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PHYTOCHEMICAL ESTIMATION OF ANACYCLUS PYRETHRUM AGAINST STREPTOZOCIN INDUCED DIABETIC NEUROPATHY

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ABSTRACT

The present drugs can treat most of the diseases which are exists today's but Still there is a continuous search for finding the best of drugs with minimum adverse effects and proper therapeutic treatment than the available drugs. Keeping in view the growing popularity and market interest of the drug, present studies is undertaken to provide scientific support for its folkloric usage. The significant hypoglycemic and antioxidant activity of Anacyclus pyrethrum extract observed in the present investigation could be the result of its synergistic action of its constituents. Anacyclus pyrethrum has shown significant increase in physical parameters, increase in pain sensitivity. This indicates its protective role against damage to the neurons. Therefore, it can be concluded that Anacyclus pyrethrum has significant hypoglycemic and neuroprotective effects in experimental animals

KEYWORDS- *Neuropathy, Diabetes, Phytochemical, Anacyclus pyrethrum, neurons, synergistic.*

INTRODUCTION

Diabetic neuropathies are a family of nerve disorders caused by diabetes. People with diabetes can, over time, develop nerve damage throughout the body. Some people with nerve damage have no symptoms. Others may have symptoms such as pain, tingling, or numbness—loss of feeling—in the hands, arms, feet, and legs. Nerve problems can occur in every organ system, including the digestive tract, heart, and sex organs. In experimental animal studies of diabetic neuropathy, a rodent model of streptozotocin (STZ)-induced diabetes has been often used for sensory behavioral testing. Tactile allodynia and hyperalgesic sensitivity to thermal and mechanical stimuli occur in the paws of animals. Several analgesic agents, such as gabapentin and mexiletine, were reported to show anti-allodynic effects in an early phase of STZ- induced diabetes. However, few studies have elucidated how temporal changes affect the effectiveness of analgesic agents against pain related behavior as the duration of diabetes is prolonged[18]. The present study therefore aims at evaluating the hydro alcoholic roots extraction of *Anacyclus pyrethrum* for the treatment of diabetic neuropathy in STZ rat model.

- **MATERIAL AND METHOD**

MATERIALS

Plants -The Roots of *Anacyclus pyrethrum DC* was procured from commercial sources.

DRUGS AND CHEMICALS

- Streptozocin- Fortune biotech pvt limited, Delhi,india
- Gliclazide- Aliza-80mg Mefro pharmaceuticals Ltd. All chemicals used were of analytical grade.

METHODOLOGY

- **Extraction**

Plant was procured from available sources and authenticated from Saifia college of Science, Dept. of Botany, Bhopal (Madhya Pradesh). Plant Extraction was done by ether (defatted) and hydro-alcoholic solvent (1:1) through soxhlet apparatus.

ACUTE TOXICITY STUDIES

Acute toxicity studies for extracts of *Anacyclus pyrethrum*. were conducted as per OECD guidelines 423 using albino Wistar rats. Each animal was administered the hydroalcoholic extract by oral route. The animals were observed for any changes continuously for the first 2h and up to 48 h for mortality.

EXPERIMENTAL DESIGN FOR DIABETIC NEUROPATHY

Healthy Wistar albino rats of either sex weighing about 150-200 grams were taken. Animals were deprived to food for 6-8 hours but allowed free access to water. after that blood sample was collected from tail of rats and measure blood glucose level by using GOD-POD kit method . Then they were injected with Streptozocin dissolved in 0.1M sodium citrate and citric acid at a dose of 55 mg/kg body weight intraperitoneally. Then animals were kept for 21 days during which food and water was allowed. After 21days of Streptozocin administration blood glucose level, body weight, grip strength and pain sensation measurements were taken.

The animals showed fasting blood glucose level above 250 mg/dl considered diabetic rats and after that they were divided into

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five groups in which each group contained six animals. the blood glucose level, body weight, measurements of each rat were taken at the start and at the end of experiment After the 3rd week of the experiment test and standard drugs were administered orally daily respectively.

GROUPS	TREATMENT
Normal Control	Control rats received only vehicle
Diabetic control	Streptozocin (55 mg/kg)+ 0.1 M sodium citrate buffer
Gliclazide Group	Gliclazide(10mg/kg/day/p.o.)
Low dose group	Streptozocin (55 mg/kg) + APE 200 mg/kg/day /p.o.)
High Dose group	Streptozocin (55 mg/kg) + APE 400 mg/kg/day /p.o.)

1. PHYSICAL PARAMETERS

- **Body weight**

  The impact of Diabetes in animals results in gradual loss of body weight; hence this will be measured at the starting and last day of experimentation.

