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## RESEARCH ARTICLE

# In Effect of Poorna Chandrodayam Chendooram (metallic drug) on liver function, kidney function and lipid profile parameters of rats

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**ABSTRACT:** Poorna chandrodayam chendooram (PCM), a Siddha preparation which is used for its effect on liver function, kidney function and lipid profile after administrations into the biological system. The experimental animal model was of Male rats. Triglycerides (TG) Total cholesterol (TC), and low density lipoprotein (LDL) were decreased in experimental groups whereas high density lipoprotein (HDL) were slightly increased in male rats. The total protein and albumin content of plasma were increased very high significantly. In case of bilirubin, the decrease was negligible for rats. The serum glutamic pyruvic transaminase (sGPT), serum glutamic oxaloacetic transaminase (sGOT) and alkaline phosphatase (ALP) content in the plasma were decreased very high significantly in the experimental groups. Creatinine, and urea were decreased in male where only change of uric acid level was significant increased.

**Keywords:** PCM, TG, TC, LDL, HDL, SGPT, SGPT and ALP

## INTRODUCTION

Indian alchemy is one of the disciplines in which Parpam, Chendooram and Chunnam were first described as intriguing formulations of metals and minerals such as gold, silver, copper, iron, zinc, mercury, and so forth, apparently associated with organic macromolecules derived from the herbal juices by alchemic processes making these biologically assimilable. (Savrikar 2004). Minerals are combined with herbs that assist the assimilation and delivery of the ingredients to the human body (Suoboda 1998). These herbo mineral medicine are prepared by repeated incineration of metals or their salts (preferably oxides) with medicinal herbs or their extracts so as to eliminate their harmful effects and are taken along with honey, milk, butter, or ghee (a preparation from milk) (Patel, 1986). Most of the medicines are mixture of compounds and because of its synergistic action; toxicity is being diminished, thereby increasing bioavailability through the cells of the body. Treating the minerals with herbal juices may lead to reduction in particulate size even up to nano levels (less than 100 nm) enable increased potency.

Poorna chandrodayam chendooram is a well-known, mercurial preparation with gold and sulphur (Thiagarajan., 1992) widely used for many ailments like tuberculosis, jaundice, fever, rat bite, cancerous ulcer, sprue and male sterility. (Muthaliar, 1987) Hibiscus and Aloe juice is added for titration. (Mahdihassan 1985) These drugs are mostly a mixture of compounds and because of its synergistic action and purification process (Austin, 2002) toxicity is being diminished. (Hardy *et al.*, 1995), thereby increasing bioavailability through the cells of our body. (Sudha *et al.*, 2009) These drugs are known to be effective even in low concentration. (Kumar *et al.*, 2006) The phytochemical studies of this drug Poorna chandrodayam chendooram has shown to contain flavonoids, phenols, and Vitamin C (Muthukumar and Hazeena Begum., 2014), but a clear picture of its toxicokinetics is still obscure. The present study was aimed at evaluating the Liver, kidney and Lipid profile of Normal and PCC treated in experimental animal model.

## MATERIALS AND METHODS

### SELECTION OF ANIMAL

Healthy and pure strain Male Wistar rats, *Rattus norvegicus*, ranging from the body weight of 120-150 g were procured from the Venkateshwara Enterprises, Bangalore and maintained in the Central Animal House, Department of Siddha Medicine, Tamil University, and Thanjavur. Experimental protocol was approved by the Institutional Animal Ethics Committee (IAEC) of Tamil University, Thanjavur. The animals were maintained on standard diet (Kamadhenu Agencies, Bangalore) and water was given ad libitum.

## DRUG PREPARATION

The (Poorna Chandrodayam Chendooram drug obtained from the SKM Siddha and Ayurvedic Medicine's India Private Limited, Saminathapuram, mudakurichi, Erode- 638104. Tamilnadu, India. The drug (Poorna Chandrodayam Chendooram) is not soluble in water therefore a suspension of gum acacia is made for oral administration. The 10 gm. of gum acacia dissolved in 100 ml of distilled water by gradual trituration in a mortar. Then well prepared solution was taken and added Poorna chandrodayam chendooram at the dose of 3 mg/ml/100 gm.

