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Anti-Saccharomyces cerevisiae antibodies in patients with COVID-19

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ABSTRACT

Background and study aim: Anti-Saccharomyces cerevisiae antibodies (ASCA) have been described in many autoimmune diseases (AIDs). Coronavirus disease 2019 (COVID-19) could trigger AIDs. This study aimed to determine the frequency of ASCA in patients with COVID-19.

Patients and methods: This study included 88 adult patients with severe COVID-19, 51 mild COVID-19, and 160 healthy blood donors. ASCA of isotype immunoglobulin (Ig)G and IgA were detected by enzyme-linked immunosorbent assay.

Results: The frequency of ASCA (IgG or IgA) was significantly higher in patients with severe COVID-19 (21.6 % vs 3.7 %, p < 10⁻³) and in patients with mild COVID-19 than in the healthy controls (13.7 % vs 3.7 %, p = 0.03). ASCA-IgA was significantly more frequent in patients with severe COVID-19 than in healthy controls (15.9 % vs 6.6 %, p < 10⁻³). ASCA-IgG was significantly more frequent in patients with mild COVID-19 than in healthy controls (13.7 % vs 3.1 %, p = 0.02). ASCA (IgG or IgA) were more frequent in severe than in mild COVID-19, but the difference was not statistically significant (21.6 % vs 13.7 %). ASCA-IgA was significantly more frequent in patients with severe COVID-19 (15.9 % vs 6.6 %, p = 0.003). The mean ASCA-IgG and ASCA-IgA levels were significantly higher in patients with severe COVID-19 than in healthy controls (5.8 U/mL ± 11.8 vs 2.3 U/mL ± 2.8, p < 10⁻³ and 9.2 U/mL ± 21.5 vs 3.4 U/mL ± 1.7, respectively, p < 10⁻³). The mean ASCA-IgG levels were significantly higher in patients with mild COVID-19 than in healthy controls (6.2 U/mL ± 12.9 vs 2.3 U/mL ± 2.8, p < 10⁻³). The mean ASCA-IgA levels were significantly higher in patients with severe than in those with mild COVID-19 (9.2 U/mL ± 21.5 vs 2.6 U/mL ± 1.2, p = 0.03).

Conclusion: ASCA was more frequent in patients with COVID-19 than in healthy controls.

Introduction

Coronavirus disease 2019 (COVID-19) is an infectious disease that is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. COVID-19 is now recognized as a multisystem disease with a broad spectrum of manifestations [2–4]. The pathophysiology of COVID-19 remained unclear; however, much evidence supports the hypothesis that SARS-CoV2 could stimulate autoimmunity in predisposed patients. Histopathological signs of autoimmune reactions have been demonstrated in many organ systems of deceased patients from COVID-19. CD8 T lymphocyte-infiltrated lungs, adrenals, liver, intestine, and other organs confirm an autoimmune process [5]. SARS-CoV-2 can break immunological tolerance by molecular mimicry, standard activation, and epitope spreading and therefore, induce autoimmune

Abbreviations: ASCA, anti-Saccharomyces cerevisiae antibodies; AID, autoimmune diseases; COVID-19, Coronavirus disease 2019; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; MBL, mannose binding lectine.

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diseases (AIDs) [6]. Moreover, the incidence of many AIDs has increased since the beginning of the COVID-19 pandemic [7–9].

Saccharomyces cerevisiae (S. cerevisiae) is a yeast that is used for bread baking and is one of the predominant yeast in our mycobionta [10]. anti-

*S. cerevisiae* antibodies (ASCA) are directed against the phosphoprotein mannan, a part of the cell wall of *S. cerevisiae*. ASCA is considered one of the serological markers of Crohn’s disease [11–12]. Combined with perinuclear anti-neutrophil cytoplasmic antibodies, ASCA has been reported as a valuable marker to discriminate between Crohn’s disease and ulcerative colitis [13]. Additionally, ASCA has been detected in many AIDs [14–19]. However, to our knowledge, only one study has determined ASCA in COVID-19 [20]. Therefore, the present study aimed to determine the frequency of ASCA in this viral disease, which could induce AIDs.

Patients and methods

Patients

We enrolled two groups of patients with a confirmed COVID-19 diagnosis by real-time polymerase chain reaction test (RT-PCR) of an oropharyngeal swab sample from December 2020 to February 2021. The first group consisted of 88 adult patients with severe COVID-19 and the second group was composed of 51 adults with mild COVID-19. Group classification was based on clinical symptoms, respiratory rate, and oxygen saturation. COVID-19 severity was categorized under the World Health Organization Clinical Progression Scale [21]. All sera samples in the second group were not hospitalized and were given health care at home, and their sera samples were collected at least 14 days after RT-PCR. Medical criteria for hospital admission were severe dyspnea, oxygen saturation on room air of ≤ 94 % or increase respiratory rate (>30 breaths per min), confusion or altered mental status or comorbid condition with worsening shortness of breath, fever, and/or increasing cough. Patients with Crohn’s disease or autoimmune disease were excluded from our study.

