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ANTIDIABETIC ACTIVITY OF *AMRITHADI CHURNAM*

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ABSTRACT: *Amrithadi Churnam – a compound ayurvedic preparation made up of Tinospora cordifolia, Salacia prenoides, Curcuma longa, Tribulus terrestris and Emblica Officinalis was screened for its antidiabetic activity. From the studies it could be established that Amrithadi churnam at a dose level of 100mg/kg b.w. was the optimum dose in alloxan diabetic rats. No toxic effects were observed as evidenced by the study of liver enzymes and blood haematoerit. An extra pancreatic role of the drug cannot be rulled out, since it (100 mg/kg b.w) produced significant decrease in blood sugar level in alloxan diabetic rats.*

INTRODUCTION

Amrithadi churnam is a compound ayurvedic preparation made from five constituent drugs viz. *Tyinospora cordifolia* (San. *Guduchi), Salacia prenoides* (San. *Ekanayakam,) Curuma longa* (San. *Haridra), Tribulus terrestris* (San. *Goksura)* and *Phyllanthus emblica* (San.*Amalaki).* The powder is known to be administered to diabetic patients by many ayurvedic physicians to cure the metabolic disorder. However, scientific evidence on the antidiabetic activity of the powder as such is lacking. The individual constants in the powder have been attributed several medicinal properties from very early times. *T. Cordifolia* has been mentioned in Ayurvedic literature as an ingredient in several compound preparations used in general debility, dyspepsia, fever and urinary disorders (Wealth of India, 1976). Antiviral effect of the plant against Ranikhet disease of the poultry has been reported by many workers (Chopra *et. al* 1958, Dastur, 1951; Kirtikar et al 1935). The active principle of the drug has been found to produce in vitro inhibitory effect on *Mycobacterium tuberculosis* (Annual report, ICMR. 1968 – 69). The acute and chronic effects of feeding plant extracts on blood sugar, glucose tolerance etc. were studied in animals and the aqueous and alcoholic extracts were found to reduce blood sugar levels. The drug has a suggested effect on endogenous insulin secretion and inhibition of peripheral glucose release. (Mehta et al., 1965, Gupta et al 1967). Roots of *Salaciaprenoides* have been reported to be used as an antidiabetic drug in the indigenous system of medicine. The medicinally active components of the root bark have been characterized by several workers (Dash & Bedi 1967; Bhatnagar *et al*, 1954, Ramanathan et al, 1960; Krishnan et al, 1967; Pillay et al 1957). Earlier studies conducted with three species of *Salacia* also indicated the hypoglycemic activity of the plant (Nair et al 1981). *C. longa* has been mentioned in ancient literature as a stomachic, tonic and blood purifier. (Wealth of India, 1950). Chemical characterization of the rhizome was made by several workers

(Health Bulletin, 1941; Mayer 35 al, 1943) and choleretic action of its essential oil has been attributed to P-tolymlthylcarbional *T. Terrestris* is being used for the treatment of renal calculus and painful micturition. The diuretic activity of the drug has been studied in detail. (Santhakumar et al, 1967). The seeds were reported to contain an alkaloid to which the diuretic effect was ascribed. (Wealth of India. 1976). The root extract is a constituent of many Ayurvedic preparations like *Dasamoolarishta* and *Amrithaprasa ghritha* prescribed for several disease. (Koman, 1919 Rama Rao, 1914; Kirtika et al, 1935, Pradhan, 1963). The medicinal properties of Amla fruit (Kirtikar et al, 1935; Nadkarni, 1914; Koman, 1918) are well documented and it form constituent of many Ayurvedic preparations.

Although individual components of *Amrithadi churnam* have been ascribed for many therapeutic effects, the synergistic action of remains relatively unknown. The antidiabetic action of the powder was studied in albino rats in comparison to the effect of standard hypoglycemic drugs tolbutamide and phenformin.

MATERIALS AND METHODS

Adult albinos rats (Sprage-Dowley strain) weighing 180 – 200g. were used for the study. Before the start of the experiment animals were fasted overnight and blood sugar values were noted.

Blood was collected by the eye puncture method using clean capillary tube. The animals were fed standard feed and water was available ad-libitum.

Healthy male albino rats from the colony were made diabetic by the single rapid ip injection of 120 mg/kg body weight of alloxan monohydrate. (5% w/v in distilled water) After 3 days the fasting blood sugar levels were rechecked and diabetic animals were selected on the elevated sugar values. These were grouped into six each containing 5 rats. Group Administered distilled water served as control Group II-IV were given *Amrthidai churnam* at dose levels of 50mg. 100mg and 200 mg/kg. body weight. Group V and VI were given tolbutamide (250 mg/kg. body weight). Group V and VI were given tolbutamide (250 mg/kg b.w) and phenformin (20 mg/kg b.w) served as reference standards. Respective drugs were administered for 4 weeks and the overnight fasted animals were sacrificed. Blood and liver samples were collected for the biochemical analyses.

