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Short Communication

Internal Medicine Section

COVID-19 and Autoimmunity: Unravelling the GAD65 Connection in Non-diabetic Patients

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ABSTRACT

Introduction: The Coronavirus Disease-2019 (COVID-19) pandemic has raised significant concerns regarding its long-term health impacts, particularly among individuals without pre-existing conditions. Emerging evidence suggests a potential link between Severe Acute Respiratory Syndrome-Corona Virus-2 (SARS-CoV-2) infection and autoimmune responses, including the development of Type 1 diabetes. GAD65 (Glutamic Acid Decarboxylase 65) antibodies have been identified as key markers in the autoimmune pathway leading to Type 1 diabetes.

Aim: To investigate the development of GAD65 antibodies at discharge in a cohort of non-diabetic COVID-19 patients as well as the relationships between glycaemic control, inflammatory markers, and severity of illness in a cohort of non-diabetic COVID-19 patients.

Materials and Methods: The present cross-sectional observational study included 69 non-diabetic patients diagnosed with COVID-19 who were admitted to Dr. D. Y. Patil medical college, hospital and research centre, Pune, Maharashtra, India between October 2020 to October 2022. Data regarding demographic and biochemical parameters, including HbA1c and various inflammatory markers {Lactate Dehydrogenase (LDH), ferritin, D-Dimer, C-Reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR)} and development of GAD65 antibodies were collected at discharge. Statistical analyses included Pearson's correlation coefficients and Kruskal-Wallis tests to evaluate associations.

Results: The study involved 69 participants with a median age of 48 years, ranging from 36 to 64 years including 47 males (67.1%) and females 22 (31.4%). The findings revealed no significant correlations between HbA1c and inflammatory markers {LDH (r-0.05, p-0.681); ferritin (r-0.069, p-0.576); CRP (r-0.027, p-0.824); ESR (r-0.009, p-0.942)}. However, D-Dimer levels were significantly higher in patients with severe illness (p= 0.022). One patient developed GAD65 antibodies at discharge, suggesting potential autoimmune development after COVID-19 infection.

Conclusion: The study underscores the non-significant association of traditional inflammatory markers with glycaemic control while highlighting D-Dimer's relevance in illness severity. The development of GAD65 antibodies in a patient raises considerations for emergence of autoimmunity which may lead to type 1 diabetes. Further research is warranted to explore these associations in broader populations.

Keywords: Coronavirus Disease-2019, Inflammatory markers, Pancreatic beta cell, Severe acute respiratory syndrome-corona virus 2, Type 1 diabetes

INTRODUCTION

Raised HbA1c in non-diabetic individuals is linked to increased risk of severe COVID-19, while COVID-19 itself may trigger autoimmune pathways leading to Type 1 Diabetes Mellitus (T1DM) [1]. This bidirectional relationship highlights the interplay between glycaemic health and immune response. There is a large body of epidemiological and animal model-based evidence to support the hypothesised role of viral infections in initiation of Islet Autoimmunity (IA) and the progression of T1DM [2-4]. These viruses include Enterovirus (EV), Rotavirus, Cytomegalovirus, Epstein-Barr virus, Parechovirus, Influenza, Parvovirus, Mumps, Rubella and Human endogenous retrovirus.

To date, the most robust evidence supports an association between EVs and IA [5]. A number of non-mutually exclusive mechanisms are proposed by which EV can induce T1DM: direct cytolysis, bystander activation, persistent infection and microRNA dysregulation. Highly homologous epitopes are expressed between 2C protease of EV and human islet autoantigen GAD65. This leads to cross reactivity generation as antiviral T-lymphocytes also destroy islet cell due to molecular mimicry between the two [5]. Similarly, coxsackie virus can induce T1DM by molecular mimicry and bystander damage. GAD65 also shows sequence homology with the coxsackie virus protein P2C, therefore it is likely that P2C specific immune response is induced which is cross reactive with GAD65 [6], islet cell autoantibodies (including GAD65 autoantibody) are widely used as biomarkers for Diabetes Mellitus 1 (DM1) diagnosis because they are present in more than 85% of patients at diagnosis [7]. Although there is no clear role of GAD65 autoantibody in the pathophysiology of COVID-19, anti-GAD65 can also be associated with specific neurologic disorders, including Stiff-Person Syndrome (SPS), Cerebellar Ataxia (CA), Epilepsy (Ep), and Limbic Encephalitis (LE) [8]. COVID-19's neurological effects may be partially mediated by such autoimmune mechanisms in some patients.

