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POST-COVID NEW-ONSET DIABETES: AUTOIMMUNE OR STEROID-INDUCED? A 2-YEAR FOLLOW-UP STUDY

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Abstract
New-onset diabetes has been increasingly reported during and after COVID-19 illness, but its clinical meaning remains uncertain. Hyperglycemia after COVID-19 may represent previously undiagnosed type 2 diabetes, stress hyperglycemia, steroid-induced diabetes, autoimmune beta-cell dysfunction, or a mixed phenotype. This proposed 2-year follow-up study examines adults who develop diabetes after confirmed COVID-19 infection and evaluates whether their condition is more consistent with autoimmune diabetes or steroid-associated metabolic dysregulation. The study is designed as a prospective observational cohort in which participants are followed at 3, 6, 12, 18 and 24 months with glycemic assessment, treatment review, C-peptide estimation, diabetes autoantibody testing where feasible, steroid exposure history, body mass index, family history and clinical outcomes. The paper presents the rationale, objectives, methodology, analytical framework, ethical safeguards, expected findings and research contribution. The central argument is that post-COVID new-onset diabetes should not be treated as a single disease category; it requires structured phenotyping to distinguish persistent autoimmune diabetes from potentially reversible steroid-associated or stress-related hyperglycemia. Such classification can support safer treatment decisions, follow-up planning and long-term diabetes care in Indian clinical settings.

Keywords: post-COVID diabetes, new-onset diabetes, steroid-induced diabetes, autoimmune diabetes, COVID-19, C-peptide, diabetes autoantibodies, two-year follow-up, glycemic persistence, India

1. Introduction

Coronavirus disease 2019 (COVID-19) created major clinical challenges not only through acute respiratory illness but also through post-acute complications affecting metabolic, cardiovascular, neurological and endocrine systems. Diabetes mellitus emerged as one of the most important metabolic concerns during the pandemic. Patients with pre-existing diabetes were found to have worse COVID-19 outcomes, while clinicians also reported cases of newly detected hyperglycemia and new-onset diabetes during or after SARS-CoV-2 infection. This raised an important question: does COVID-19 directly trigger diabetes, or does it reveal underlying metabolic vulnerability that was already present?

The proposed topic, post-COVID new-onset diabetes as autoimmune or steroid-induced, addresses a clinically significant uncertainty. During COVID-19 treatment, many patients received systemic corticosteroids to control inflammation and hypoxia. Steroids are known to increase insulin resistance and can precipitate hyperglycemia, especially in individuals with obesity, family history, prediabetes or severe infection. At the same time, COVID-19-related inflammation, immune activation and possible pancreatic beta-cell involvement have been discussed as mechanisms that may contribute to new diabetes phenotypes. Differentiating these possibilities is important because steroid-associated hyperglycemia may improve after recovery and dose withdrawal, whereas autoimmune diabetes may require sustained insulin-based treatment and close monitoring.

A 2-year follow-up design is relevant because early post-COVID hyperglycemia may not indicate permanent diabetes. Some patients may return to normal glycemic status, some may remain diabetic, and others may progress from impaired glucose regulation to persistent diabetes. Short follow-up cannot adequately distinguish transient stress or steroid-related hyperglycemia from durable beta-cell dysfunction. Therefore, a structured longitudinal study can help determine whether post-COVID diabetes persists, remits, or changes phenotype over time.

The January 2022 publication context is important because the literature available until that time had identified the problem but had not fully resolved its mechanisms or long-term course. Early reports called for registries and

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prospective metabolic follow-up. This paper is therefore written as a sample academic study protocol suitable for that time period, using references available before January 2022 and avoiding claims based on later evidence.

