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Glycemic parameters in patients with new-onset diabetes during COVID-19 pandemic are more severe than in patients with new-onset diabetes before the pandemic: NOD COVID India Study

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ABSTRACT

Background and aims: It is not known if new onset diabetes during Coronavirus-19 disease (COVID-19; NOD COVID) is phenotypically or biochemically different than new onset diabetes before COVID-19 (NOD).

Methods: All adults diagnosed with new onset diabetes from during the time of COVID-19 were compared with new onset diabetes prior to COVID-19 from two tertiary care hospitals in Chennai and Delhi. RTPCR test for SARS-CoV-2 virus was done as appropriate, and COVID-19 antibody test was done in all other NOD COVID patients.

Result: A total of 555 patients with new onset diabetes were included in the study (282 NOD and 273 NOD COVID patients). Patients with NOD COVID had higher fasting and post prandial blood glucose and glycated hemoglobin levels *vs.* NOD patients. Both the groups had high average body mass index; ~28 kg/m^2. Interestingly, fasting C-peptide levels were significantly higher in the NOD COVID group *vs.* NOD group. There was no difference in C-peptide levels or glycemic parameters between the COVID-19 antibody positive and negative NOD COVID cases.

Conclusion: Individuals who were diagnosed with diabetes during COVID-19 epidemic (NOD COVID) do not significantly differ from those diagnosed before COVID-19 in symptomatology, phenotype, and C-peptide levels but they had more severe glycemia.

1. Background

Diabetes is one of the most important comorbidities significantly contributing to adverse outcomes during the ongoing novel COVID-19 pandemic caused by the novel Severe Acute Respiratory Syndrome- Coronavirus 2 (SARS-CoV-2) [1]. Specifically, those with uncontrolled diabetes are at high risk for developing COVID-19 related complications, including acute respiratory distress syndrome leading to increased mortality [2,3].

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In order to slow down the COVID-19 pandemic, a strict lockdown was enforced in India in March 2020, initially for a period of 21 days and it was subsequently extended till the end of May. During this time, people faced a number of challenges; inability to exercise in open spaces, poor availability of healthy food options and difficulty in accessing medical support. These challenges continue to exist, albeit to a lesser extent, even after lifting of the lockdown. There is, therefore, the possibility of an increase in various lifestyle-related diseases including new-onset diabetes (NOD, henceforth termed as NOD COVID) during this pandemic period. The adverse lifestyle changes occurring as a result of lockdown might also lead to acceleration of progression from prediabetes to diabetes in many individuals. In addition, it is now being recognized that several factors related to COVID-19 infection and its treatment may play a role in the development of NOD [4].

It is well-known that viral infections can trigger diabetes. Previously, diabetes has been reported following infections with influenza and dengue viruses in India [5,6]. Further, islet cell autoimmunity and subsequent type 1 diabetes was reported after infection with H1N1 pandemic influenza virus [7]. Cases of pancreatic damage and new-onset diabetes following COVID-19 (including some with diabetic ketoacidosis and presentation similar to type 1 diabetes) have been reported from India [8] and other countries [9]. Pancreatic beta-cells carry Angiotensin Converting enzyme-2 (ACE-2), a receptor which facilitates entry of SARS-CoV-2 into the cell, followed by damage to the cell and subsequent development of diabetes [10].

We hypothesized that both the COVID-19 pandemic and the response to it (in the form of lockdown) could increase the incidence and severity of diabetes compared to before the pandemic because of the multiple putative pathophysiological factors discussed above.

The present study was undertaken in newly diagnosed adults with type 2 diabetes mellitus (T2DM) from two sites in India, namely Chennai and Delhi, to examine whether diabetes with onset during COVID-19 (NOD COVID) was different from new onset diabetes before COVID-19 (NOD) in terms of clinical and biochemical profiles.