- **(Grip-strength) Rota-rod test**

  The test was being used to assess muscular strength or neuromuscular function with the help of rota rod apparatus in which the rats were placed on a horizontal rod running at a speed of 25 rpm. The rats which were capable of remaining on the top for 25 sec or more, in three successive trials were selected for the study. The selected animals were divided into five groups (n= 6). The time spent on the rod was calculated

2. MEASUREMENT OF NOCICEPTIVE THRESHOLD

- **Tail Flick**

  Animals produced abnormal response when subjected to noxious stimuli such as tail flick. Animals are placed into individual cages leaving the tail hanging out freely. The animals are allowed to adapt to the cages for 30 min before testing. The lower 5 cm portion of the tail is marked and the tail of the rat was immersed in hot water maintained at **55°C**. the basal tail flick latency (withdrawal response of tail) or signs of struggle were observed. The cut off time was 10 sec[92, 93, 94].

- **Eddy's Hot plate method**

  Thermal sensitivity was determined using eddys hot plate, temperature at 55-56 ˚C was maintained and observation is done up .the cut off time was taken as 10 sec. Then the average basal reaction time was noted after the oral administration of the drugs and test compounds.

- **Formalin Test**

  It is used as a model for chemical sensitivity or chemical stimuli**,** In this test, each animal was given a subcutaneous injection hind paw using a 25- gauge syringe needle, after having acclimatized into the box. A nociceptive score was determined for each rat after 15 min in a 5 min block during that period by measuring the amount of time spent in each of the four behavioral categories: 0, the position and posture of the injected hind paw is indistinguishable from the contralateral paw 1, the injected paw has little or no weight placed on it; 2, the injected paw is elevated and is not in contact with any surface; 3, the injected paw is licked, bitten, or shaken. Then, a weighted nociceptive score, ranging from 0 to 3 was calculated by multiplying the time spent in each category and summing these products and dividing by the total time for each 5 or (300 sec) min block of time. The observations were noted for next 60 mins after placing animals into the box.

1. BIOCHEMICAL PARAMETERS

- **Blood Glucose Level Measurement**

  The animal's blood was collected from tail of rats for the determination of the blood glucose levels. The plasma was obtained after centrifugation (3000 rpm for 10 min, 4°C). Blood glucose was estimated by GOD-POD kit method[101,]102,103.

- **Total protein**

  Since the total protein is thought to be abnormal in disease animals therefore the amount **o**f total protein measured at 546 nm according to standard method by using commercially diagnostic kits

1. ENZYMATIC ESTIMATION

Antioxidants level

Preparation Of Sciatic Nerve For Estimation Of Oxidative Stress Marker:

The sciatic nerve of each animal was removed after completion of experiment, weighed and homogenized with 10 times (w/v) ice cold phosphate buffer saline (50mM pH 7.8) in teflon glass homogenizer.

The homogenate was centrifuged at 1000 rpm 4 ◦C for 3 min and the supernatant divided into two portions, one of which was used for measurement of lipid peroxidation (LPO) and the remaining supernatant was again centrifuged at 12,000 rpm at 4 ◦C for 15 min and used for the measurement of superoxide dismutase (SOD), catalase (CAT), and glutathione

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(GSH).

1. Estimation of Lipid peroxidation (LPO)

The level of Lipid peroxides was estimated by Thio barbituric acid reaction method described by Ohkawa.et.al.

Reagents

1. Sodium dodecyl sulphate (SDS) (8.1 %)
2. Acetic acid (20%; pH 3.5)
3. Thiobarbituric acid (TBA) (0.8%)
4. N-butanol/pyridine mixture (15:1, v/v)

Procedure

To 0.2 ml of test sample, 0.2 ml of SDS, 1.5 ml of acetic acid and 1.5 ml of TBA were added. The mixture was made up to 4 ml with water and then heated in a water bath at 95°C for 60 minutes. After cooling, 1 ml of water and 5 ml of n-butanol/pyridine mixture were added and shaken vigorously. After centrifugation at 4000 rpm for 10 minutes, the organic layer was taken and its absorbance was read at 532 nm. The level of lipid peroxides was expressed as nmoles of MDA released/ g wet tissue.

RESULTS

1.1 ACUTE TOXICITY STUDY

There was no change in normal behavioural pattern of animals and no sign and symptoms of toxicity were observed during the observations which was done continuously for the twenty four hours and then observed up to seven days for mortality. The extracts were safe up to a maximum dose of 2000 mg/ kg body weight. The biological evaluation was carried out at doses of 200 and 400 mg/kg body weight.