1.1 Research Problem

The research problem is that post-COVID new-onset diabetes is frequently recorded as a single diagnostic label, even though it may arise through different mechanisms. In routine practice, many patients are diagnosed after steroid therapy, acute inflammatory stress or hospitalization, while others may show features suggestive of autoimmune beta-cell dysfunction. Without structured follow-up and phenotyping, clinicians may overestimate permanent diabetes in some patients or miss progressive autoimmune diabetes in others. There is a need for a 2-year follow-up framework that separates autoimmune, steroid-associated and mixed/indeterminate patterns using clinical history and biochemical markers.

1.2 Objectives of the Study

- To identify adults who develop new-onset diabetes after confirmed COVID-19 infection and classify their baseline clinical phenotype.
- To compare autoimmune markers, C-peptide status and steroid exposure among post-COVID new-onset diabetes cases.
- To assess glycemic persistence, remission or progression over a 2-year follow-up period.
- To evaluate the proportion of cases more consistent with autoimmune diabetes, steroid-associated diabetes or mixed/indeterminate diabetes.
- To examine treatment patterns, insulin requirement, oral drug use and patient follow-up adherence over 24 months.
- To develop a practical clinical framework for post-COVID diabetes monitoring in outpatient settings.

Table 1. Research objectives, analytical focus and supporting literature

Objective area	Analytical focus	Main supporting sources
Case identification	New diabetes after COVID-19 with no prior diagnosis	Rubino et al. (2020); Sathish et al. (2021)
Autoimmune phenotype	Autoantibodies, low C-peptide and insulin need	Rubino et al. (2020); Müller et al. (2021)
Steroid-associated phenotype	Steroid exposure, insulin resistance and remission pattern	Bornstein et al. (2020); Apicella et al. (2020)
Longitudinal outcome	Persistence, remission and glycemic trajectory	Montefusco et al. (2021); Khunti et al. (2021)
Clinical management	Monitoring, risk stratification and treatment adjustment	American Diabetes Association (2021); Bornstein et al. (2020)

2. Conceptual Background

2.1 Post-COVID New-Onset Diabetes

Post-COVID new-onset diabetes refers to diabetes first detected during or after COVID-19 in a person with no known previous diagnosis of diabetes. The condition may include truly new disease, previously undiagnosed diabetes discovered during acute care, stress hyperglycemia that later resolves, steroid-induced hyperglycemia, or immune-mediated diabetes. Because these possibilities overlap clinically, a single fasting glucose or HbA1c value at one time point may not be enough to explain the mechanism or prognosis.

Clinical classification requires careful documentation of pre-COVID glycemic status where available, date of COVID-19 diagnosis, severity of infection, hospitalization status, oxygen requirement, steroid dose and duration, family history, body mass index, symptoms of hyperglycemia, ketosis, C-peptide and autoantibody status. A structured approach can separate patients who need long-term diabetes care from those who need close observation after recovery from acute illness and steroid exposure.

2.2 Autoimmune Diabetes After COVID-19

Autoimmune diabetes occurs when immune-mediated beta-cell injury reduces insulin production. In adults, this may present as type 1 diabetes or latent autoimmune diabetes in adults. After COVID-19, autoimmune mechanisms have

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been proposed because viral infection and immune activation can theoretically trigger or accelerate beta-cell autoimmunity in susceptible individuals. Features supporting autoimmune diabetes include positive GAD, IA-2 or ZnT8 antibodies, low or declining C-peptide, ketosis, weight loss and early insulin requirement.