2. Methodology

All adults diagnosed with new onset T2DM from 1st April till 30th October 2020 from two tertiary care hospitals for diabetes in Chennai and Delhi were included in the study. These patients constituted the NOD COVID group. As a comparator group, we selected age- and sex-matched adults diagnosed with new onset diabetes from 1st September 2019 to 29th February 2020, from the same hospitals. This was the NOD group. A structured questionnaire was used to capture data on symptomatology, family history of diabetes, behavioral habits, anthropometry, and glycemic parameters. These measurements were done at both sites 'in person' for the NOD COVID group and as a telephonic interview for the NOD group. For the NOD COVID group, responses to questions on symptomatology for COVID-19, a basic questionnaire to assess stress, whether a reverse transcriptase polymerase chain reaction (RTPCR) COVID-19 test was done, or was already carried out previously, and information on hospitalization for COVID-19 including drug usage, specifically use of corticosteroids, were recorded. For all patients of NOD COVID, other than those in whom RTPCR was performed based on symptoms, immunoassay to qualitatively detect antibodies against SARS-CoV-2 i.e. the COVID-19 antibody test was done. The Elecsys Anti-SARS-CoV-2 assay uses a recombinant protein representing the nucleocapsid (N) antigen for the determination of antibodies against SARS-CoV-2 using the Sandwich principle. The test has sensitivity of 100% and specificity of 99.8%–99.9% [11].

Institutional review board permission was taken independently at both sites. Informed consent was obtained from all patients before the start of the study.

3. Results

A total of 555 patients with new onset diabetes were included in the study (Chennai n = 386, Delhi n = 169). This comprises 282 NOD and 273 NOD COVID. Table 1 shows the characteristics of the two groups of patients. There were no significant differences in the mean age, sex distribution, body mass index (BMI) and family history of diabetes between the NOD and the NOD COVID groups. However, fasting blood glucose, post prandial blood glucose, glycated hemoglobin and fasting C-peptide were found to be significantly higher in the NOD COVID group. These differences, except C-peptide levels, held true even after stratifying patients by study site i.e. North India *vs*. South India (Table 2). There were also no significant differences in the treatment pattern for diabetes between the groups, though these data were incomplete.

COVID-19 antibody levels were assessed for 167 patients (n = 67, Delhi and n =100 in Chennai). A total of 44 patients (26.3%) were found to be positive (Delhi, 15/67, 22.3%, Chennai, 29/100, 29%) probably reflecting higher numbers of COVID-19 cases in Chennai compared to Delhi at the time of the study.

In addition, a total of 25 patients self-reported that they had been COVID-19 positive (16 in North India and 9 in South India), of whom confirmed RT-PCR test reports were available for 13/16 north Indian patients and 8/9 South Indian patients.

Table 3 compares the characteristics of patients with NOD based on COVID-19 antibody status. There were no differences in the risk factors for diabetes between COVID-19 antibody positive and negative patients. There were also no differences in the biochemical parameters based on COVID-19 antibody status.

As shown in Fig. 1, more patients diagnosed with diabetes during COVID-19 reported symptoms such as weakness, polyuria, polyphagia, headache, giddiness, burning micturition, cough, running nose, sneezing and breathlessness compared to those diagnosed before COVID-19. A higher proportion of NOD COIVID individuals (50.9%) reported increased "stress" levels compared to NOD group (12.8%) (Fig. 2).

Patients with new onset of diabetes who had a history of having suffered from COVID-19 (self-reported COVID-19 infection) had higher fasting blood glucose, post prandial blood glucose and glycated hemoglobin levels compared to those without a similar history. There was a history of steroid use in 15 patients. However, there were no differences in symptomatology or risk factors of diabetes or other biochemical characteristics between those who reported having had COVID-19 compared to those who did not. Two patients of NOD COVID presented with diabetic ketoacidosis, which later resolved and glycemia was controlled on oral antihyperglycemic drugs after initial insulin therapy. These have been previously reported [8, 12].

4. Discussion

This is the first study to have looked at new onset diabetes during the COVID-19 pandemic in India. We show the following:

1. New onset diabetes during COVID-19 (NOD COVID) had worse glycemic parameters and higher C-peptide levels compared to NOD; however, there was no difference in clinical, anthropometric, behavioural, or other biochemical parameters.
2. There was also no difference in the above parameters between NOD COVID and NOD based on COVID-19 antibody levels.

Table 1
Clinical & biochemical characteristics of the new onset diabetes cases before (NOD) and during COVID-19 (NOD COVID).

