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The Development of Cellular Oxidative Injury due to Ongoing Diabetes Requirements Implant of the Pancreas

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Abstract

There is a lot of evidence that hyperglycaemia-induced cellular oxidative stress is a major factor in the onset and progression of chronic diabetic lesions. In this study, we determined whether pancreas transplantation prevented the imbalance between the lungs of diabetic rats' antioxidant defences and the excessive production of reactive oxygen species. After four and twelve weeks of follow-up, each group was further divided into two subgroups of ten rats each. These subgroups were then killed. All rats had their plasma glucose, glycosylated hemoglobin, and insulin levels measured. In the pulmonary tissue of all of the rats, the concentrations of lipid hydro peroxide as well as the activities of superoxide dismutase, catalase, and glutathione peroxidase enzymes were measured. The DC rodents showed raised blood glucose and glycosylated hemoglobin levels, with insulin blood levels altogether lower than the NC rodents. They likewise showed altogether expanded LPO focuses in the lungs following 4 and 12 weeks of follow-up. SOD, CAT, and GSH-Px antioxidant activities, on the other hand, decreased significantly during these times 12 weeks after diabetes induction. Effective PT adjusted all clinical and metabolic changes in the diabetic rodents, with supported normoglycemia all through the review. Four weeks after PT, low SOD, CAT, and GSH-Px antioxidant activities and excessive lung LPO production were back to normal. In diabetic rats, PT can reduce oxidative stress in the pulmonary tissue. It may serve as the foundation for preventing lungs with chronic diabetic lesions.

Keywords: Cellular oxidative injury; Diabetes; Pancreas implant; Enzymes

Introduction

Histopathologic findings in animal and human lungs strongly suggest that the respiratory system is also affected by diabetes mellitus. However, the causative mechanisms of pulmonary injury in diabetes are still widely discussed. Although hyperglycemia has been recognized as the causal link between diabetes and diabetic complications, there is much evidence to suggest that it probably represents only an initial step in a complex cascade of events that lead to chronic diabetic lesions [1].

Recent studies have shown that oxidative stress from increased generation of reactive oxygen species plays an important role in the genesis and progression of diabetic complications. A previous study in our laboratory showed that pancreatoduodenal was capable of controlling oxidative stress in kidneys of alloxan-induced diabetic rats. The present study we investigated whether PT could be a potential method for restoring a sustainable state of normoglycemia in diabetic rats as well as for controlling ROS generation in their lungs. We hoped to make a contribution to knowledge on the effective control of chronic diabetic complications [2].

Methods

Sixty inbred male Lewis rats were randomly assigned to experimental groups: NC, 20 non-diabetic control rats; DC, 20 untreated diabetic control rats; and PT, 20 diabetic rats that received syngeneic PT from normal donor Lewis rats. Each group was further divided into 2 subgroups of 10 rats each, which were killed after 4 and 12 weeks of follow-up. Diabetes was induced by intravenous administration of a single dose of 42 mg/kg body weight [3]. Only diabetic rats showing severe clinical and metabolic alterations were included in the experiment. Any animals which died during follow-up were replaced by standbys in their respective subgroups. Pancreas transplants were performed according to Lee's technique modified in our laboratory.

The animals were housed in metabolic cages and the following data recorded: body weight, water intake, food intake, and urine output. Laboratory parameters such as blood glucose, glycosylated hemoglobin, and plasma insulin were also determined in these animals. Diabetic animals were also categorized according to blood glucose and glycosylated hemoglobin rates [4]. At the follow-up times, pulmonary tissue of each killed animal from all groups was analyzed for oxidative stress markers: LPO concentration and SOD, CAT, and GSH-Px enzyme activities by using specific reagents.

Clinical and laboratory variables and biochemical markers of oxidative stress were analyzed by analysis of variance complemented with multiple-comparisons tests described by Tukey or Mann-Whitney and Kruskal-Wallis. All statistical analyses were based on a significance level of 5% Results. The NC rats had clinical and laboratory parameters compatible with the norms for their species and age throughout the study period. In contrast, the DC rats evolved with progressive weight loss and significant increases in water and food intake and diuresis [5]. They also presented elevated blood glucose and hemoglobin levels, and plasma insulin levels were significantly lower than in the NC rats

Discussion

Twenty-one animals from the initial lot died in the first 14 days after diabetes induction owing to metabolic disorders and drug toxic action. 15% died in late (up to 12 weeks) owing mainly to ketoacidosis and pulmonary infection In contrast, PT rats with technically successful transplantations presented with complete correction of polydypsia,