1.1 PRELIMINARY PHYTOCHEMICAL SCREENING:

Table No. - Data Showing Preliminary Phytochemical Screening of the hydroalcoholic extract of *Anacyclus pyrethrum.*

S. No.	PHYTOCONSTITUENTS	HYDROALCOHOLIC EXTRACT(APE)
1	Carbohydrates	-
2	Proteins	-
3	Saponins	+
5	Alkaloids	-
6	Tannin and Phenolic compound	+
7	Flavonoids	+
8.	Glycosides	-
9.	Fixed oil and fat spot test	-

- ABESNT, + PRESENT

- ✦ Body weight
- ✦ Grip strength (Rota- rod)

High blood glucose levels along with weight loss were seen in adult rats treated with STZ in diabetic control group. The body weight increased normally in normal control rats (181.3± 1.21), while Diabetic control group showed a significantly *(P<0.001)* decreased (136±5.02) in body weight. The extract treated groups showed significantly *(P<0.001)* increased in body weight when compared to the diabetic control group.

APE 400 produced maximum response on body weight (173.8±1.47) when compared to (136.0 ± 5.02) diabetic control group. (Table no.10 & Fig. no.15)

The muscle grip strength reduced significantly *(P<0.001)* in all STZ treated groups. The grip strength of Gliclazide (26.33±1.21) and APE 200 mg/kg (17.67± 1.03) and APE 400 mg/kg (24.33±1.211) treated animals were increased significantly *(P<0.001)* compared to the Diabetic control group(136.0 ± 5.02). The grip strength of Gliclazide drug and APE 400 mg/kg treated animals were found to be comparable with normal control group. (Table no.10 & Fig. no.14.)

Table.10 Effect of different doses of APE on the body weight and grip strength in STZ induced diabetic neuropathy model in rats.

Groups	Body weight (gms)	Grip strength (sec)
Control	181.3± 1.21	28.33± 1.21
Diabetic control	136.0 ± 5.02$^{\neq}$	10.17± 1.47$^{\neq}$
Gliclazide10mg/kg	179.2 ± 4.80***	26.33 ±1.21 ***
APE 200mg/kg	161.5± 1.04***	17.67± 1.03 ***
APE400mg/kg	173.8± 1.47***	24.33±1.21***

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Data were expressed as mean±S.D. (n=6) and analyzed by one way ANOVA followed by, dunnets test. *** P<0.001, when compared to diabetic control group, ≠P<0.001 when compared to Normal control group.

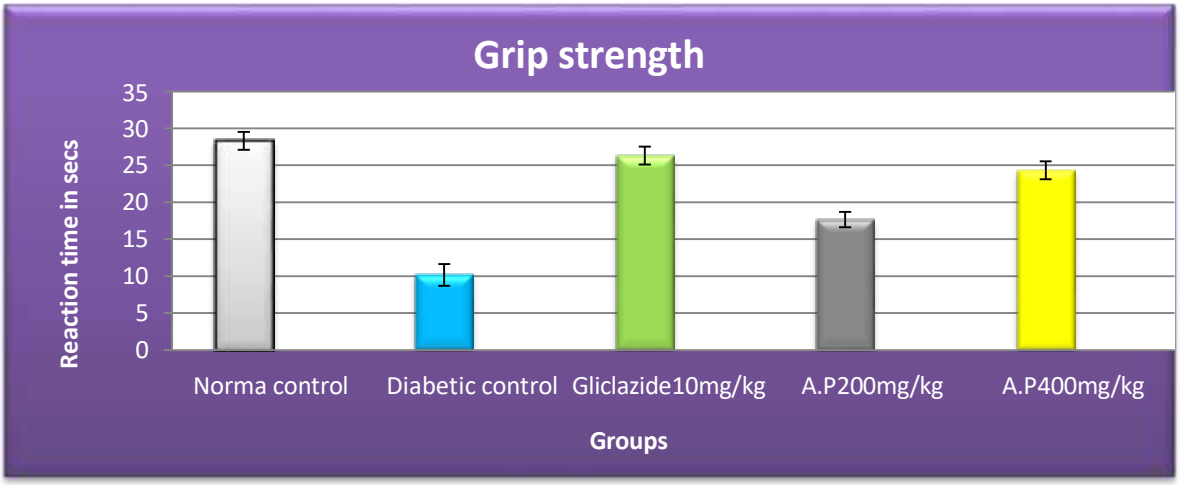

Fig.14 Effect of different doses of APE on grip strength in STZ induced diabetic neuropathy model in rats.