Variable	NOD (n = 282)	NOD COVID (n = 273)	P value
Age (years)	46.4 ± 12.8	46.2 ± 12.6	0.852
Gender (male)	172 (61.0)	154 (56.4)	0.156
Body mass index (kg/m^2)	27.9 ± 5.0	28.3 ± 5.5	0.405
Family history of DM n (%)	106 (37.6)	106 (38.8)	0.416
Smoking (yes) n (%)	21 (7.4)	17 (6.2)	0.345
Alcohol (yes) n (%)	45 (16.0)	33 (12.1)	0.117
Fasting plasma glucose (mg/dl)	163 ± 66	203 ± 97	<0.001
Post prandial blood glucose (mg/dl)	238 ± 97	303 ± 127	<0.001
HbA1c (%)	9.2 ± 2.4	10.1 ± 2.5	<0.001
C-peptide fasting (pmol/ml)	1.16 ± 0.73	2.1 ± 2.6	<0.001
COVID-19 Antibody Tested (positive) n (%)	–	44/167 (26.3)	
Treatment n (%)[a]			
Insulin	20 (7.1)	55 (20.1)	
Oral anti-diabetes drugs + Insulin	26 (9.2)	36 (13.2)	0.064
Anti-diabetes drugs	171 (60.6)	32 (11.7)	

NOD, New Onset Diabetes prior to COVID-19.
NOD COVID, New Onset Diabetes diagnosed during COVID-19.
[a] Treatment data are incomplete for some NOD patients. In many NOD COVID patients, treatment data were recorded when it was not initiated.

Table 2
Clinical and biochemical characteristics of patients the before COVID-19 (NOD) and during COVID-19 (NOD COVID) in South and North India, respectively.

Variable	South India (n = 386)		p value	North India (n = 169)		p value
	NOD (n = 193)	NOD COVID (n = 193)		NOD(n = 89)	NOD COVID (n = 80)	
Age (years)	46.2 ± 12.3	46.4 ± 12.5	0.873	46.9 ± 13.8	45.7 ± 12.7	0.586
Gender (male)	112 (58.0)	112 (58.0)	1.000	60 (67.4)	42 (52.5)	0.048
Body mass index (kg/m^2)	27.4 ± 4.7	27.6 ± 5.0	0.629	29 ± 5.3	29.8 ± 6.3	0.376
Family history of DM n (%)	56 (29.0)	57 (29.5)	0.911	50 (56.2)	49 (61.3)	0.504
Smoking yes n (%)	18 (9.3)	10 (5.2)	0.116	3 (3.4)	7 (8.8)	0.139
Alcohol yes n (%)	32 (16.6)	22 (11.4)	0.142	13 (14.6)	11 (13.8)	0.873
Fasting plasma glucose (mg/dl)	156 ± 59	199 ± 87.2	<0.001	180 ± 75	211.6 ± 117.4	0.040
Post prandial blood glucose (mg/dl)	238 ± 96	314 ± 123	<0.001	237 ± 99	278 ± 135	0.024
HbA1c (%)	9.4 ± 2.4	10.2 ± 2.7	0.002	8.7 ± 2.3	9.8 ± 2.0	0.002
C-peptide fasting (pmol/ml)	1.11 ± 0.57	0.92 ± 0.40	0.003	2.9 ± 2.3	3.8 ± 3.5	0.564
COVID-19 Antibody Tested (positive) n (%)	–	29/100 (29.0)	–	–	15/67 (22.3)	–

NOD, New-onset diabetes prior to COVID-19; NOD COVID, New-onset diabetes diagnosed during COVID-19.

Table 3
Clinical and biochemical characteristics of NOD COVID cases stratified according to the COVID-19 antibody positivity.