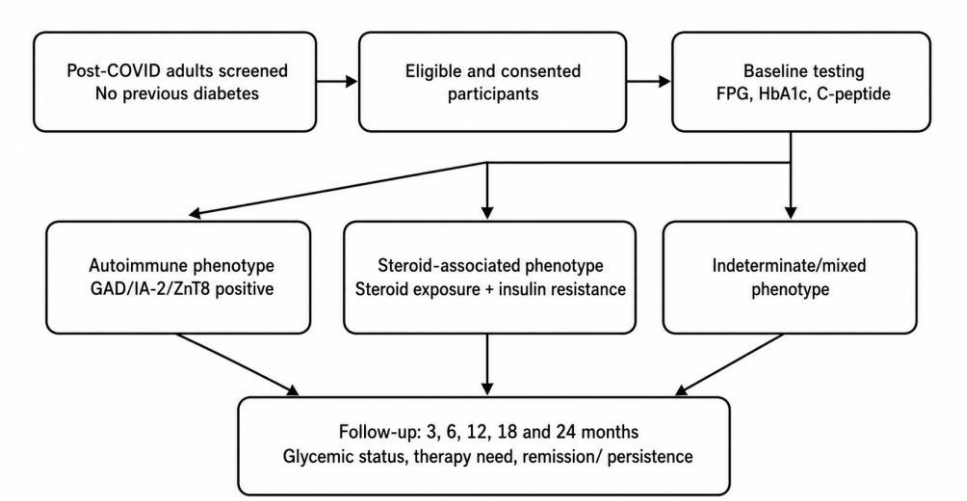
However, autoimmune attribution must be made cautiously. A temporal association with COVID-19 does not automatically prove causation. Autoimmune diabetes may have been developing before infection and may become clinically visible during acute illness. Therefore, this study treats autoimmune diabetes as a phenotype to be identified through markers and longitudinal progression, not as an assumption.

2.3 Steroid-Induced and Stress-Related Hyperglycemia

Steroid-induced diabetes or hyperglycemia occurs when glucocorticoid therapy increases hepatic glucose production and peripheral insulin resistance. In COVID-19 care, corticosteroids were widely used for selected patients with hypoxia or severe inflammatory illness. Patients receiving steroids may develop high postprandial glucose, insulin requirement during hospitalization, or temporary hyperglycemia that improves after steroid withdrawal.

Stress hyperglycemia is also common during severe infection due to inflammatory cytokines, counter-regulatory hormones and acute illness physiology. The challenge is that steroid-induced and stress-related hyperglycemia can unmask underlying diabetes risk. A 2-year follow-up allows researchers to determine whether glycemia normalizes after recovery or remains persistently abnormal, suggesting underlying diabetes vulnerability.

Figure 1. Proposed participant flow for the 2-year follow-up study



3. Review of Literature

The available literature before January 2022 showed a complex relationship between COVID-19 and diabetes. Early commentaries and registry proposals emphasized that new-onset diabetes was being observed in patients with COVID-19 and that long-term follow-up was required to define its phenotype and prognosis. Researchers suggested that SARS-CoV-2 infection might affect glucose metabolism through stress responses, inflammation, insulin resistance, beta-cell dysfunction or indirect effects of treatment. However, they also warned that many cases may represent previously undiagnosed diabetes discovered during hospitalization.

Clinical literature also highlighted the important role of steroid therapy. Corticosteroids were recommended for appropriately selected hospitalized COVID-19 patients requiring oxygen or ventilatory support, but they are also known to worsen hyperglycemia. This created a difficult clinical interpretation problem: when diabetes is diagnosed after COVID-19, it may be difficult to distinguish steroid-related hyperglycemia from persistent diabetes without follow-up. Therefore, studies focusing on duration of steroid exposure, glucose trajectory and remission are necessary.

Mechanistic studies available before 2022 suggested that pancreatic endocrine and exocrine tissues may be relevant to SARS-CoV-2 research. Some studies discussed possible infection of pancreatic cells, inflammatory damage or indirect metabolic stress. At the same time, other authors emphasized that insulin resistance and systemic

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inflammation may be more important than direct beta-cell destruction in many cases. This uncertainty supports a study design that includes both autoimmune markers and insulin-resistance-related clinical variables.