Blood sugar was determined by the method of Astoor and King (1954). Serum total proteins were determined using Biuret method (1945). Serum and liver transaminase (GOT and GPT) were determined by the method of Reitman & Frankel (1957). Blood hematocrit was recorded following standard procedures.

RESULTS AND DISCUSSION

The initial fasting blood sugar values of alloxan diabetic rats ranged from 206 – 305 mg/ 100 ml. (Table I). It was found that administration of *Amrithadi churnam* at a dose level of 50 mg/kg b.w. could bring down the blood sugar level to 152.7 ± 9.99 mg/100ml. A dose level of 100 mg/kg b.w. could significantly reduce the hyperglycemia observed before the treatment (Table. I). However the higher dose level of 200 mg/kg b.w. could not further bring down the blood sugar. Its effective was comparable to the lowest dose level tried. From the studies it could be established that 100 mg/kg b.w. was the optimum dose level in rats. Only non significant hypoglycemeic changes were

noted in rats given tolbutamide while phenoformin exhibited a significant hypoglycemic effect (Table I). This was observed from the results of earlier studies also (Nair et. al. 1986).

Serum and liver protein changes in rats given *Amrithadi churnam* are given in Table II. It could be seen that at the lower dose levels the drug produced no effect on serum protein levels. However the higher dose level produced significant decrease in the serum protein liver protein remained unaffected at all dose levels (Table II).

The pattern of changes in serum and liver transaminase depicted in Table III to show that the drug did not alter the serum and liver glutamate oxalo acetate transaminase activity. While the serum GPT remained unaffected at the three dose levels tried, liver GPT activity was considerably reduced at the lowest dose level 50 mg/kg b.w. tried. However the optimum dose level of the drug as judged from the maximum hypoglycemic effect being 100mg / kg b.w, this decrease in GPT activity of liver has little significance.

Tolbutamide was found to be ineffective in alloxan diabetic animals. Since alloxan administration has bee reported to damage the B-cells of langerhans of pancreas, the site of insulin release. The hypoglycemic effect of tolbutamide is mainly through enhancing insulin release in normal diabetic condition. (Jenkin's et al; 1978) Phenformin on the other hand increase glucose uptake by peripheral tissues and accelerates anaerobic glycolysis through an uncoupling of exudative phosphorylation (Nair and Santhakumar,1986).

The hypoglycemic effect of *Amrithati churnam* observed in alloxan diabetic animals indicates an extract pancreatic action of the drug. The exact mode of action of the drug needs further investigations. The serum and liver protein levels as well as transaminase activities indicate the nontoxic nature of the drug at lower level of 50 mg and 100 mg/kg b.w. Also the blood haemtocrit indicates the non-toxic nature of thedrug (Table IV). The study shows that *Amrithadi churnam* has got potent antidiabetic / hypolycaemic effect at the optimum dose level of 100 mg/kg b.w in rats.

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TABLE – I

Effect of *Amirthadi Churnam* on Blood Glucose level in alloxan Diabetic Rats.

Group / Drug dose levels	Initial blood Sugar mg/100ml	Blood glucose levels after 4 weeks mg/100ml
Group I	230.8	220.4
(Diabetic control)	± 22.63	±17.79
Group II	207.04	152.7
50 mg/kg b.w	± 14.15	±9.99 **
Group III	206.2	126.54
100 mg/kg b.w.	± 15.49	±5.93 **
Group IV	233.8	152.84
200 mg/kg b.w	± 13.06	±10.28 **
Group V	305.5	290.30
Tolbutamide	±18.2	± 8.5
Group VI	277.8	147.5
Phenformin 20 mg/kg b.w	± 13.8	±15.9 ***

** P<.01

*** P<.001

Figures without superscripts are insignificant

TABLE – II

Serum and Liver protein values in Alloxan diabetic rats administered *Amrithadi Churnam*

Group / Drug dose levels	Serum g/100ml	Liver g/100gm wet tissue
Group I	7.85	19.36
(Diabetic control)	± 0.23	±1.25
Group II	7.92	18.90
50 mg/kg b.w	± 0.35	±1.26
Group III	7.18	19.06
100 mg/kg b.w.	± 0.32	±1.88
Group IV	6.14	162.88
200 mg/kg b.w	± 0.30	±0.55
.P<.001		

Others are non-significant values

TABLE III
Serum and liver got and GPT activities in rats administered *Amrithadi churnam*

