

DIABETES MELLITUS AND RENAL FAILURE EFFECT ON INTESTINAL INSULIN

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ABSTRACT

The two most prevalent causes of end-stage renal disease nowadays are thought to be diabetic mellitus (DM) and hypertension (ESRD). In addition to discussing the function of DM in ESRD, this study reviews glucose metabolism and the treatment of hyperglycemia in these patients. Although strict glycemetic control and ESRD patient survival were not significantly correlated in numerous big trials, it is advised that glycemetic control be prioritised as the primary therapeutic objective in the care of these patients to minimise harm to other organs. When fasting blood sugar is less than 140 mg/dL, 1-hour postprandial blood sugar is less than 200 mg/dL, and glycosylated haemoglobin (HbA1c) is 6-7 in type 1 diabetes patients and 7-8 in type 2 diabetes patients, glycemetic control is ideal. Given its potentially deadly side effect, lactic acidosis, metformin administration should be avoided in patients with chronic renal failure (CRF), while glipizide and repaglinide seem to be preferable options.

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INTRODUCTION

Diseases can cause Stress so Covid-19 and Anaemia also cause stress [1,2,3,4,5] As it is viral disease and viral disease don't have any treatment[6-9] Bacterial disease have treatment as we can use antibiotics[10,11] But In this paper our focus is on Diabetes mellitus and renal failure effect on intestinal insulin End-stage renal disease (ESRD) is a disorder when the kidneys are unable to function normally in response to daily demands. One of the most serious and life-threatening disorders, ESRD often follows chronic renal disease. It places a heavy psychological and financial cost on civilizations. [In both industrialised and developing nations, diabetes and hypertension are now the leading causes of ESRD. [12,13,14] In a research done in the USA, diabetes and hypertension were shown to be the two main causes of end-stage renal disease (ESRD)[15], while in a study done in Khuzestan, Iran, diabetes was found to be the main cause of illness and glomerulonephritis to be the cause of roughly 10% of cases. [15] Diabetes mellitus and renal failure's effects on one another Renal failure is a complication of DM, a metabolic disorder, and in diabetic patients, renal failure increases the need for insulin.[17,18,19] In individuals with chronic renal failure (CRF), the buildup of uremic toxins and elevated parathyroid hormone levels result in insulin resistance in tissues, notably skeletal muscle tissues. This has been ascribed to damage that occurs as a result of insulin attaching to its receptors, which disrupts the formation of glycogen and the metabolism of glucose. [20-24] Additionally, it seems that the anaemia brought on by CRF affects insulin resistance, and erythropoietin treatment of anaemia has been proven to improve insulin sensitivity in the body. [16-18].

LITERATURE REVIEW

Oxidative Stress and the Rise in Diabetes Mellitus and Renal Failure

Reactive oxygen species (ROS) are overproduced under stressful situations, resulting in oxidative stress. Therefore, oxidative stress results from an imbalance between the production of free radicals and the ability of antioxidant defences. [25] As a consequence of this oxidative stress, chronic, difficult-to-treat illnesses like diabetes,[26,27,28] hypertension,[29] cardiovascular disorders,[25,26] cancer,[27-30] cognitive problems, and pain may develop, or other diseases like infectious diseases may get worse. Additionally, oxidative stress is a major factor in the progression of diabetic kidney disease. Diabetes-related kidney damage is becoming more common everywhere. Renal failure has been attributed mostly to an increase in ROS, which causes oxidative stress. In addition to diabetes, renal failure itself raises oxidative stress levels.[30,31] Numerous macromolecules, including specific defects in the polyol pathway, glycolysis, advanced glycation, xanthine oxidase, reduced nicotinamide adenine dinucleotide phosphate [NAD(P) H] oxidase, and uncoupling of nitric oxide synthase [NAD(P) H] oxidase, have been shown to be associated with the increased generation of ROS[32,33,34,35].

Antioxidants' Function in the Management of Diabetes and Kidney Disease

A possible treatment strategy to reduce diabetes and/or kidney impairment would be to buffer the formation of ROS or consume these substances [36-39]. Many disorders caused by ROS have been proven to benefit from antioxidant treatment. Although there are a number of preclinical publications demonstrating the efficiency of antioxidants in the prevention and treatment[40-42] of both diabetes and renal failure, the function of antioxidant therapy in diabetes and/or renal failure in people remains unclear. Many plants have antioxidant activity, and it has been shown that antioxidants having plant origins are a preferable option for this use. In this sense, it is preferable that we attempt employing plants that have had success managing both diabetes mellitus and renal illness. [43-44].

METHOD

New Methods for Renal Failure Control

Diabetes management and early diabetes risk factor therapy are crucial for avoiding or postponing kidney disease. It has been shown that both the incidence and the course of diabetic kidney disease may be reduced by controlling hypertension using drugs that modify the renin-angiotensin system (RAS). Angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor antagonists are used by individuals with type II diabetes to lower the incidence of ESRD. [45] The effects of ACEIs and angiotensin II receptor antagonists are peculiarly unrelated to any antihypertensive properties, which raises the possibility of a direct renal action.

RESULT

Control of Blood Glucose is Important in Patients with Renal Failure

Large studies revealed no significant association between tight glycemic control and survival of dialysis patients, despite the relevance of tight glycemic control having been emphasised in multiple small studies[24]. Additionally, strict blood glucose management may raise the chance of hypoglycemia crises.

DISCUSSION

According to research, strict blood glucose monitoring is less crucial for diabetic dialysis patients than it is for those who do not have renal failure for the following reasons:

1. Patients on dialysis who have tight glycemic control are more likely to have hypoglycemia, particularly those who have decreased appetite;
2. Dialysis patients have less hyperglycemic symptoms than individuals with normal renal function. Despite these facts, some scientists believe that if blood sugar levels do not remain below a reasonable range, damage to organs like the heart and eyes would worsen.

CONCLUSION

Intestinal Insulin

As insulin's renal and hepatic metabolism is slowed in CRF, its recommended dosage should be adjusted as follows: There is no need for dosage decrease when the GFR is more than 50 mL/min. The insulin dosage should be lowered to around 75% when the GFR is 10–50 mL/min. GFR should be less than 10 to 50 mL/min for the dosage to be lowered by around half. These modifications are broad, and insulin dose modifications need to be based on routine blood glucose checks. It should be highlighted that reducing insulin resistance and increasing insulin degradation are two separate things that happen when uremia is corrected with dialysis; instead, glycemic control should be determined by the final result.

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