Table 2. Thematic synthesis of selected literature available before January 2022

Theme	Representative insight	Relevance to proposed study	Key sources
COVID-19 and diabetes	Bidirectional relationship; diabetes worsens COVID-19 outcomes and COVID-19 may disturb glucose metabolism.	Supports post-COVID metabolic follow-up.	Apicella et al. (2020); Bornstein et al. (2020)
New-onset diabetes	New diabetes after COVID-19 requires registry-based and longitudinal investigation.	Justifies 2-year design and careful phenotype definition.	Rubino et al. (2020); Sathish et al. (2021)
Steroid-related hyperglycemia	Glucocorticoids can increase insulin resistance and precipitate hyperglycemia.	Supports steroid exposure as a key explanatory variable.	Bornstein et al. (2020); Rayman et al. (2020)
Beta-cell dysfunction	Possible pancreatic and metabolic disruption has been proposed in COVID-19.	Supports C-peptide and autoimmune testing.	Müller et al. (2021); Montefusco et al. (2021)
Follow-up need	Long-term outcomes of newly detected diabetes remain uncertain.	Supports repeated assessments over 24 months.	Khunti et al. (2021); Sathish et al. (2021)

3.1 Research Gap

Existing studies before January 2022 recognized the association between COVID-19 and new-onset diabetes, but there remained limited clarity regarding whether post-COVID diabetes is autoimmune, steroid-induced, stress-related, previously undiagnosed, or mixed. Many reports were based on acute hospitalization data and did not include 2-year follow-up. There was also limited practical guidance on combining steroid history, C-peptide, autoantibody markers and glycemic trajectory into a clinically useful classification system. This proposed study addresses that gap by designing a structured follow-up model for post-COVID new-onset diabetes.

4. Research Methodology

4.1 Research Design

The study is designed as a prospective observational cohort study with 2-year follow-up. Adults with confirmed COVID-19 infection and newly diagnosed diabetes after infection will be enrolled after informed consent. Participants will not be randomized because the study aims to observe natural clinical phenotypes and treatment trajectories rather than assign treatment. The follow-up schedule will include baseline, 3 months, 6 months, 12 months, 18 months and 24 months.

4.2 Study Setting and Population

The proposed setting includes outpatient departments, post-COVID clinics, diabetes clinics and collaborating hospitals. The study population will include adults with no known history of diabetes before COVID-19 who meet diagnostic criteria for diabetes during or after COVID-19. The study may include both hospitalized and non-hospitalized patients, provided clinical records are adequate to document COVID-19 timing, steroid exposure and baseline metabolic status where available.

4.3 Inclusion and Exclusion Criteria

Table 3. Eligibility criteria for participant selection

Inclusion criteria	Exclusion criteria
Adults aged 18 years and above with confirmed COVID-19 infection.	Known diabetes diagnosed before COVID-19 infection.
New diabetes diagnosed during or after COVID-19 based on accepted glucose/HbA1c criteria.	Pregnancy-related diabetes or gestational diabetes.

Available information on steroid exposure, COVID-19 severity and treatment history.	Use of long-term steroids for non-COVID chronic disease before infection.
Willingness to attend follow-up visits for up to 24 months.	Severe illness preventing follow-up or inability to provide informed consent.
No documented pre-COVID diabetes; prior normal glucose/HbA1c where available is preferred.	Pancreatic surgery, chronic pancreatitis, or known endocrine disorder causing diabetes.

4.4 Phenotype Classification

Participants will be classified into provisional phenotypes based on baseline and follow-up data. An autoimmune phenotype will be suspected when diabetes autoantibodies are positive, C-peptide is low or declining, ketosis is present, or insulin requirement is early and persistent. A steroid-associated phenotype will be suspected when there is significant corticosteroid exposure, absence of autoantibodies, preserved C-peptide, overweight or insulin resistance features, and partial or complete glycemic remission after steroid withdrawal. A mixed or indeterminate phenotype will be used when findings overlap or data remain insufficient.