Sera of 160 blood donors were included as normal controls. All sera were stored at –80 °C until use. The study was approved by the local ethics committee and all patients gave their informed consent.

Laboratory measurements

ASCA immunoglobulin (Ig)G and IgA were detected by a commercial enzyme-linked immunosorbent assay (ELISA) kit (Orgentec Mainz® Germany). The antigen consisted of highly purified mannan from *S. cerevisiae*. Results were expressed as arbitrary units with a cut-off for positivity of 10 U/mL following the manufacturer’s instructions.

Statistical analysis

The comparison of frequencies of ASCA was performed using Chi-square or Fisher’s test. We used a parametric Student’s *t*-test to compare the mean titer of ASCA. A Pearson correlation test was used to show the relationship between variables. A *p*-value of < 0.05 was considered significant.

Results

Table 1 shows the epidemiologic characteristics of patients and healthy controls.

Frequency of ASCA in patients with COVID-19 and healthy controls

The frequency of ASCA (IgG or IgA) was significantly higher in patients with severe COVID-19 (21.6 % vs 3.7 %, *p* < 10^-3) and those with mild COVID-19 than in healthy controls (13.7 % vs 3.7 %, *p* = 0.03).

| Table 1 | Epidemiologic features of patients with COVID-19 and healthy subjects. |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                | Severe-COVID-19 | Mild-COVID-19 | Control group   | Severe-COVID-19 |
| (n = 88)        | (n = 51)        | (n = 160)      |                 | (n = 160)       |
| Sex-ratio (M/F) | 1.1 (46/42)     | 0.2 (9/42)     | 4 (128/32)      | <10^-3          |
| Median age (IQR) (Years) | 62 (55–70) | 34 (22–44) | 20 | NS |
| Age range (years) | 21–82 | 19–58 | 17–45 | – |

M: male; F: female; IQR: interquartile range.

ASCA-IgA were significantly more frequent in patients with severe COVID-19 than in healthy controls (15.9 % vs 0.6 %, *p* < 10^-3). ASCA-IgG were significantly more frequent in patients with mild COVID-19 than in healthy controls (13.7 % vs 3.1 %, *p* = 0.01) (Table 2).

Comparison of the frequency of ASCA between patients with severe and mild COVID-19

ASCA (IgG or IgA) were more frequent in severe than in mild COVID-19, but the difference was not statistically significant (21.6 % vs 13.7 %). ASCA-IgA was significantly more frequent in patients with severe than in mild COVID-19 (15.9 % vs 0 %, *p* = 0.003) (Table 2).

Levels of ASCA

The mean ASCA-IgG levels were significantly higher in both patients with severe and mild COVID-19 than in healthy controls (5.8 U/ml ± 11.8 vs 2.3 U/ml ± 2.8, *p* < 10^-3; 6.2 U/ml ± 12.9 vs 2.3 U/ml ± 2.8, *p* < 10^-3 respectively). The difference in the mean ASCA-IgG levels between patients with severe and mild COVID-19 was not significant (5.8 U/ml ± 11.8 vs 6.2 U/ml ± 12.9; *p* = 0.85) (Fig. 1).

The mean ASCA-IgA levels were significantly higher in patients with severe COVID-19 than in healthy controls (9.2 U/ml ± 21.5 vs 3.4 U/ml ± 1.7, *p* < 10^-3). The mean ASCA-IgA levels were significantly higher in healthy controls than in patients with mild COVID-19 (3.4 U/ml ± 1.7 vs 2.6 U/ml ± 1.2, *p* = 0.002). The mean ASCA-IgA levels were significantly higher in patients with severe COVID-19 than those with mild COVID-19 (9.2 U/ml ± 21.5 vs 2.6 U/ml ± 1.2; *p* = 0.03) (Fig. 2).

| Table 2 | Frequency of ASCA in patients with COVID-19 and healthy controls. |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                | Severe-COVID-19 | Mild-COVID-19 | Control group   | Severe-COVID-19 |
| (n = 88)        | (n = 51)        | (n = 160)      |                 | (n = 160)       |
| ASCA IgG        | 21.6 (19)       | 13.7 (7)       | 3.7 (6)         | <10^-3          |
| ASCA IgA        | 3.4 (3)         | 0 (0)          | 0 (0)           | NS              |
| ASCA IgG and IgA | 9 (8)     | 13.7 (7)       | 3.1 (5)         | NS              |
| ASCA IgG        | 15.9 (14)       | 0 (0)          | 0.6 (1)         | <10^-3          |