Variable	COVID-19 Antibody Positive (n = 44)	COVID-19 Antibody Negative (n = 123)	P value
Age (years)	43.3 ± 9.9	46.6 ± 13.8	0.152
Gender (male)	23 (52.3)	67 (54.5)	0.802
Body mass index (kg/m^2)	27.8 ± 5.5	28.6 ± 5.6	0468
Family history of DM n (%)	18 (40.9)	54 (43.9)	0.731
Smoking (Yes) n (%)	2 (4.5)	9 (7.3)	0.525
Alcohol (Yes) n (%)	5 (11.4)	16 (13.0)	0.778
Fasting plasma glucose (mg/dl)	192 ± 70	200 ± 84	0.587
Post prandial blood glucose (mg/dl)	286 ± 100	295 ± 113	0.655
HbA1c (%)	10.2 ± 2.5	10.2 ± 2.4	0.869
C-peptide fasting (pmol/ml)	1.9 ± 1.7	2.5 ± 3.07	0.254
Treatment n (%)[a]			
Insulin	6 (13.6)	19 (15.4)	
Oral anti-diabetes drugs + Insulin	4 (9.1)	12 (9.8)	0.863
Oral anti-diabetes drugs	24 (54.5)	58 (47.2)	

[a] Some treatment data are incomplete.

3. There appears to be no major damage to beta cells (as assessed by C-peptide levels) from COVID-19 infection.

There could be multiple reasons for higher magnitude of glycemia in patients with NOD COVID as compared to NOD. The first reason which seems to be apparent from our data is significantly higher levels of stress, although we have not characterised the type of stress in our study. The panic and fear due to the COVID-19, as well as the lockdown, have led to an increase in stress levels across all sections of society. Fear of contracting the virus, of losing loved ones to the disease, of losing one's job and livelihood as well as challenges associated with working from home, may have all contributed [13]. Stress can precipitate hyperglycemia by different mechanisms: adoption of harmful health behaviors including binge eating and decreased physical activity, dysregulated physiological stress response with outpouring of counterregulatory hormones (such as glucocorticoids) and development of chronic low-grade inflammation [14]. While hyperglycemia resolves with

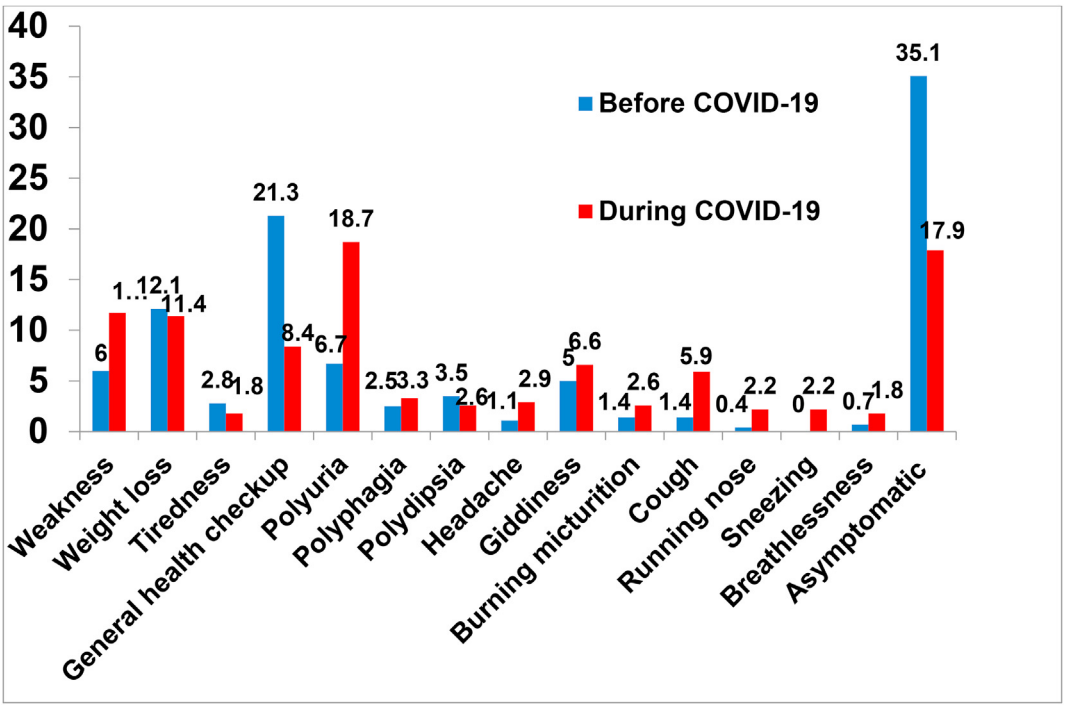

Fig. 1. Symptoms at onset of diabetes before and during COVID-19 i.e. in patients with NOD *vs.* NOD COVID.

