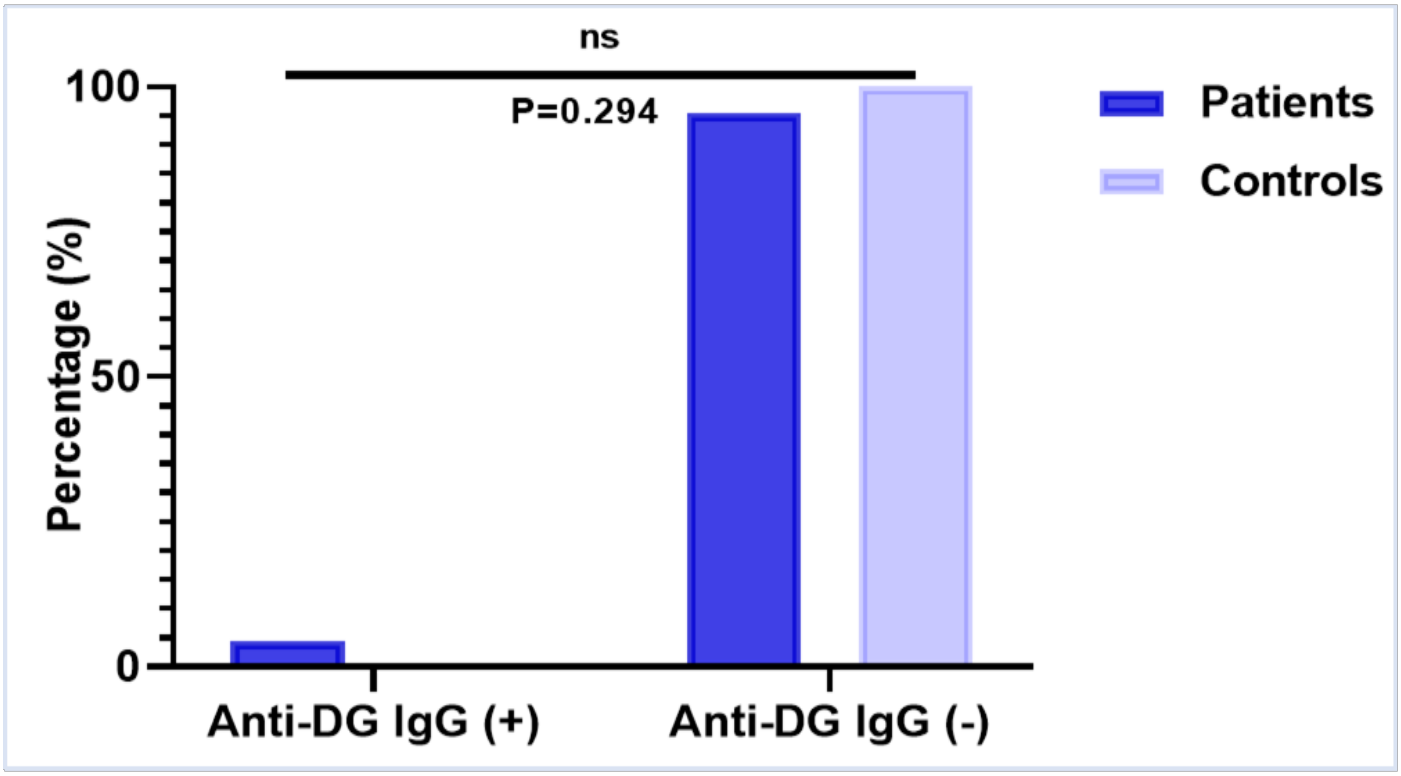


Abbreviations: TG: Tissue Transglutaminase; (+): Positive; (-): Negative.

Seroprevalence of Anti-Deamidated Gliadin IgG Antibodies

The evaluation of anti-deamidated gliadin IgG antibodies showed that 4.44% (N=2) of patients were positive, while all controls tested negative. Statistical analysis did not reveal a significant difference between the groups (p=0.294) (**Figure 3**).

Figure 3. Results of Anti-GD IgG Antibodies in Patients and Controls.



Abbreviations: GD: Deamidated Gliadin; (+): Positive; (-): Negative.