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polyuria, and polyphagia from the third day after surgery. Animals from the PT group also maintained sustainable glycemia and HbA1c, and normal plasmatic insulin levels throughout follow-up, with values similar to the NC animals [6, 7]. Three of the PT animals died in the immediate postoperative period owing to technical failure related to the transplantation surgery, including thrombosis from portacaval anastomosis or hemorrhage and graft ischemia. There were no deaths in the PT group during late follow-up. All laboratory data are shown in

The LPO concentration in pulmonary tissue of the DC rats was significantly higher than in the NC rats at 4 and 12 weeks of follow-up. There were significant reductions in SOD, CAT, and GSH-Px enzyme activity values in DC pulmonary tissue, which were statistically 12 weeks after diabetes induction compared with NC. Pulmonary oxidative stress was also strongly related to diabetes duration and was more intense in late follow-up 12 wk. However, oxidative stress in the DC rats had no relationship with glycemia or hemoglobin levels [8].

The PT rats presented complete correction of excessive pulmonary LPO generation with significant increases from low SOD, CAT, and GSH-Px enzyme activity levels. These changes were already backing to normal 4 weeks after PT. There were no statistically significant differences between NC and PT groups for all analyzed markers. Despite the severe effects of DM on all organs and systems in the organism, the intrinsic mechanisms involved in the development and progression of chronic diabetic lesions are still poorly understood [9, 10]. Evidence that elevated ROS generation and altered redox balance can be present during the glucose auto-oxidation process and protein glycation have corroborated the hypothesis that oxidative damage plays a key role in the genesis of diabetic complications.

Results

The results of the present study strongly suggest that oxidative stress is also seen in pulmonary tissue from alloxan-induced diabetic rats, demonstrated by elevated LPO concentrations, and reduced SOD, CAT, and GSH-Px activity. In this study pulmonary oxidative stress also had a strong relationship with diabetes duration; this was more intense after 12 weeks of diabetes. Otherwise, this metabolic imbalance had no linear correlation with blood glucose and HbA1c levels, suggesting that hyperglycemia duration may be more relevant in diabetes pathophysiology than in glucose or hemoglobin level severity. These findings corroborate our previous11 and other6 studies [11].

Nevertheless, it is controversial whether oxidative damage induces or promotes diabetic lesions, or whether it is simply just one step in a complex phenomenon with several other mechanisms. it is widely known that oxidative damage can play an important role in regulating cellular adhesion, proliferation, migration, and cell signaling of the extracellular can alter the structure and permeability of the cellular membrane and intracellular organelles such as mitochondrias and rough endoplasmic reticulum, affecting ionic turnover through the membrane, cellular oxidative process, and protein synthesis. ROS generation may also attack lysosomes and cellular DNA, making the cell more susceptible to damage from toxic products and mutations which can lead to cell death. Another harmful mechanism, observed in glucose-independent tissues (eg, nervous tissue), is activation of polio enzyme pathways, where Hyperglycemia may increase sorbitol levels in the cells, elevating their osmolality, which may cause cellular death [12, 13].

Hyperglycemia may also activate the hexosamine pathway, which is responsible for converting glucose into compounds derived from acetylglucosamine that modulate the expression of various proteins and substances, such as plasminogen activator inhibitor 1 and transforming growth factor β2 which may contribute to vascular thrombosis and excessive production of collagen matrix on the vascular endothelium. These mechanisms are directly implicated in the genesis and evolution of diabetic microangiopathy. Augmented production of protein kinase C formed from free fatty acid oxidation may reduce nitric oxide concentrations in diabetic tissue as well as elevate the amount of TGF-β2 and vascular endothelial growth factor A [14, 15]. These substances may primarily contribute to narrowing of capillary net, reduction of vascular blood flow, and increase of collagen matrix deposits on the vascular endothelium, elevating the risks of vascular thrombosis and occlusions. Based on these premises, no doubt remains that any treatment proposed to prevent, stabilize, or reverse chronic diabetic lesions must first include the effective control of Hyperglycemia, which seems to be the only way to control ROS and AGE generation as well as the mechanisms directly involved in the molecular pathways responsible for cellular damage.

Unfortunately, despite improvements in quality of life and prognosis of diabetic subjects treated with exogenous insulin, diet, and oral hypoglycaemic agents, there is no evidence that these treatments can avoid the development of chronic diabetic complications.

Conclusion

The present study also showed that PT was capable of achieving a permanent state of insulin independence and normoglycemia in diabetic animals as well as blocking the sequence of events responsible for pulmonary oxidative stress, as demonstrated by inhibiting ROS generation and restoring antioxidant activity of the lungs. These findings suggest that benefits achieved with normoglycemia through PT may be extended to the control of other cellular damage pathways in diabetes, offering a promising perspective for definitive control of pulmonary and other chronic diabetes complications. Further investigations are needed to better understand these questions.

Acknowledgement

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Conflict of Interest

None

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