It is reasonable to suspect increased prevalence of T1DM in post COVID-19 era [9], but for concrete conclusion, evidence is required to accept or refute this hypothesis. Since COVID-19 has affected populations around the globe, increased prevalence of autoimmune diseases in wake of COVID-19 infection due to induced autoimmunity will require different strategic management in terms of screening and prevention policies. This will bear a huge impact on national policies conception. Understanding the development of GAD65 autoantibodies in COVID-19 patients may help identify individuals at increased risk of progressing to T1D.

Early identification of high-risk individuals may facilitate timely interventions, including monitoring, lifestyle modifications, and possible immunomodulatory therapies, to prevent or delay the onset of T1D.

The present study report comprised a part of a broader ongoing study that has a aim to find out whether COVID-19 infection predisposes to development of autoimmune T1D and GAD65 can be used as a predictor marker for development of diabetes or not. This study provides valuable insights into the clinical and biochemical profile of

non-diabetic COVID-19 patients and the rare incidence of GAD65 antibody development.

This study aimed to investigate the development of GAD65 antibodies at discharge in a cohort of non-diabetic COVID-19 patients as well as the relationships between glycaemic control, inflammatory markers, and severity of illness in a cohort of non-diabetic COVID-19 patients.

MATERIALS AND METHODS

The present cross-sectional observational study was conducted in Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, India, from October 2020 to October 2022. (IEC approval number- DYPV/EC/542/2020) written informed consent was taken from all participants before inclusion in the study.

Inclusion criteria: Patients were included based on their confirmed COVID-19 status via Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) and absence of a prior diabetes diagnosis after taking a written consent.

Exclusion criteria: Patients who were already diagnosed diabetic or newly diagnosed diabetic at the time of admission were excluded. Patients who were diagnosed with malignancy or who had a history of autoimmune disorders were also excluded as it may act as confounding factor for the development of GAD65 antibody. Any other condition which the principal investigator thought might jeopardise the study were also excluded for practical reason.

Sample size selection: During the study period, a total of 69 patients who presented with the confirmed RT-PCR satisfying inclusion criteria were enrolled.

Study Procedure

Demographic details and biochemical parameters were recorded at the time of admission and discharge. Clinical severity was assessed on the basis of respiratory rate and SpO_2 as per the Government of India Ministry of Health and Family Welfare Directorate General of Health Services (EMR Division) [10]. RR ≥30/minute and/or SpO_2 <90% on room air was considered as severe; RR ≥24/minute and/or SpO_2 <95% on room air was considered as moderate and upper respiratory tract symptoms without breathlessness or hypoxia was considered as mild disease.

Biochemical parameters included haemoglobin (Hb-13.0-17.0 g/dL for males, 12.0-15.0 g/dL for females), Total Leucocyte Count (TLC)-4,000-11,000 cells/µL, platelets (150,000-450,000/µL), liver function tests including Serum Glutamic-Oxaloacetic Transaminase (SGOT) (5-40 U/L) and Serum Glutamic Pyruvic Transaminase (SGPT) (7-56 U/L), kidney function tests including urea (15-40 mg/dL) and creatinine (0.6-1.2 mg/dL), LDH- 140-280 U/L, ferritin (30-400 ng/mL for males, 15-150 ng/mL for females), D-dimer (<500ng/mL Fibrinogen equivalent Units), C-Reactive Protein (CRP) <5 mg/L, Erythrocyte Sedimentation Rate (ESR) <20 mm/hour for males, <30 mm/hour for females, HbA1c (<5.7% for non-diabetics), and random blood sugar levels (BSL – <200 mg/dL). GAD65 antibody status using Enzyme linked immunosorbent assay (<5 units/mL considered negative and ≥5 units/mL considered positive) was assessed at discharge. The reference ranges were obtained from the hospital laboratory, while the cut-off value for GAD65 was defined according to the manufacturer's specifications. Patients are being followed up for the development of type 1 diabetes at 2 years postdischarge.