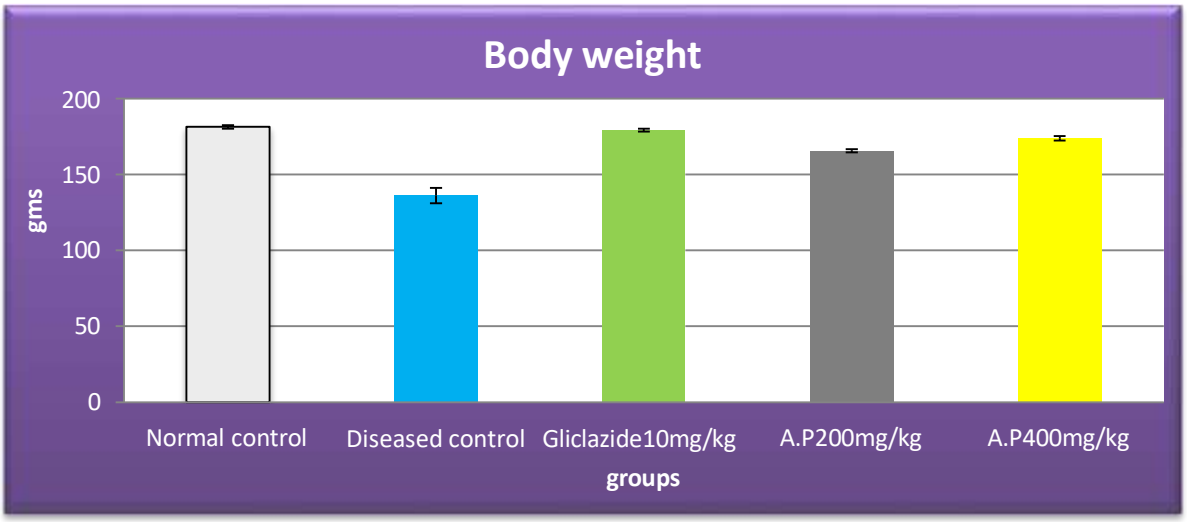

Fig. Effect of different doses of APE on body weight in STZ induced diabetic neuropathy model in rats.

2.MEASUREMENT OF NOCICEPTIVE THRESHOLD

- Tail Flick
- Hot plate
- Formalin Test

3. Effect of different doses of APE on the reaction time in tail flick, hot plate and formalin test in STZ induced diabetic neuropathy model in rats.

Groups	Tail flick (Reaction time in sec)	Hot plate (Reaction time in sec)	Formalin test (Test score)
Normal Control	9.00 ±0.70	8.333 ± 0.81	1.23 ± 0.10
Diabetic Control	3.40±1.140$^{\neq}$	3.000 ± 0.89$^{\neq}$	2.81 ±0.07$^{\neq}$
Gliclazide10mg/kg	7.80±0.83***	8.000 ± 0.894***	1.35 ± 0.10***
APE 200mg/kg	3.60±0.54^{ns}	4.500 ± 0.54***	2.50 ± 0.08***
APE400mg/kg	7.00± 0.70***	7.167± 0.75***	1.467 ± 0.12***

4. Data were expressed as mean±S.D. (n=6) and analyzed by one way ANOVA followed by, dunnets comparison test. ***P<0.001, when compared to Diabetic control group, ≠P<0.001 when compared to Normal control group, ns= when compared to Diabetic control group.

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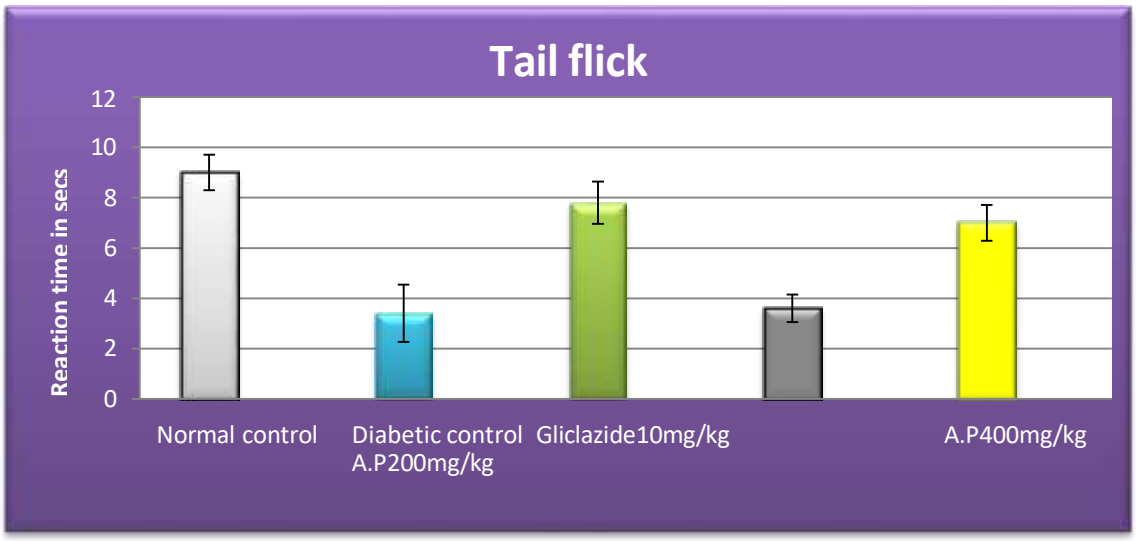


Fig. Effect of different doses of APE on formalin test in STZ induced diabetic neuropathy model in rats.