Seroprevalence of Anti-HepAk7 IgG Antibodies

The evaluation of anti-HepAk7 IgG antibodies showed that all patients and controls tested negative. Thus, no statistically significant difference was observed (p=1) (**Table 4**).

Table 4. Results of Anti-HepAk7 IgG Antibodies in Patients and Controls.

	Patients N=45 (%)	Controls N=24 (%)	P value
Anti-HepAk7 IgG antibody (+)	00 (00.00)	00 (00.00)	1
Anti-HepAk7 IgG antibody (-)	45 (100.0)	24 (100.0)	

DISCUSSION

In December 2019, an outbreak of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection occurred in Wuhan, China, and rapidly spread worldwide. The World Health Organization (WHO) officially named the disease coronavirus disease 2019 (COVID-19), now considered the greatest global public health emergency. Although most COVID-19 patients are asymptomatic, with an incidence of 1.6%, or develop only mild illness, a significant proportion of patients develop severe forms of the disease, characterized by pneumonia, coagulopathy, hyperinflammation, and multi-organ failure, affecting organs such as the heart and lungs. Previous studies have reported that antiphospholipid antibodies (aPLs) may be associated with thromboembolic complications, and some clinical features of the infection may mimic those observed in systemic autoimmune diseases [13-14]. This prompted us to investigate one or more autoantibodies that could serve as specific markers of an autoimmune reaction in COVID-19 (+) patients, particularly in advanced stages of the disease (moderate to severe).

The objective of our study is to assess the prevalence of certain autoantibodies in COVID-19 patients, with a particular focus on IgG autoantibodies against nuclear antigens, phospholipids, tissue transglutaminase, deamidated gliadin, and HepAk7. More specifically, it aims to identify potential immunological markers specific to SARS-CoV-2-induced autoimmunity, particularly in the advanced stages of the disease (moderate to severe).

In this context, we examined the presence of anti-nuclear IgG antibodies in 45 COVID-19 (+) patients. The results revealed positivity for anti-SSA/RO antibodies in 6.66% (N=3) of patients. In parallel, the analysis of 24 seronegative control sera showed a positivity rate of 8.33% (N=2), including 4.16% (N=1) for anti-SSA/RO antibodies and 4.16% (N=1) for anti-Sm-RNP antibodies (**Table 2**). These results suggest that immune system activation during SARS-CoV-2 infection is not necessarily accompanied by an autoimmune response against these autoantigens, although the sporadic presence of these antibodies may be due to transient immune activation or preexisting autoimmune susceptibility.

Our results are comparable to those reported in the literature, although some discrepancies exist. For example, a study conducted by Pascolini et al. (Italy, 2021) reported autoantibody reactivity in 45.4% of COVID-19 patients, with 33.3% presenting anti-nuclear antibodies. Another study conducted by Zhou et al. (China, 2020) reported a slightly higher positivity rate for anti-nuclear antibodies (50%) [15-16]. Moreover, a study by Fujii et al. (Japan, 2020) examined two patients, a 65-year-old man and a 78-year-old woman, with no history of autoimmune diseases, whose sera exhibited very high levels of anti-SSA-RO antibodies. The woman had a level of 203 U/ml (measured by ELISA), and the man had a level of 442 U/ml (by ELISA), whereas the normal value should be below 40 U/ml. The study suggested that the pulmonary pathophysiology of both patients resembled interstitial lung disease (ILD). There remains uncertainty about whether the elevated anti-SSA-RO antibodies are a cause or a consequence of COVID-19 pneumonia aggravation, leading to the hypothesis that there may be a link between worsening COVID-19 pneumonia and an autoimmune response in these two patients [17].

The discrepancies between these studies and ours could be explained by several factors, including the techniques used for detecting anti-nuclear antibodies. We used the immunodot technique, while the Italian study used immunofluorescence, the Chinese study used ANA immunoblot, and the Japanese study used ELISA [15-16, 17]. Additionally, the profile of the recruited subjects is an important factor. In the Chinese study, all patients were in severe (31.8%) or critical (61.9%) condition and were in the intensive care unit of Huangshi Central Hospital [15]. In our study, we recruited hospitalized COVID-19 patients with moderate to severe disease. Moreover, differences in sample size, which was smaller in our study compared to the Chinese and Italian studies, as well as genetic differences among populations (Italy, China, Morocco), could lead to different antibody phenotypes.