STATISTICAL ANALYSIS

Statistical analysis was conducted using MS Excel (Microsoft 365) and IBM Statistical Package for Social Sciences (SPSS) Statistics for Windows, Version 27.0 (IBM Corp. Released 2020. Armonk, NY: IBM Corp). Data was presented using mean and standard deviation or median Interquartile Range (IQR) for skewed variables for the quantitative data and frequency (percentage) for all the categorical variables. The correlation between HbA1c and the inflammatory markers was checked using Pearson's correlation coefficient. The association between the severity of illness and the inflammatory markers were calculated using Independent-Samples Kruskal-Wallis Test. For all the tests p-value<0.05 was considered as statistically significant.

RESULTS

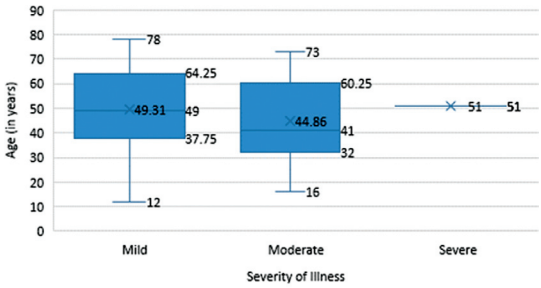
The study involved 69 participants with a median age of 48 years, ranging from 36 to 64 years. Among the cohort, a majority were male 47 (67.1%), while females comprised 22 (31.4%). Glycaemic control was reflected by an average HbA1c of 5.00±0.86% and a random blood sugar level of 143.64±21.95 mg/dL. The majority of patients exhibited mild severity of illness 54 (77.1%), with moderate and severe cases representing 14 (20.0%) and 1 (1.4%), respectively. The average duration of hospital stay was approximately 11.46±3.92 days [Table/Fig-1].

Parameters (n=69)	Descriptive statistics
Demographic details	
Age (in years)‡	48 (36-64)
Sex; n (%)	
Male	47 (67.1%)
Female	22 (31.4%)
Biochemical parameters	
Hb (g/dL)	12.26±1.95
TLC‡ (cells/µL)	7000 (4860-8900)
Platelets (cells/µL)	2.31±0.94
Total bilirubin (mg/dL)	0.67±0.46
SGOT‡ (U/L)	29 (19.5-52.5)
SGPT‡ (U/L)	28 (19-45.5)
Urea‡ (mg/dL)	26 (19.5-37.5)
Creatine (mg/dL)	1.11±1.05
LDH‡ (U/L)	320 (263.5-424)
Ferritin ‡ (ng/mL)	250 (85.5-346.5)
D-dimer ‡ (ng/mL)	412 (280-598)
CRP‡ (mg/L)	36.5 (16.25-80.25)
ESR‡ (mm/hr)	24 (16.5-47)
HbA1c (%)	5.00±0.86
BSL Random (mg/dL)	143.64±21.95
GAD65 Antibodies; n (%)	1 (1.4%)
Duration of stay (days)	11.46±3.92
Severity of illness; n (%)	
Mild	54 (77.1%)
Moderate	14 (20.0%)
Severe	1 (1.4%)

[Table/Fig-1]: Summary of all the parameters (n=69).
Values represented are mean±SD or Median (Interquartile range) for skewed variables (indicated by ‡) for quantitative variables. Categorical variables are represented by frequency (%) {indicated as n (%)}

Out of 69 patients, 41(59.4%) were unvaccinated against COVID-19, 28 (40.5%) were vaccinated with Covishield vaccine out of which one was vaccinated with one dose and rest were vaccinated with two doses. The patient who developed GAD65 antibody was not vaccinated. Overall, these findings provide a comprehensive overview of the demographic and clinical characteristics of the patient population.

The box plot illustrates the comparison of age distribution across three categories of illness severity [Table/Fig-2]. In the mild severity group, ages ranged from 12 to 78 years, with a median age of 49.31 years and an IQR of 37.75 to 64.25 years. For the moderate severity group, ages ranged from 16 to 73 years, with a median age of 44.86 years and an IQR of 32 to 60.25 years. The severe category included only one patient, aged 51 years. Overall, the data indicates that age may play a role in the severity of illness, with



[Table/Fig-2]: Age distribution as per the severity of illness.

the mild severity group encompassing a broader age range, and moderate severity displaying a more concentrated age distribution.