ASCA: anti-Saccharomyces cerevisiae antibodies; NS: not significant.
Association of ASCA with age, sex, and clinical characteristics in patients with severe and mild COVID-19

No correlation was detected between ASCA-IgG or ASCA-IgA and sex, age, or clinical characteristics (fever, cough, and fatigue) in patients with severe COVID-19. A positive correlation was detected between ASCA-IgA and age ($r = 0.35, p = 0.01$) and ASCA-IgG and sex ($r = 0.3, p = 0.03$) in patients with mild COVID-19. No correlation was detected between ASCA-IgG or ASCA-IgA and clinical characteristics in patients with mild COVID-19.

Discussion

The present study demonstrated a significantly higher ASCA frequency in both patients with mild and severe COVID-19 than in healthy controls. These results confirm those of Sacchi et al. [20] who determined ASCA in a group of symptomatic and hospitalized patients with COVID-19. We found that IgA was the predominant isotype of ASCA in patients with severe COVID-19 (15.9 % vs 9 %). ASCA-IgA was more frequent than ASCA-IgG (25 % vs 17.5 %) even in the study of Sacchi et al. [20]. However, in all AIDs in which we previously detected ASCA [14–19] and in inflammatory bowel disease [22], IgG was the predominant isotype (Table 3). The discrepancy between AIDs and this severe viral infection could be because SARS-CoV-2-induced mucosal damage preferentially stimulates the IgA immune response [5]. Severe COVID-19 was associated with elevated serum IgA [23].

The frequency of ASCA-IgA in our patients with severe COVID-19 is lower than that of Sacchi et al. [20] (15.9 % vs 25 %). The difference
The nucleic acid binding domain of SARS-CoV non-structural protein

immunoassay. Moreover, we included 88 patients in our study but

ASCA assay and the number of patients studied. We used ELISA to
demonstrated in patients with COVID-19 [32]. The sterile alpha motif of the
nsp3 particularly shows close similarity to the RNA-binding site of
Vts1p protein [27]. Hence, both anti-SARS-CoV-2 antibodies and ASCA could
reactive epitopes between SARS-CoV and
Saccharomyces cerevisiae [5,42]. Thus, both ASCA and anti-SARS-CoV-2 antigens [41]. Remarkably, SARS-CoV-2 has also cross-reactive epitopes with many human proteins [5,42]. Third, a common epitope between S. cerevisiae and Candida albicans has been demonstrated [39]. Furthermore, a high risk factor for candidemia was found in patients with severe- COVID-19 [40]. Therefore, a high frequency of ASCA in patients with severe COVID-19 could also be explained by the high incidence of candidemia among these patients.

ASCAs could explain some extrapulmonary manifestations of COVID-
19. S. cerevisiae has structural similarities with many human auto-
tgens [41]. Additionally, SARS-CoV-2 has also cross-reactive epitopes with many human proteins [5,42]. Thus, both ASCA and anti-SARS-CoV-2 antibodies could bind to self-antigens and initiate a complement activation, which has a multigorgan impact on COVID-19 [36]. Cross-reactive epitopes between SARS-CoV and S. cerevisiae have been re-
ported [27]. Hence, both anti-SARS-CoV-2 antibodies and ASCA could bind to SARS-CoV-2 or S. cerevisiae which translocates from the gut to other organs via the vascular compartment because of a leaky intestinal wall [35] secondary to gut microbiota dysbiosis.

In conclusion, we have demonstrated a high frequency of ASCA in
patients with COVID-19 and revealed that IgA was the predominant
isotype of ASCA detected in patients with mild COVID-19.

Authors | Autoimmune diseases | ASCA IgG (%) | ASCA IgA (%) | p |
---|---|---|---|---|
Toumi D et al. 2007 (14) | Celiac disease | 24.8 | 8.8 | <10^-3 |
Sakly W et al. 2008 (15) | Primary biliary cholangitis | 18.9 | 11.6 | NS |
Sakly W et al. 2010 (16) | Type 1 diabetes | 21 | 9.8 | <0.002 |
Mankai A et al. 2013 (17) | Systemic lupus erythematosus | 29.3 | 12.1 | 0.001 |
Mankai A et al. 2013 (18) | Graves’ disease | 11.8 | 0.8 | 0.001 |
Mankai A et al. 2016 (19) | Antiphospholipid syndrome | 15.6 | 7.8 | 0.04 |
Wang et al. 2017 (22) | Crohn’s disease | 52.1 | 33.8 | 0.03 |
Wang et al. 2017 (22) | Ulcerative colitis | 31.7 | 17.1 | NS |

ASCAs: anti-Saccharomyces cerevisiae antibodies; NS: not significant.

between these results could be explained by both the method used for
ASCA assay and the number of patients studied. We used ELISA to
determine ASCA and Sacchi et al. [20] used fluorescent enzyme
immunoassay. Moreover, we included 88 patients in our study but
Sacchi et al [20] had only 40 patients.