4.5 Variables and Outcome Measures

Table 4. Variables and outcome measures

Outcome type	Measure	Assessment method
Primary outcome	Persistence of diabetes at 24 months.	Fasting glucose, HbA1c, treatment status and physician diagnosis.
Phenotype outcome	Autoimmune, steroid-associated or mixed/indeterminate classification.	Autoantibodies, C-peptide, steroid history and clinical course.
Secondary outcome	Glycemic remission or improvement.	HbA1c normalization or withdrawal/reduction of diabetes medicines under medical care.
Treatment outcome	Insulin requirement and oral drug use.	Prescription review at each follow-up.
Safety outcome	Ketosis, hypoglycemia, hospital visits or uncontrolled hyperglycemia.	Clinical notes and adverse-event checklist.
Follow-up outcome	Retention and completion of 2-year assessments.	Visit records and contact logs.

4.6 Data Collection Procedure

Baseline data will include age, sex, residence, body mass index, family history of diabetes, COVID-19 severity, hospitalization status, oxygen requirement, steroid dose and duration, glucose values during acute illness, HbA1c at diagnosis, symptoms of diabetes and treatment started. Laboratory evaluation may include fasting plasma glucose, postprandial glucose, HbA1c, fasting C-peptide, lipid profile and diabetes autoantibodies such as GAD, IA-2 and ZnT8 where feasible. Follow-up visits will record glycemic status, therapy changes, insulin requirement, complications, remission, recurrence and adherence to follow-up.

4.7 Statistical Analysis Plan

Descriptive statistics will summarize baseline characteristics and phenotype groups. Continuous variables such as age, HbA1c and C-peptide will be presented as mean with standard deviation or median with interquartile range, depending on distribution. Categorical variables such as steroid exposure, autoantibody positivity, insulin requirement and diabetes persistence will be presented as frequencies and percentages. Comparisons between phenotype groups may use t-tests, Mann-Whitney U tests, chi-square tests or Fisher exact tests as appropriate. Logistic regression may be used to identify predictors of persistent diabetes at 24 months, including age, BMI, COVID-19 severity, steroid exposure, baseline HbA1c, C-peptide and autoantibody status.

4.8 Ethical Considerations

The study must follow ethical principles for human participant research. Written informed consent will be obtained before enrollment. Participants will not be denied clinically indicated diabetes care, and treatment decisions will remain under the supervision of qualified physicians. Blood samples for C-peptide and autoantibody testing will be collected only after consent. Confidentiality of medical records will be maintained, and participants will be free to withdraw at any stage without affecting their treatment. Any patient with severe hyperglycemia, ketosis or

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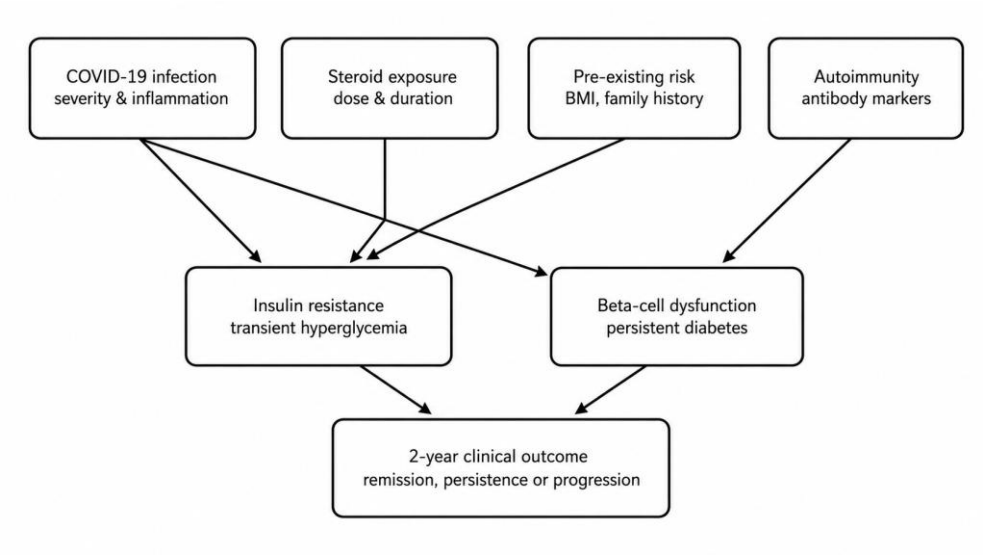
hypoglycemia will receive immediate clinical attention.