The results indicate weak correlations with LDH (0.05, p=0.681), ferritin (0.069, p=0.576), and D-dimer (0.162, p=0.183), none of which reached statistical significance. Additionally, the correlation between HbA1c and CRP was minimal (0.027, p=0.824), while the correlation with the ESR was virtually non-existent (-0.009, p=0.942). Overall, none of the inflammatory markers showed a statistically significant correlation with HbA1c [Table/Fig-3].

Correlation of HbA1c with inflammatory markers	Pearson's correlation coefficient	p-value
LDH	0.05	0.681
Ferritin	0.069	0.576
D-dimer	0.162	0.183
CRP	0.027	0.824
ESR	-0.009	0.942

[Table/Fig-3]: Correlation of HbA1c with inflammatory markers.
Values represented are Pearson's correlation coefficient. Test used: correlation test. p-value <0.05*; statistically significant i.e., Correlation is present

[Table/Fig-4] analyses the association of severity of illness with various inflammatory markers among the study participants. The mean LDH levels were highest in the severe group (515.00±0), followed by moderate (386.14±132.40) and mild (343.02±121.32), but this difference was not statistically significant (p=0.176). Ferritin levels also showed no significant variation among the groups, with means of 345.00±382.11 in mild cases, 220.09±204.87 in moderate, and 52.66 in severe (p=0.259). D-Dimer levels, however, displayed a significant difference (p=0.022), with a mean of 1054.46±2370.43 in mild cases, compared to 349.07±284.97 in moderate cases, and a markedly elevated level of 5286.00 in the severe case. Inflammatory markers CRP and ESR showed no significant differences across the severity groups.

Inflammatory markers	Mild 54 (77.1%)	Moderate 14 (20.0%)	Severe 1 (1.4%)	p-value
LDH	343.02±121.32	386.14±132.40	515.00	0.176
Ferritin	345.00±382.11	220.09±204.87	52.66	0.259
D-dimer	1054.46±2370.43	349.07±284.97	5286.00	0.022*
CRP	73.33±96.73	72.11±139.46	135.00	0.299
ESR	33.43±22.83	26.57±15.49	80.00	0.236
HbA1c	4.95±0.86	5.14 ±0.87	5.8	0.499

[Table/Fig-4]: Association of severity of illness with inflammatory markers.
Values represented are mean±SD. Test used: Independent-Samples Kruskal-Wallis Test. p-value <0.05*; statistically significant

DISCUSSION

The findings from this study indicate that while various inflammatory markers are commonly thought to correlate with glycaemic control and disease severity, the correlations observed with HbA1c and the severity of illness were minimal. This is in contrast with study conducted by Alhakak A et al., who reported among patients without diabetes, varying HbA1c levels were associated with higher risk of the composite outcome [1]. The discrepancies may arise from differences in study populations, such as variations in age and the presence of co-morbidities. The present study cohort was predominantly middle-aged and included a relatively low number of severe cases, potentially limiting the generalisability of the findings.

The current study found no significant correlations between HbA1c and inflammatory markers such as LDH, ferritin, CRP, and ESR. This finding aligns with the finding from another study by Subramanian VB et al., where inflammatory markers like ESR, CRP, D-dimer, ferritin and LDH were compared between diabetic and non-diabetic groups. These markers were significantly elevated irrespective of diabetic status and were elevated in all severe cases and its various complications like ventilator requirement and other COVID-19 related complications and death [11].

D-Dimer levels exhibited a significant relationship with illness severity in this study, which aligns with previous research highlighting the role of hypercoagulability in patients with severe illness. For instance, a study by Thachil J et al., demonstrated that elevated D-Dimer levels correlate with disease severity in COVID-19 patients, suggesting that systemic inflammation and coagulopathy are interconnected [12].

In a study by Zhou F et al., a higher D-dimer level on admission was related to a worse prognosis of COVID-19 [13]. The lone severe case in the present study may have skewed the results, highlighting the need for larger samples to draw more robust conclusions.

In terms of other inflammatory markers such as LDH and ferritin, while findings of the current study indicated no significant correlation with severity, literature suggests that elevated LDH and ferritin levels often reflect tissue damage and inflammation. A study by Zhao K et al., (2020) reported elevated LDH and ferritin levels in severe COVID-19 cases, linking these markers to poor outcomes [14]. The difference in results could be due to the timing of blood sample collection relative to the onset of illness or the heterogeneity in patient populations.