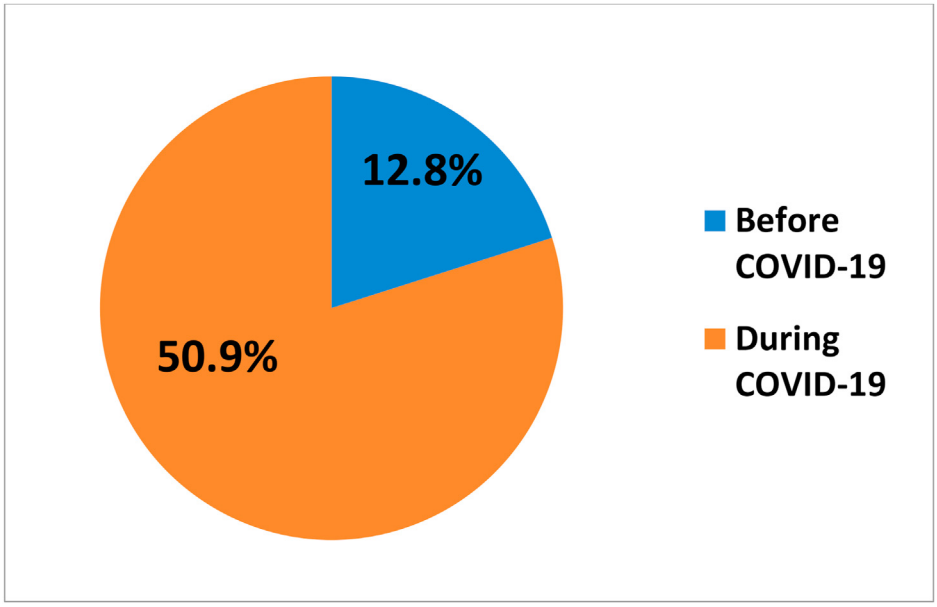

Fig. 2. Stress levels before and during COVID-19 i.e in NOD patients *vs.* NOD COVID patients.

amelioration of stress in many cases, it may persist in some, leading to development of NOD, progression from pre-diabetes to diabetes or worsening of existing diabetes.

A likely explanation relates to changes in dietary and exercise profiles during the pandemic and lockdown. However, the direction of these changes depends on the previous lifestyle of patients. Previous studies from North India have shown a trend of weight gain during the lockdown period. Specifically, carbohydrate consumption and frequency of snacking increased in 21% and 23%, respectively; exercise duration was reduced in 42% and weight gain occurred in 19% patients [15]. Even in apparently non-diabetic individuals, weight gain was seen in 40% of the cohort during lockdown, with 16% of the population experiencing a 2.1–5 kg weight increment [13,16]. Imbalance in eating habits and increase in weight have also been shown in studies from other parts of India [17] and from other countries as well [18,19]. In particular, increased snacking during lockdown was correlated with increase in BMI in a UK based study [19]. In this context, it is important to note trend of higher BMI of patients with NOD COVID (27–28 kg/m^2) *vs* NOD in the current study. While obesity most likely predated the onset of COVID-19 in these patients, it is likely that the enforced lifestyle changes during the lockdown accelerated development of

diabetes. It is also likely that many of these patients were already having undiagnosed diabetes, or rapidly converted from prediabetes to diabetes during the pandemic, for the same reasons. However, some other studies from south India have shown no major changes (or even improvement) in glycemia and improved eating habits and exercise regimen during the lockdown [20]. These differences likely represent socioeconomic or cultural differences but do emphasize the fact that the adverse effects of lockdown on glycemia are not inevitable.

Yet another explanation for higher magnitude of glycemia among NOD COVID vs. NOD patients could be delayed diagnosis, either due to difficulty in accessing lab facilities for blood testing or fear of contracting COVID-19 while visiting healthcare facilities. It is also more likely that only individuals with more severe hyperglycemia approached healthcare providers during the pandemic, as suggested by the higher prevalence of various symptoms of hyperglycemia in the NOD COVID group.