Figure 2. Analytical framework for autoimmune versus steroid-associated pathways



5. Follow-up Protocol

The follow-up protocol will be designed to capture both short-term glycemic change and long-term persistence. At 3 months, the main focus will be early stabilization after recovery from acute COVID-19 and steroid withdrawal. At 6 months, the study will assess whether glucose levels remain abnormal, improve or require ongoing medication. At 12 months, participants will be evaluated for phenotype stability, insulin need and possible remission. At 18 and 24 months, the study will determine whether diabetes has persisted, progressed, relapsed or resolved.

Table 5. Proposed follow-up schedule and clinical purpose

Visit	Key assessment	Clinical purpose
Baseline	COVID history, steroid exposure, HbA1c, FPG, C-peptide and autoantibodies where feasible.	Initial phenotype classification.
3 months	Glycemic review, treatment status and symptoms.	Differentiate transient hyperglycemia from continuing diabetes.
6 months	Repeat HbA1c, medicine review and insulin requirement.	Assess early remission or persistence.
12 months	Phenotype reassessment and complications screen.	Evaluate medium-term metabolic course.
18 months	Treatment changes, recurrence and adherence.	Identify delayed progression or improvement.
24 months	Final glycemic status and phenotype conclusion.	Classify remission, persistence or progression.

6. Analytical Indicators for Study Assessment

Although real conclusions can be drawn only after data collection, the following indicators can help organize analysis and interpretation. These indicators should be calculated from valid collected data only and should not be treated as assumed findings.

Diabetes Persistence Rate (%) = (Number of participants meeting diabetes criteria at 24 months / Total participants analysed) x 100

Autoimmune Phenotype Rate (%) = (Number of participants classified as autoimmune phenotype / Total participants analysed) x 100

Steroid-Associated Remission Rate (%) = (Number of steroid-exposed participants achieving remission / Total

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steroid-exposed participants analysed) x 100

Insulin Requirement Rate (%) = (Number of participants requiring insulin beyond 6 months / Total participants analysed) x 100

Table 6. Formula-based interpretation for post-COVID diabetes assessment

Indicator	Question answered	Clinical implication
Diabetes persistence rate	How many participants remain diabetic at 24 months?	Higher rate suggests durable metabolic disease rather than transient stress hyperglycemia.
Autoimmune phenotype rate	How many show markers of autoimmune beta-cell dysfunction?	Supports need for insulin planning and close monitoring.
Steroid-associated remission rate	How often does steroid-associated diabetes improve after recovery?	Higher remission supports reversible insulin-resistance pathway.
Insulin requirement rate	How many require sustained insulin therapy?	May identify more severe or autoimmune phenotype.
Follow-up completion rate	Were participants retained for full follow-up?	Low completion affects validity and feasibility.

7. Expected Findings and Interpretation Plan

As this document is a sample manuscript/protocol for academic formatting, no real findings are reported. After data collection, the study may show that post-COVID new-onset diabetes is not a uniform condition. Some participants may demonstrate steroid-associated or stress-related hyperglycemia with improvement over time, while others may show persistent diabetes requiring long-term therapy. A smaller subgroup may have autoimmune features such as autoantibody positivity and low C-peptide, requiring earlier insulin-based management.