In this study, we detected anti-phospholipid IgG antibodies in 80% of the tested COVID-19 patients. More specifically, 75.56% (34/45) were positive for both anti-phosphatidic acid IgG and anti-cardiolipin IgG, while 4.44% (2/45) were positive for anti-annexin V IgG (**Table 3**). These two patients were also positive for anti-phosphatidic acid IgG and anti-cardiolipin IgG. None of the controls tested positive for anti-phospholipid IgG antibodies. Our results suggest a potential role of aPLs in the pathophysiology of COVID-19, with a possible involvement of autoantibodies in disease progression and severity. The complete absence of these antibodies in controls supports the hypothesis that their emergence is directly related to SARS-CoV-2 infection and its effects on the immune system.

Previous studies have highlighted the involvement of aPLs

in thromboembolic events in severe COVID-19 patients, with rates ranging from 8% to 96%, particularly for lupus anticoagulant (LA) followed by anti-cardiolipin antibodies (aCL). These two antibodies are among the three main antibodies (LA, aCL, and anti-β2 glycoprotein) tested for in the diagnosis of antiphospholipid syndrome (APS) [18].

Regarding annexin, our study revealed two (male) positive cases among the 45 COVID-19 (+) sera tested. The detected annexin was associated with cardiolipin and phosphatidic acid. A study conducted at Tor Vergata University in Italy (2021) using ELISA on 48 hospitalized COVID-19 (+) patients showed that three patients (6.25%) tested positive for anti-annexin IgG, with levels of 23.75 U/ml, 11.31 U/ml, and 20.87 U/ml. Three other patients were positive for anti-annexin IgM, with levels of 16.25 U/ml, 11.37 U/ml, and 10.6 U/ml [21].

The high frequency and diversity of aPLs strongly suggest that these antibodies are actively induced during acute SARS-CoV-2 infection. It is noteworthy that this prevalence is similar to that observed in several autoimmune diseases, although lower than in patients with primary APS.

Regarding anti-tissue transglutaminase (TG) and anti-deamidated gliadin (GD) IgG antibodies, we detected positivity in 13.33% (N=6) and 4.44% (N=2) of patients, respectively. One control was positive for anti-TG IgG, while none were positive for anti-GD IgG (**Figures 2, 3**). These autoantibodies are typically associated with celiac disease and certain pathologies. However, their presence in COVID-19 patients may reflect non-specific immune activation or an alteration in immune tolerance induced by SARS-CoV-2 infection. The fact that these antibodies are almost absent in controls suggests a possible role of SARS-CoV-2 in their emergence. However, due to the relatively low prevalence and the need for larger studies, these results should be interpreted with caution.

For anti-HepAk7 IgG antibodies, including anti-M2, agp210, sp100, LKM1, LC1, SLA, and F-actin, none of the tested samples, whether from COVID-19 patients or controls, were positive (**Table 4**). These markers are generally associated with autoimmune liver diseases such as autoimmune hepatitis and primary biliary cholangitis. Their absence in our cohort suggests that SARS-CoV-2 infection, in the cases studied, does not seem to induce a detectable autoimmune hepatic response through these biomarkers.

Although encouraging, our results require further confirmation through larger studies to determine the diagnostic and prognostic value of these autoantibodies in COVID-19 patients.

CONCLUSION

This study highlights a notable prevalence of autoantibodies, particularly antiphospholipid antibodies, in COVID-19 patients. These autoantibodies may play a role in the immuno-thrombotic complications observed in severe forms of the disease. However, further studies with larger sample sizes are needed to confirm these observations and elucidate their clinical significance.

Acknowledgements

Conflict of Interest

We declare that there are no conflicts of interest to disclose.

Authors Contribution

The authors of this article have made significant contributions to the design, data collection, analysis, and manuscript writing. Their individual contributions are as follows:

Dr. Aazzane Oussama: Writing - original version, Conceptualization, Methodology, Data collection.

Dr. Bazhar Hasnaa and Pr.Nabil Gaougaou: Formal analysis, Visualization.

Pr. Fellah Hassan: Investigation, Supervision, Conceptualization, Methodology, Validation, Revision and Editing.

Availability of Data

The data used in this research is available upon request from the authors (Dr. Oussama Aazzane, and Pr. Hassan Fellah).

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