A recently debated question is whether SARS-CoV-2 could attack and destroy pancreatic beta cells sufficiently to cause hyperglycemia, given that ACE-2 receptors are present on endocrine pancreas [10]. A direct attack of virus on pancreas is a real possibility, given previously known data of other viruses implicated in the aetiology of type 1 diabetes as discussed earlier. Previous studies on SARS viruses showed that some affected patients developed acute onset diabetes [10]. In a series of 52 patients suffering from COVID-19 pneumonia from China, 9 had mild pancreatic injury. Out of these, 6 had varying degrees of hyperglycemia. The authors stated that this could be due to direct pancreatic injury by SARS-CoV-2 or due to 'harmful immune response' [21]. Pancreatic necrosis ascribed to COVID-19 in a patient with diabetes has been reported from India from our group [22]. It is possible that the 'cytokine storm' as seen in some patients with severe COVID-19 may also cause beta cell apoptosis. Some anecdotal reports have described acute hyperglycemia and diabetic ketoacidosis caused by COVID-19 infection, including two cases reported from India, which are also included in the data of current study(8). Interestingly, these cases were initially treated with insulin therapy, but shortly after resolution of ketoacidosis, were controlled on oral antihyperglycemic therapy alone [12]. However, in our study C-peptide levels were not lower in the NOD COVID group, even in those who were antibody positive for COVID-19 indicating that there was perhaps no major damage to beta cells due to COVID-19 infection. However, it is interesting to note that as compared to data from north India, patients from South India showed lower C-peptide levels. More specifically, in South Indian patients, C-peptide levels were signicantly lower in NOD COVID patients *vs* NOD patients (Table 2). The significance of these regional variation in C-peptide response is unclear. Overall, It could be reasonably stated that in our cohort, direct severe pancreatic damage due to SARS-CoV-2 was seen in only two patients, and even this seems to be transient.

The strengths of the study are that it includes a fairly large number of individuals with new onset diabetes from both North and South India. Secondly, we have compared patients diagnosed pre and during COVID-19 epidemic. We have also compared those who were COVID-19 antibody positive and negative. These findings are described for the first time from India. The limitation of the study is that our results represent the profile of patients seen at two large private diabetes centres in North and South India. One cannot rule out the possibility that patients belonging to low socio-economic stratum admitted with acute COVID-19 in Government hospitals in India may have a different clinical pattern. Finally, a detailed elucidation of the dietary and exercise patterns would have been more appropriate.

5. Conclusions

This study from two tertiary care diabetes centres in India shows that individuals diagnosed with diabetes during COVID-19 do not significantly differ from those diagnosed before COVID-19 in symptomatology and clinical characteristics. While individuals diagnosed during COVID-19 had worse indicators of glycemia, this could be due to lifestyle factors and delayed diagnosis rather than a direct effect of the virus. Indeed, it appears fortuitous that there is no evidence to show that the virus has caused beta cell damage or destruction.

Declaration of competing interest

The authors do not mention any conflict of interest regarding this article.