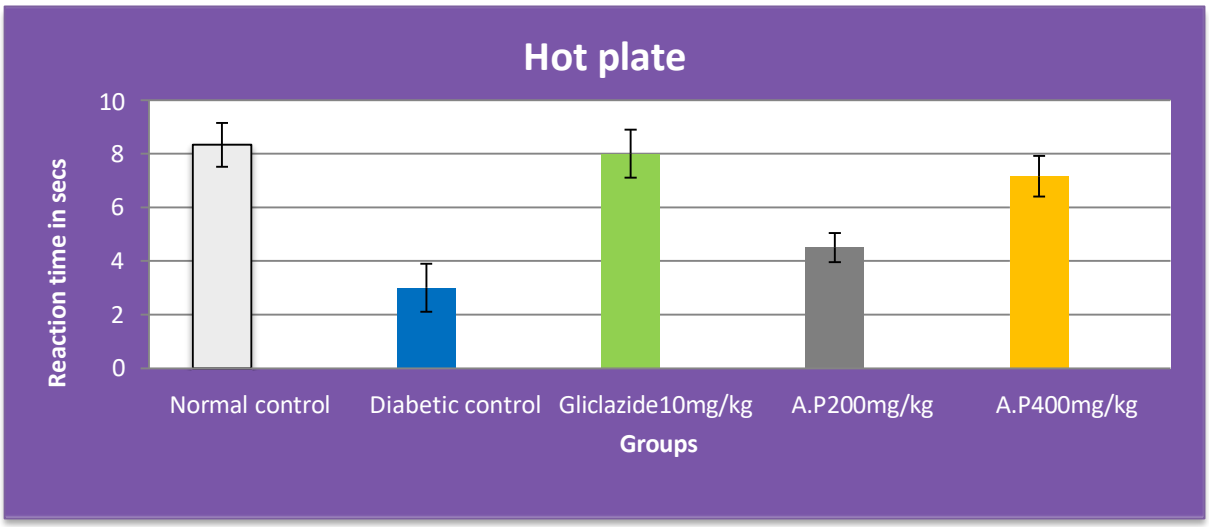
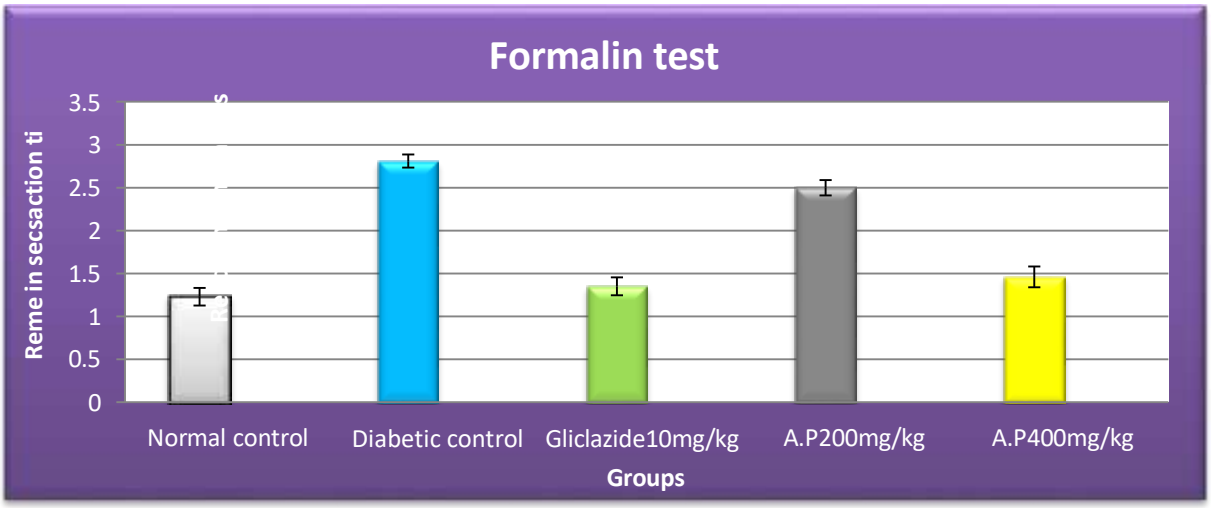


Fig. Effect of different doses of APE on hot plate in STZ induced diabetic neuropathy model in rat



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5. Enzymatic Estimation

The lipid peroxidation level significantly *(P<0.001)* increased in diabetic group as compared to normal control group. In lipid peroxidation level of gliclazide (1.39± 0.02) APE 200 mg/kg (15.32±0.37) & APE 400 mg/kg (28.13±0.58) were significantly *(P<0.001)* decreased when compared to diabetic group. The high doses of APE exhibited comparable effect to standard group (Table.no.13 and Fig.no.22).

