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Original Research Paper

Medicine

A STUDY OF NEW ONSET HYPERGLYCEMIA IN COVID-19 INFECTION

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ABSTRACT Covid-19 has proven to be metabolic disease resulting in adverse outcome in individuals. Emerging data increasingly suggests that "new-onset" hyperglycaemia is a frequently observed finding especially in admitted patients with Covid-19, who had no history of dysglycemia or diabetes in the past and were currently not on corticosteroids. This entity of "new-onset" hyperglycaemia could be classified as:

1) "stress-induced" hyperglycaemia,
2) "New-onset diabetes" in previously unrecognised pre-diabetes,
3) Hyperglycaemia possibly related to SARS-CoV-2 direct effect on pancreas,
4) drug-induced hyperglycaemia or "secondary diabetes" during course of treatment for Covid- 19, especially with frequent use of corticosteroids.

KEYWORDS :

Aims And Objectives

1) To study relationship between new onset diabetes mellitus / hyperglycaemia and Covid-19 infection.
2) To study mechanisms of onset of hyperglycaemia in Covid-19

Pathophysiology

- The angiotensin converting enzyme (ACE) receptor, which acts as the portal of entry for Covid-19, has been identified not only on respiratory epithelial cell, but also in kidney, GI tract and pancreas.
- Virus infect and replicate in cells of human endocrine and exocrine pancreas with subsequent cell destruction has been postulated to underlie the development of new-onset, insulin requiring diabetes/ hyperglycaemia in some patients with covid-19.
- Virus mediated damage to beta cells releases sequestrated antigens that lead to activation of auto reactive T-lymphocytes, culminating in an autoimmune response that ultimately destroys the reminder of the Beta-cell mass, leading to insulin-dependent type-1 diabetes.

Diabetogenic Effects of Host Responses to Covid-19 Infection

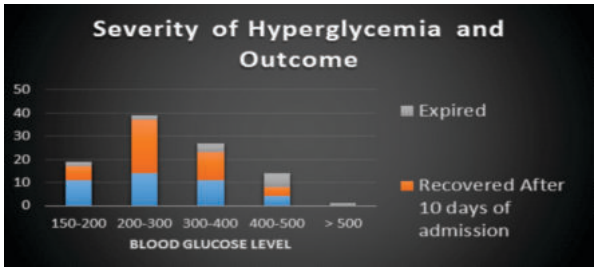
- C-reactive protein (CRP) and inflammatory cytokines are significantly elevated in Covid-19 patients. Cytokine storm has been associated with multi-organ failure in Covid-19 patients with severe illness. Outpouring of counter regulatory hormones inflammatory markers like TNF-α, IL-6 both of which are known to induce insulin resistance and hyperglycaemia.
- Proteins/cytokines secreted from adipose tissue into the bloodstream, collectively called adipokines have been shown to regulate beta cell function. Adipokine was found to be decreased by 50-60% in serum of patients suffering from severe Covid-19.Sudden reduction in insulin sensitivity can precipitate diabetes in individuals with borderline Beta-cell function and may even manifest as hyperglycaemic crises in those with previously undiagnosed (and untreated) diabetes.

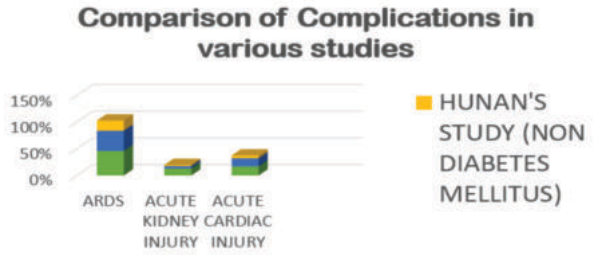
Stress Induced Hyperglycaemia

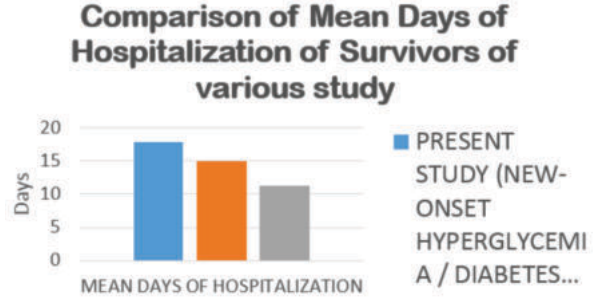
- The release of stress hormones including catecholamine, cortisol, glucagon and growth hormone in response to critical illness motivate gluconeogenesis and glycogenolysis in the liver which increase blood glucose levels and induce insulin resistance by inhibiting tyrosine kinase activity, insulin binding and glucose uptake (inhibit GLUT-4).

Iatrogenic Hyperglycaemia

- Corticosteroids such as dexamethasone have become mainstay of management of severe Covid-19 infection but they are also known to be highly diabetogenic drugs.







DISCUSSION

- Age group most commonly affected is 41-60 years followed by 61-80 years.
- Most commonly affects the male patients.
- Patients who were having RBS >300 mg/dl had higher mortality as compared to those with RBS <300 mg/dl.
- Patients who were having RBS between 150-300 mg/dl had highest recovery rate.
- Most common complication is ARDS.
- Mean days of hospitalization were higher in new onset hyperglycaemia in compare to pre-existing diabetes mellitus and normoglycemia.

CONCLUSION

- In our study of 100 patients, we conclude that new-onset hyperglycaemia or diabetes mellitus is common in middle aged men with more hospitalization stay and most commonly complicated with acute respiratory distress syndrome in COVID-19 infection.

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