In the present study, one patient tested positive for GAD65 antibodies at discharge, suggesting the development of an autoimmune response. A similar study including 95 patients conducted by Kayhan S et al., showed that anti-islet autoantibody and GAD autoantibody positivity were 4.2% and 1.1%, respectively in COVID-19 patients in the initial evaluation [15]. Follow-up after 3rd month anti-islet autoantibody positivity decreased by 3.1% however anti-GAD autoantibody positivity remained the same. Although they concluded that COVID-19 infection can induce autoimmunity against pancreatic autoantigens and the patient should be followed up for development of T1DM.

Another case report by Marchand L et al., showed a 29-year-old woman who developed one month after her first symptom of COVID-19 acute polyuria-polydipsia syndrome. Patient was diagnosed with raised blood sugar and autoantibodies GAD65 was positive. Although this patient had family history of diabetes (aunts with T2DM; a cousin with T1DM), the temporality of development of diabetes one month after onset of first symptom of COVID-19 reveals the fact COVID-19 infection may trigger onset of type 1DM in genetically susceptible individuals [16].

In this study, only one patient with severe COVID-19, who had the longest hospital stay of 22 days, developed GAD65 antibodies upon discharge. Rest of the patients were found to be negative for the development of GAD65 antibodies at discharge. This finding may be attributed to the timing of the antibody tests, as antibody development can vary in duration. The development of these antibodies in a previously non-autoimmune patient could be attributed to several factors, including environmental triggers or viral infections, which have been implicated in the onset of autoimmune responses in genetically predisposed individuals. According to a narrative review by Hansen N, evidence from research suggests that it is highly likely that neural autoantibody production is facilitated by SARS-CoV-2 infection, and that more neuropsychiatric patients than control subjects will present neural autoantibodies. It underscores the importance of monitoring of neurological manifestations other than development of type 1 diabetes in these patients [8].

There are growing concerns about the potential for COVID-19 vaccines to induce autoimmune responses, particularly autoimmune diabetes, through mechanisms such as molecular mimicry, bystander activation, and epitope spreading. Studies, including one by Tang X et al., highlight the role of genetic susceptibility in the development of Fulminant Type 1 Diabetes Mellitus (FT1DM) after vaccination. Tang X et al., reported a case of a 50-year-old male who developed FT1DM five days after receiving an inactivated COVID-19 vaccine. Laboratory tests revealed hyperglycaemia, ketosis, metabolic acidosis, and the presence of specific Human Leukocyte Antigen (HLA) alleles, which are linked to FT1DM susceptibility [17]. This suggests that in genetically predisposed individuals, vaccination might trigger autoimmune responses. While mRNA vaccines have been associated with autoimmune conditions in some studies, the mechanisms for inactivated vaccines remain unclear [18]. In this study however, where patients received inactivated vaccines and also developed COVID-19 disease, it is difficult to attribute autoimmune reactions solely to the vaccine, as COVID-19 infection itself could also play a role.

Overall, while this finding is isolated, it underscores the importance of considering autoimmune mechanisms in the development of diabetes in patients with particularly severe COVID-19 disease. Further studies with broader patient population may reveal more discrete evidence for development of autoimmunity and development of diabetes in these patient populations.

Limitation(s)

The small sample size (69 participants) limits the generalisability of the findings. The study lacks a control group of non-COVID-19 individuals for comparison. Other potential confounding factors, such as genetic predisposition and environmental influences, were not accounted for. Also, antibody levels were only measured at discharge, missing possible transient or delayed autoimmune responses. Larger, longitudinal studies are needed to validate these findings and assess long-term risks.

CONCLUSION(S)

In summary, the present study findings highlight the complex relationships between glycaemic control, inflammatory markers, and disease severity, with D-Dimer showing a significant correlation with illness severity. Additionally, the development of GAD65 antibodies in one patient at discharge suggests potential autoimmune processes that warrant further investigation. These observations emphasise the need for comprehensive evaluations in post COVID-19 patients developing diabetes, considering autoimmune factors to guide management and prognosis. Further research including larger sample size and longitudinal design are needed to validate these findings and assessment of long-term risks.

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