The diabetic control group showed significant *(P<0.001)* decreased in SOD (11.37±0.98) level as compared to (29.66±0.59) normal control group. SOD level was significantly *(P<0.001)* improved with the treatment of APE 200 mg/kg (15.32±0.370),
400 mg/kg (28.13±0.58) and Gliclazide (28.68±0.97) doses when compared with diabetic group. The higher doses of extract exhibited comparable effect to standard group (Table.no.13 and Fig.no.21).

The diabetic control group (1.16±0.01) showed significantly *(P<0.001)* reduction in CAT level as compared to (2.87 ±0.23) normal control group. CAT level was significantly *(P<0.001)* improved treatment in extracted treated groups when compared to diabetic control group. The higher doses (2.49±0.18) of extract exhibited comparable effect to standard group (Table.no.13 and Fig.no.23).

Similarly the diabetic control group showed significantly *(P<0.001)* decreased in GSH (glutathione reductase) level (0.01±0.0012) as compared to normal control group (0.03±0.0018). APE treated groups were showed significant *(P<0.001)* increased in GSH level. The higher doses (0.02±0.0035) of extract exhibited comparable effect to standard group.

Table Effect of different doses of APE on the GSH, LPO, SOD and CAT level in therat's sciatic nerve in STZ induced diabetic neuropathy model.

GROUP	SOD (unit/mg protein)	LPO (nm/mg protein)	GSH (units/mg protein)	CAT (K/ min)
Normal Control	29.66±0.59	1.35±0.03	0.03±0.0018	2.87 ±0.23
Diabetic Control	11.37±0.98$^{\neq}$	3.51±0.05$^{\neq}$	0.01±0.0012$^{\neq}$	1.16±0.01$^{\neq}$
Gliclazide (standard)	28.68±0.97***	1.39± 0.02***	0.02±0.0018***	2.66±0.19***
APE 200	15.32±0.37***	2.27±0.03***	0.02±0.0017***	1.57±0.05***
APE 400	28.13±0.58***	1.44±0.03***	0.02±0.0035 ***	2.49±0.18***

Data were expressed as mean±S.D. (n=6) and analyzed by one way ANOVA followed. by, dunnets comparison test. *** = P<0.001when compared to diabetic control group, $^{\neq}$P<0.001 when compared to Normal control group

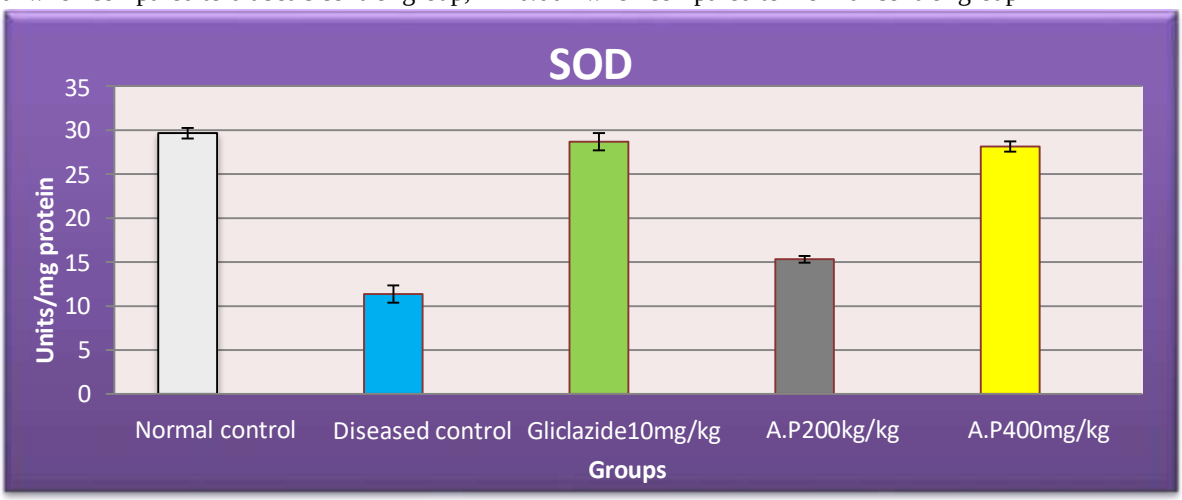

Fig. Effect of different doses of APE on the SOD level in the rat's sciatic nerve in STZ induced diabetic neuropathy mode