If most steroid-exposed patients show glycemic improvement after steroid withdrawal and recovery, the findings may support a cautious classification of steroid-associated diabetes in selected cases. If autoantibody-positive patients show persistent hyperglycemia and insulin dependence, the findings would support the need for autoimmune screening in clinically suspicious cases. If a large proportion remains mixed or indeterminate, the study would highlight the complexity of post-COVID metabolic disease and the need for larger multicentre studies.

The interpretation must consider that COVID-19 may unmask pre-existing risk rather than directly cause diabetes. Therefore, conclusions should be based on clinical trajectory, laboratory markers and baseline risk factors rather than timing alone. The study should avoid overclaiming causation and should emphasize structured follow-up as the most practical clinical contribution.

8. Discussion

The proposed 2-year follow-up study is important because it addresses a common post-pandemic clinical problem: many patients developed diabetes or hyperglycemia during and after COVID-19, but the mechanism and prognosis were unclear. Some cases may be related to steroid therapy, some may reflect stress physiology, and some may represent autoimmune or progressive metabolic disease. Without follow-up, these groups may be treated in the same way even though their long-term needs differ.

A major strength of the proposed study is its clinical practicality. It uses information that can be collected in outpatient and hospital settings, including steroid history, HbA1c, C-peptide, autoantibodies where available, and repeated follow-up visits. The study design recognizes that not all centers may have advanced testing for every participant, so classification includes an indeterminate group to avoid forced or inaccurate labeling.

The study also has limitations. Autoantibody testing may not be affordable for all participants. Pre-COVID HbA1c values may be unavailable, making it difficult to exclude undiagnosed diabetes in every case. The observational design cannot prove causation between COVID-19 and diabetes. Loss to follow-up over 2 years may affect results. Despite these limitations, the proposed study provides a systematic approach to a clinically important question and can guide future larger studies.

9. Findings / Expected Contributions

- The study will help clarify whether post-COVID new-onset diabetes is commonly persistent or transient over 2 years.
- It will distinguish probable autoimmune, steroid-associated and mixed/indeterminate phenotypes using structured criteria.
- It will document the role of steroid exposure, COVID-19 severity and baseline metabolic risk in diabetes persistence.
- It will identify the proportion of patients requiring sustained insulin or long-term oral antidiabetic therapy.
- It will support practical outpatient follow-up protocols for post-COVID metabolic care.
- It will provide a foundation for larger multicentre studies in Indian populations.

10. Recommendations

- Patients with newly detected hyperglycemia after COVID-19 should receive follow-up testing rather than a one-time diagnosis only.
- Clinicians should document steroid dose, duration and timing when evaluating post-COVID diabetes.
- C-peptide and diabetes autoantibodies should be considered when patients are lean, ketotic, rapidly insulin-requiring or clinically suggestive of autoimmune diabetes.
- Patients with steroid-associated hyperglycemia should be monitored after steroid withdrawal to identify remission or persistence.
- Post-COVID clinics should integrate metabolic screening for high-risk individuals such as those with obesity, family history or severe COVID-19.
- Future research should use longer follow-up, larger samples, standardized phenotype definitions and multicentre Indian data.

11. Conclusion

Post-COVID new-onset diabetes is a clinically important and scientifically complex condition. It may represent autoimmune diabetes, steroid-induced diabetes, stress-related hyperglycemia, previously undiagnosed type 2 diabetes or a mixed phenotype. A 2-year follow-up study provides an appropriate framework for examining persistence, remission and progression over time. The proposed study emphasizes that accurate classification requires more than the timing of diagnosis; it requires steroid history, metabolic risk assessment, C-peptide, autoantibodies where feasible, treatment pattern and longitudinal glycemic trajectory.

If implemented with ethical approval and valid data collection, this study can contribute to better post-COVID diabetes care by helping clinicians identify which patients need long-term treatment, which require autoimmune evaluation and which may improve after recovery from acute illness and steroid exposure. Ultimately, the study promotes patient-centred, evidence-informed and follow-up-based management of diabetes diagnosed after COVID-19.

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