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EVIDENCE OF OXIDATIVE IMBALANCE IN THE GASTRIC MUCOSA OF DIABETIC RATS

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INTRODUCTION

The term Diabetes mellitus (DM) comprises a group of metabolic diseases of various etiologies, characterized mainly by chronic hyperglycemia. The incidence of oxidative stress and damage to the gastric mucosa is increased in patients with diabetes, so this study aimed to characterize the changes in oxidative stress parameters caused by diabetes in the gastric mucosa of diabetic rats.

MATERIAL AND METHODS

In the present study, rats (200-250g) were diabetized with streptozotocin (STZ, 60 mg / kg, ip) and divided into 3 groups (n = 8): normoglycemic control, hyperglycemic negative control and positive insulin control (6 IU / day). The animals were treated for 26 days and were euthanized 24 hours after the last administration. After euthanasia, glutathione reductase (GSH), lipoperoxides (LOOH), superoxide dismutase (SOD), catalase (CAT) and glutathione S-transferase (GST) were quantified. The study was approved by the Animal Ethics Committee, under opinion 057/17.

RESULTS

A 30% reduction in body weight was observed in diabetic animals treated with hyperglycemia. Diabetic rats treated with insulin showed no weight loss and remained normoglycemic. Furthermore, it was possible to verify a 38% reduction in GSH levels and a 31% increase in LOOH levels in the gastric mucosa of diabetic animals treated with vehicle, but not with insulin. SOD activity levels were similar in all experimental groups. Unexpectedly, both the vehicle-treated diabetic group and the insulin-treated diabetic group had reduced CAT activity levels and increased GST levels.

CONCLUSION

Impairment of GSH levels and LOOH accumulation in the gastric mucosa are the most evident findings, which may expose the mucosa to oxidative damage and consequently to more susceptible ulcerative processes.

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