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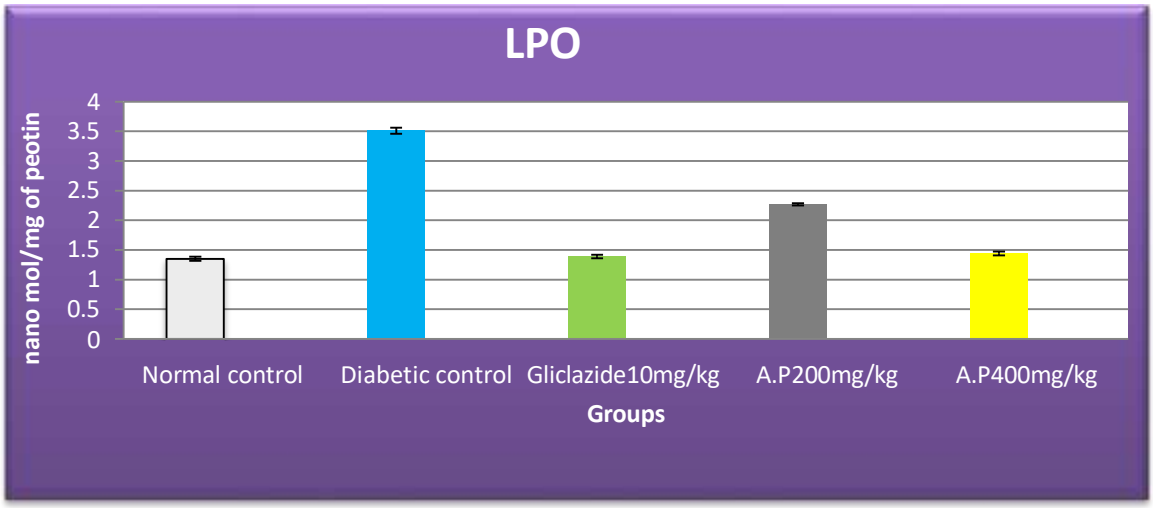

Fig. Effect of different doses of APE on LPO level in the rat's sciatic nerve in STZ induced diabetic neuropathy model.

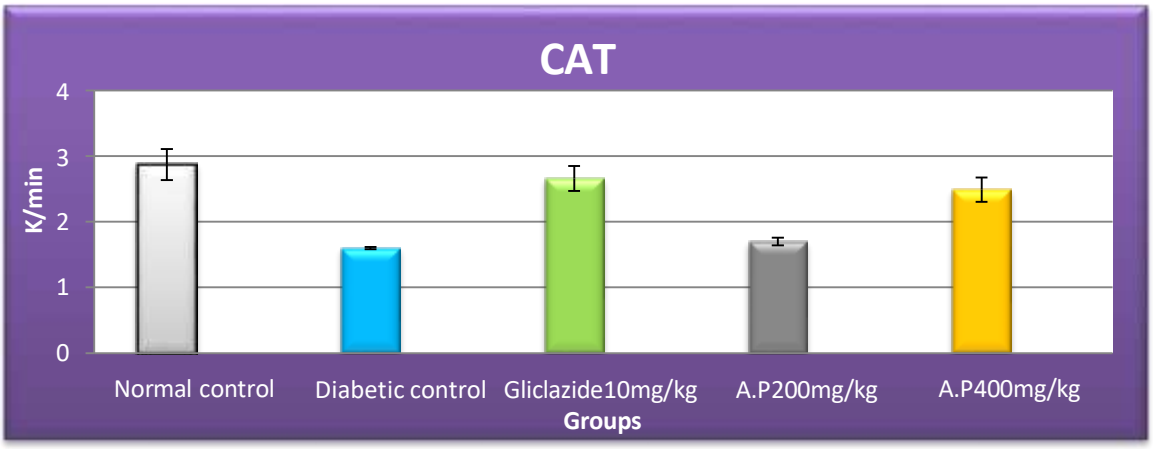

Fig. Eff ect of different doses of APE on the CAT level in the rat's sciatic nerve in STZ induced diabetic neuropathy model.

