Role of Embelin on Lithium Induced Nephrogenic Diabetes Insipidus in Albino Rats

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Abstract

Objective - The aim of this study was to investigate nephroprotective effect of Embelin in lithium induced nephrogenic diabetic insipidus

Method – Nephrogenic diabetes insipidus were induced in rats by injection of Lithium (4meq/kg/day or 27.76mg/kg/day LP.), dissolved in normal saline for 6 day. Animals with profound polyuria daily urinary output 50-70% of the body weight were selected for study. daily urine output was assessed for confirmation of NDI.

Result – The result of this study that embelin show nephroprotective effect by lower plasma creatinine urinecreatinine ,urine sodium, potassium, magnesium, & nitric oxide level of kidney and also increase glutathione level of kidney

Conclusion – The renoprotective effect action of embelin due to free radical scavenging effect & vasodilatory action. Thus embelin prevent lithium induced oxidative stress and renal ischemia.

Keywords- Nephrogenic diabetic insipidus, lithium

Conflict of Interest: None Declared!
INTRODUCTION

Diabetes insipidus (Latin=without taste) is a syndrome manifested by voluminous urine output, low urine-specific gravity, high plasma osmolarity, and high plasma sodium. Diabetes insipidus occurs when the kidney is unable to control plasma osmolality due to a defect in the action of arginine vasopressin (AVP).  

Nephrogenic diabetes insipidus (NDI) is characterized by a decrease in the ability to concentrate urine due to a resistance to ADH action in the kidney. NDI can be observed in chronic renal insufficiency, lithium toxicity, hypercalemia, hypokalamia, and tubulointerstitial disease, rarely, diabetes insipidus may be hereditary. 

Diabetic nephropathy (DN) is clinically defined as the progressive development of renal insufficiency in the setting of hyperglycaemia. Diabetic nephropathy (DN) is one of the most frequent life threatening complications of diabetes mellitus that occurs approximately 30-40%. This disease is now the major single cause of end-stage renal failure in many countries. 

Diabetic nephropathies (DN) is one of the important microvascular complications of diabetes mellitus. It is usually attributed to metabolic consequences of abnormal glucose regulations, such as elevated blood and tissue levels of glycosylated proteins and haemodynamic changes within the kidney tissue. Recently there has been a renewed interest in understanding the role of reactive oxygen species (ROS), which play a key intermediate role in the pathophysiology of DN. Chronic hyperglycemia the main determinant of the initiation and progression DN, not only generates more reactive oxygen metabolites but also attenuates anti oxidative mechanisms through non-enzymaticglycation of the scavenging enzymes. 

Diabetic nephropathy, a major chronic complication of diabetes mellitus, is a leading cause of end-stage renal disease, and hyperglycemia is considered a major risk factor for the development of nephropathy. Diabetes control and complication trial studies have shown that improved metabolic control is associated with decrease development and progression of nephropathy in diabetes.

Diabetic nephropathy refers to damage to or disease of kidney. Diabetes is the most cause of kidney failure, accounting for more than 40% of new cases. Even when drug and diet are able to control diabetes, the diseas can lead to nephropathy and kidney failure. Most people with diabetes do not develop nephropathy that is severe enough to cause kidney failure. About 16 million people in US have diabetes, and about 100000 people have kidney failure as a result of diabetes. Diabetic sugar level damage the kidney blood vessel. 

MATERIALS AND METHOD:

Procurement and selection of animals. Male albino rats (100-200 gm) wister strain were procured from Animal House. A suspension of embelin (20 mg/ml) was prepared using 1% tween 80 as suspending agent. Nephrogenic diabetes insipidus were induced in rats by injection of lithium (4meq /kg/day or 27.76 mg /kg /day I.P.) dissolved in normal saline for 6 day. Animals with profound polyuria daily urinary output 50-70% of the body weight were selected for study. Daily urine output was assessed for confirmation of NDI.

Experimental Procedure-Rats were divided into five groups and each group having six animals and animals will be divided into following groups. group 1: Normal control, receive normal saline (i.p.) group 2: Negative control, receive lithium chloride (i.p.) group 3: Disease treated with Embelin 50mg/kg (p.o.) group 4: Disease treated with Embeline 100mh/kg (p.o.) group 5: Positive control (N-acetylce cysteine10 mg/kg twice daily i.e. at 6 am and at 6 pm, i.p.) as a standard.

Biochemical parameter were assessed at 0th, 7th, 14th, 21th days.

RESULT

Change in body weight, urine volume, plasma creatinine, urine creatinine, blood urea nitrogen, urine protien and quantity of reduced glutathione content of various groups of kidney of rats treated with lithium. The result of this study that embelin show nephroprotective effect by lower plasma creatinine, urincreatineine, urine sodium,potassium,magnesium,&nitric oxide level of kidney and also increase glutathione level of kidney.
**DISCUSSION**

Lithium cause acute reduction in renal function characterized by polyuria, polydipsia, & electrolyte imbalance & produce oxidative stress and decrease glutathione level.\(^{10}\) The renoprotective effect action of embelin due to free radical scavenging effect & vasodilatory action. Thus embelin prevent lithium induced oxidative stress and renal ischemia.\(^{11}\) Nephrogenic diabetes insipidus (NDI) is characterized by a decrease in the ability to concentrate urine.\(^{12}\) Our study was focused to investigate nephroproprotective effect of Embelin in lithium induced nephrogenic diabetic insipidus in rats. Lithium (4meq/kg/day; i.p.), was injected for 6 day to induced Nephrogenic diabetes insipidus. Plasma creatinine, urine creatinine, BUN, urine protein and glutathione were assessed as nephrogenic parameters. Embelin shows significant effects as compared to diseased control.\(^{13}\) The renoprotective effect of embelin may be due to free radical scavenging effect & vasodilatory action. From our study we concluded that embelin prevent lithium induced oxidative stress and renal ischemia and can serve as a nephroprotective agent.\(^{14}\)

**REFERENCES**