References

[1] Gupta R, Misra A. COVID19 in South Asians/Asian Indians: heterogeneity of data and implications for pathophysiology and research. Diabetes Res Clin Pract 2020 Jul;165:108267.
[2] Singh AK, Misra A. Impact of COVID-19 and comorbidities on health and economics: focus on developing countries and India. Diabetes Metab Syndr 2020;14(6):1625–30.
[3] Gupta A, Madhavan MV, Sehgal K, Nair N, Mahajan S, Sehrawat TS, et al. Extrapulmonary manifestations of COVID-19. Nat Med 2020;26(7):1017–32. 2020/07/01.
[4] Rubino F, Amiel SA, Zimmet P, Alberti G, Bornstein S, Eckel RH, et al. New-onset diabetes in covid-19. N Engl J Med 2020;383(8):789–90.
[5] Krishna SV, Sunil K, Prasad RD, Modi KD. Precipitation of new onset diabetes by H1N1 infection. Indian J Endocrinol Metab 2012;16(Suppl 2):S438–40.
[6] Sudulagunta SR, Sodalagunta MB, Kumbhat M, Bangalore Raja SK. New onset diabetes mellitus in dengue shock syndrome. J Assoc Phys India 2018;66(8):104.
[7] Ruiz PLD, Tapia G, Bakken IJ, Haberg SE, Hungnes O, Gulseth HL, et al. Pandemic influenza and subsequent risk of type 1 diabetes: a nationwide cohort study. Diabetologia 2018;61(9):1996–2004.
[8] Reddy PK, Kuchay MS, Mehta Y, Mishra SK. Diabetic ketoacidosis precipitated by COVID-19: a report of two cases and review of literature. Diabetes Metab Syndr 2020;14(5):1459–62.
[9] Unsworth R, Wallace S, Oliver NS, Yeung S, Kshirsagar A, Naidu H, et al. New-onset type 1 diabetes in children during COVID-19: multicenter regional findings in the U.K. Diabetes Care 2020;43(11):e170–1.
[10] Yang JK, Lin SS, Ji XJ, Guo LM. Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. Acta Diabetol 2010;47(3):193–9.
[11] https://diagnostics.roche.com/in/en_gb/products/params/elecsys-anti-sars-cov-2.html. [Accessed 29 November 2020].
[12] Kuchay MS, Reddy PK, Gagneja S, Mathew A, Mishra SK. Short term follow-up of patients presenting with acute onset diabetes and diabetic ketoacidosis during an episode of COVID-19. Diabetes Metab Syndr 2020;14(6):2039–41.
[13] Grover S, Sahoo S, Mehra A, Avasthi A, Tripathi A, Subramanyan A, et al. Psychological impact of COVID-19 lockdown: an online survey from India. Indian J Psychiatr 2020;62(4):354–62.
[14] Kelly SJ, Ismail M. Stress and type 2 diabetes: a review of how stress contributes to the development of type 2 diabetes. Annu Rev Publ Health 2015;36:441–62.
[15] Ghosh A, Arora B, Gupta R, Anoop S, Misra A. Effects of nationwide lockdown during COVID-19 epidemic on lifestyle and other medical issues of patients with type 2 diabetes in north India. Diabetes Metab Syndr 2020;14(5):917–20.
[16] Ghosal S, Arora B, Dutta K, Ghosh A, Sinha B, Misra A. Increase in risk for type 2 diabetes due to lockdown for COVID19 pandemic in apparently non-diabetic individuals in India: a cohort analysis. Sep-Oct 2020;14(5):949–52. https://doi.org/10.1016/j.dsx.2020.06.020. Epub 2020 Jun 19.
[17] Chopra S, Ranjan P, Singh V, Kumar S, Arora M, Hasan MS, et al. Impact of COVID-19 on lifestyle-related behaviours- a cross-sectional audit of responses from nine hundred and ninety-five participants from India. Diabetes Metab Syndr 2020;14(6):2021–30.
[18] Ruiz-Roso MB, Knott-Torcal C, Matilla-Escalante DC, Garcimartin A, Sampedro-Nunez MA, Davalos A, et al. COVID-19 lockdown and changes of the dietary pattern and physical activity habits in a cohort of patients with type 2 diabetes mellitus. Nutrients 2020;12(8).
[19] Robinson E, Boyland E, Chisholm A, Harrold J, Maloney NG, Marty L, et al. Obesity, eating behavior and physical activity during COVID-19 lockdown: a study of UK adults. Appetite 2021;156:104853.
[20] Sankar P, Ahmed WN, Mariam Koshy V, Jacob R, Sasidharan S. Effects of COVID-19 lockdown on type 2 diabetes, lifestyle and psychosocial health: a hospital-based cross-sectional survey from South India. Diabetes Metab Syndr

2020;14(6):1815–9.
[21] Wang F, Wang H, Fan J, Zhang Y, Zhao Q. Pancreatic injury patterns in patients with coronavirus disease 19 pneumonia. Gastroenterology 2020;159(1):367–70.
[22] Ghosh A, Gupta V, Misra A. COVID19 induced acute pancreatitis and pancreatic necrosis in a patient with type 2 diabetes. Diabetes Metab Syndr 2020;14(6):2097–8.