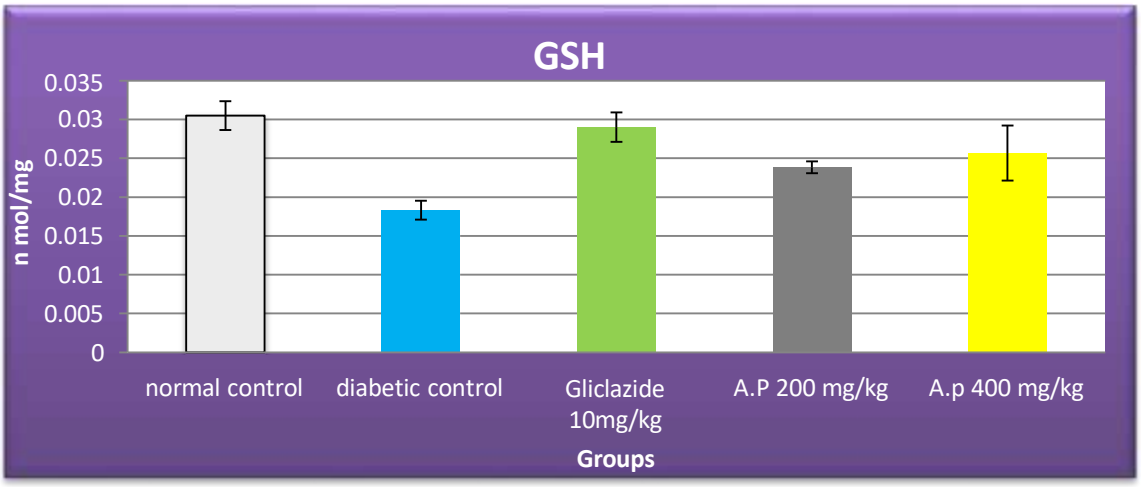

Fig. Effect of different doses of APE on the GSH level in the rat's sciatic nerve in STZ induced diabetic neuropathy model.

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DISCUSSIONS

Diabetic neuropathy is a devastating complication of diabetes mellitus that makes quality life of patients more than worst. Diabetic neuropathy is characterized by clinical features like allodynia, hyperalgesia due to elevated nociceptive response, reduced motor nerve condition velocity, neuronal hypoxia, reduced threshold to painful stimuli, etc. Similar symptoms are exhibited by STZ induced animals. STZ injected rat's exhibit clinical pathological features including biochemical, oxidative and metabolic changes which also presented in human.

Streptozotocin causes significantly *(P<0.001)* decreased in total protein level, amino acids, Phenolics, alkaloids, tannins have been reported as anti hyperglycemic agents, The decreased rate of total protein may be due to several reasons like increased rate of amino acids conversion to glucose, decreased amino acids uptake, and increased conversion rate of glycogenic amino acids to CO2 and H2O, a loss of transitional factor, reduction of ribosomal protein synthesis. Total protein may be increased by antioxidant defensive mechanisms[104, 105].

Prolonged hyperglycemia causes overproduction of ROS. Once these formed they deplete antioxidant defenses (Superoxide dismutase, glutathione peroxidase, catalase) rendering the affected cells and tissues, more susceptible to oxidative damage, increased peroxidation and ALE catalyzed by hyperglycemia and oxidative stress may play critical role in diabetic neuropathy in which damage dorsal root ganglion mitochondrial DNA, adding to long-term nerve dysfunction[61-65]. As both vascular and metabolic factors impairment have found to play role in genesis of Diabetic neuropathy[106].

Moreover administration of APE restored various biochemical parameters lipid peroxidation, decreased levels of antioxidant warrior enzymes (GSH, Catalase and SOD) generated by ROS, levels of ROS rise with poor metabolic control. ROS are also produced by macrophages during their respiratory burst, to destroy infectious organisms and in inflammatory diseases. For peripheral nerve, ROS can directly damage neurons and Schwann cells and in combination with diabetes antioxidant protection mechanisms are compromised. Thus, in diabetic rats, sciatic nerve lipid peroxidation was increased and levels of superoxide dismutase (SOD) and the reduced form of glutathione (GSH) were decreased.

Treatment with APE for 3 weeks results in significant improvement in SOD, GSH, Catalase and decrease in LPO. *Anacyclus pyrethrum* is a potent vasodilator & considered as tonic to the nervous system so it may help in improving nerve conduction velocity. Overall it can be summarized that APE having antioxidant properties behind protection of neurons. However further studies are warranted to explore the exact mechanism of APE.

CONCLUSION

Diabetic neuropathic pain, an important microvascular complication in diabetes mellitus, is recognized as one of the most difficult types of pain to treat. Streptozotocin is widely used to induce diabetic neuropathy in experimental animals. The development of tolerance, inadequate relief and potential toxicity of classical antinociceptive alarms the investigation of the newer agents to relieve this unbearable pain. Hyperglycemia induced oxidative stress are responsible for diabetic neuropathy.

The significant hypoglycemic and antioxidant activity of *Anacyclus pyrethrum* extract observed in the present investigation could be the result of its synergistic action of its constituents. *Anacyclus pyrethrum* has shown significant increase in physical parameters, increase in pain sensitivity. This indicates its protective role against damage to the neurons. Therefore, it can be concluded that *Anacyclus pyrethrum* has significant hypoglycemic and neuroprotective effects in experimental animals.

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