Group / Drug	GOT Serum[1]	Liver[2]	GPT Serum[1]	Liver[2]
Group I	9.82	3.35	24.25	28.27
(Diabetic control)	±0.62	±0.38	±2.18	±1.60
Group II	10.08	2.76	24.16	23.08
50 mg/kg b.w	±0.99	±0.44	±0.28	±1.58*
Group IV	9.88	3.38	21.86	25.34
200 mg/kg b.w	±0.50	±0.36	±1.70	±1.86

P <0.05
1. m pyruvate liberated/min/litre at 37^0C.
2. m pyruvate liberated /min/mg protein at 37^0C

TABLE IV
Blood Hematocrit in alloxan rats administered *Amrithadi churnam*

Group / Drug Dose levels	Total count (TC) cell /c.m.m	Differential count (DC)		
		Polymorph (P)	Lymphocytes (L)	Eosinophile (E)
Group I	6975	43	47	10
(Diabetic control)	±100.3	±3.5	±2.8	±.50
Group II	7838	38	49	13
50 mg/kg b.w	±430.2	±4.3	±5.6	±.95
Group III	5835	51	43	6
100 mg/kg b.w	±330.6	±2.60	±3.6	±.76
Group IV	6836	45	47	8
200 mg/kg b.w	±530.2	±3.2	±4.1	±0.70

All are non significant values.

REFERENCES

1. Anonymous – Traditional treatment.

2. Annual Report. Indian Council of Medical Research P.92 (1968 – 69).

3. Asatoor, AM and King E.J. Biochem. J. 56 (X-IV) (1954).

4. Bhaskaran Nair R, Bhat A.V. and Santhakumari G.J. Res. Ay. Sid. Vol III, No. 1&2, P 53 (1981).

5. Bhaskaran Nair R, Bhat A.V and Santhakumari G J. Res. Ay. Sid. Vol. III, No. 1 &2, P 53 (1981).

6. Bhatnagar S.S. etal. J. Amer. Chem. Sec. Vol. 786, P. 3689, (1954).

7. Chopra R.N. Chopra I.C. Handa K.L., and Kapur L.D. Chopra.

   Indigenous Drugs of India, (U.N. Dhur & Sons Private Ltd., Calcuta) 2nd Edn. P. 427 (1958).

8. Dash and Bedi, I.S.I. Bulletin, Vol. 19 p. 392 (1967).

9. Destur J.F. Medicinal Plants of India and Pakistan (D.B. Taraporevale Sons & Co., Ltd., Bombay) P 232, (1951).

10. Fritz Mayer and Cook A.H. The Chemistry of Natural colouring matters (Reinhold publishing corporation, New York) P. 93, (1943).

11. Gupta B.L. et al. Indian Journal of Medical Research Vol.55 p. 733 (1967).

12. Health bulletin, India No.23, P. 37 (1941).

13. Jenkins D.J. A et al., Br. J. Med. Res. Vol.2, p. 1714 (1978).

14. Kirtikar, K.R. Basu B.D and ICS (retired) (lalit Mohan Basu, Allahabad) 2nd Edn. Vol. I P. 421. (1935).

15. Kritikar K.R. Basu B.D. and an ICS (retired) (Lalit Mohan Basu, Allahabad) 2nd Edn. Vol. III.,) 2220 (1935).

16. Koman M.C. Report on the Investigation of Indigenous Drugs (Govt. Press. Madras) p.6 (1918).

17. Koman M.C. Report on the Investigation of Indigenous Drugs (Govt. Press, Madras) P.6 (1919).

18. Krishnan M.S. and Rangaswami S. Tetrahedron Lett. P. 2441 (1967).

19. Mehl. J.N.J Biol Chem. P. 157 (1945).

20. Mehta and Naik K.C.J. Ind. Med. Ass. Vol. 45, P. 38 (1965).

21. Nadkarni K.N. Indian Plants and Drugs (Norton & Co.,) P. 151 (1914).

22. Pillay P.Petal Bull. Res. Inst. University of Kerala, Vol. 5A. (1), P.29 (1957).

23. Pradhan S.S. Ind J. Pharm Vol 25.P. 303, (1963).

24. Ramanathan K.R. and Seshadri T.R. Curr. Sci. Vol. 29, P. 131. (1960).

25. Rama Rao M. Flowering Plants of Travancore, (Govt. Press. Trivandrum) P. 57 (1914).

26. Reitman, Sand Frankel S. Amer. J. Cli. Path. Vol. 28, P. 56 (1957).

27. Santhakumari G. et al. Ind J. Med. Res. Vol. 54, p. 714 (1967).

28. Wealth of India, Vol. II, Raw materials. Publications and information Directorate, CSIR, New Delhi P. 404, (1950)

29. Wealth of India, Vol X Raw materials, Publication and Information Directorate, CSIR, New Delhi P. 251. (1976).

30. Wealth of India, Vol. X, Raw material, Publications ad Information Directorate, CSIR, New Delhi. 284. (1976).