Only the IgG isotype of ASCA was detected in our patients with mild
COVID-19 in contrast to those with severe COVID-19. This is because
serum samples has been done at the hospital admission for patients with severe COVID-19 and at least 14 days after RT-PCR analysis for
those with mild COVID-19. IgA has been demonstrated to dominate the early neutralizing antibody response to SARS-CoV-2 [24], while IgG could be detected by day 14 after the onset of symptoms [25]. Additionally, severely SARS-CoV-2-infected patients sustained increased IgA
SARS-CoV-2 antibodies compared with those with mild COVID-19 who
demonstrated an immunodominant IgG [26]. The present study deter-
mained ASCA but not anti-SARS-CoV-2 antibodies; however, interest-
gingly, cross-reactive epitopes exist between S. cerevisiae and SARS-CoV-
2. The Nucleic Acid Binding Domain of SARS-CoV non-structural protein
3 (nsp3) particularly shows close similarity to the RNA-binding site of
the sterile alpha motif of the S. cerevisiae Vts1p protein [27].

The present study revealed that ASCA were slightly more frequent in
patients with severe COVID-19 than in those with mild COVID-19 (21.6
% vs 13.7 %). We hypothesized that when ASCA are produced by the
immune system, they could neutralize S. cerevisiae which is a beneficial
yeast for our health [28,29]. Beta-glucan, derived from S. cerevisiae, primes the immune system to respond better to any viral infection [30]. Additionally, the use of oral beta-glucan was hypothesized to boost
immune responses and abrogate symptoms in COVID-19 [31]. Beta-
glucan is a polysaccharide that is abundantly found in the cell wall of
S. cerevisiae [31], and ASCA is directed against the peptidomannan of the
cell wall of S. cerevisiae.

The presence of ASCA in patients with COVID-19 could be explained by
three mechanisms. First, gut microbiota dysbiosis has been demon-
strated in patients with COVID-19 [32–34]. The consequent barrier
dysfunction [35] will allow S. cerevisiae to reach the intestinal mucosa.
The immune mucosa is therefore stimulated and ASCA is synthesized.
Second, during COVID-19, the lectin pathway of complement activation
is triggered by the binding of mannose-binding lectin (MBL) with SARS-
CoV spike protein [36]. Elevated MBL plasma levels have been reported
in patients with COVID-19, and it is associated with thrombosis and
coagulopathy in patients with severe COVID-19 [37]. Furthermore, the
transmembrane spike glycoprotein of SARS-CoV-2 is covered with N-
linked glycan having oligomannose and complex sugars [38]. Thus,
could we explain the high frequency of ASCA in patients with COVID-19
by an eventual molecular mimicry between mannan of the yeast cell
surface of S. cerevisiae and mannan of MBL or mannose of SARS-CoV-2?
Fascinatingly, plasma MBL levels are also high in AIDs, in which we have
previously detected ASCA, particularly TID, celiac disease, primary
biliary cholangitis, and Grave’s disease [18]. Third, a common epitope
between S. cerevisiae and Candida albicans has been demonstrated [39].

In conclusion, we have demonstrated a high frequency of ASCA in
patients with COVID-19 and revealed that IgA was the predominant
isotype of ASCA detected in patients with mild COVID-19.

Author’s contribution

SM, AM, MJ, MG, and IG contributed to the study concept and
design. SM, AM, MJ, ABC, ABA, KA, NG, MD, WB, MB, SM, and
WN contributed to the acquisition of data. SM, AM, MG, ABA, KA, NG, MD,
WB, MB, SM, WN, and IG contributed to the analysis and interpretation of
data. SM, AM, MJ, MG, and IG contributed to the drafting of the
manuscript. SM, AM, MG, ABA, KA, NG, MD, WB, MB, SM, WN, and IG
carried out the critical revision of the manuscript for important
intellectual content. All authors identified above approved the final
version of this paper, including the authorship statement.

Declaration of competing interests

The authors declare that they have no known competing financial
interests or personal relationships that could have appeared to influence
the work reported in this paper.

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Monastir, Tunisia.

Ethical approval

All procedures performed in studies involving human participants
were in accordance with the ethical standards of the institutional and/or
national research committee and with the 1964 Helsinki Declaration and
its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants
included in the study.

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