## EXPERIMENTAL DESIGN

After acclimatization, the rats were divided into 2 groups, each having 8 rats.

**Group I:** Untreated control were received water only.

**Group II:** Young rats were treated with Poorna chandrodaya chendooram<sup>4</sup> (3.0 mg / kg body wt. calculated from human dose) with honey for 7 weeks (orally administered).

## BLOOD SAMPLES COLLECTION AND PREPARATION OF PLASMA

Blood samples were collected from post vena cava and transferred into heparinised tubes immediately. Blood was then centrifuged at 4,000 g for 10 min using bench top centrifuge to remove red blood cells and recover plasma. Plasma samples were separated and were collected using dry Pasteur pipette and stored in the refrigerator for analyses. All analyses were completed within 24 h of sample collection.

## DETERMINATION OF BIOCHEMICAL PARAMETERS

To assess the state of the liver and kidney, biochemical studies involved analysis of parameters such as total protein, serum albumin, blood urea nitrogen (BUN), bilirubin, creatinine, and liver enzymes such as aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP). For lipid profile study, triglycerides (TG), total cholesterol (TC) and high density lipoprotein (HDL) were determined but low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were calculated.

Biuret method (Plummer, 1971) was followed to determine the Total protein and serum Albumin concentration was determined by using the method of Doumas *et al* (1971). TG, TC and HDL concentration were evaluated according to Friedewald's formula (Friedewald *et al.*, 1972) Serum bilirubin was determined according to the method of Evelyn and Malloy (1938). The procedure of Tietz *et al* (1994) was used to determine serum creatinine concentration while the serum urea concentration was determined by the method of Kaplan (1965), King and King (1954) method was employed to determine serum glutamic pyruvic transaminase (SGPT), serum glutamic oxaloacetic transaminase (SGOT) and alkaline phosphatase (ALP). The absorbance of all the tests were determined using spectrophotometer (UV-Visible Spectrophotometer Model No. UV-1601 PC.).

## RESULT AND DISCUSSION

### LIVER FUNCTION

Proteins are important organic substances required by an organism in the tissue building, the cellular organelles repair and also cellular metabolism (Yeragi *et al.*, 2000). Albumin constitutes a major antioxidant defense against oxidizing agents (Halliwell *et al* 1998). Bilirubin estimation is reliably sensitive in the diagnosis of hepatic disease (Harper., 1991), because bilirubin is a by-product of the breakdown of hemoglobin. The determination of the pathophysiological enzymes like SGOT and SGPT is a common mean of detecting the liver status. Alterations in SGOT and SGPT values are reported in hepatic disease or damage. SGOT, SGPT and bilirubin are the bio-markers for liver functions (Martin *et al.*, 1981; Ronald and koretz, 1992; Mazumder, 1999). Alkaline phosphatase is a membrane bound enzyme and its inactivation leads to membrane damage of hepatic cells (Flora *et al.*, 1994). Increased Alkaline phosphatase is responsible for intra-and extra-hepatic disease.

Table. 4. presents the effect of PCC on serum, serum protein, albumin bilirubin, SGPT, SGOT and ALP in control and experimental group of rats. It shows the slightly increased level of serum total protein, albumin, and level of serum PCC treated rats. The increase level was 35.24% for total protein, 17% for albumin and 48 % for bilirubin. But, the SGPT, SGOT and ALP levels were decreased. The decrease level was 25 % for SGPT, 24 % for SGOT and 32% for ALP in serum PCC treated rats when compared to normal rats

These proteins are important liver function marker. According to Naganna (1989), increase in bilirubin is indicating the abnormal liver function which may be the results of higher synthetic function of the liver. Statistically no important data of bilirubin, another liver function indicator. This is indicating the normal liver function which is contradictory with the total protein and albumin observation. SGPT, SGOT and ALP content in the plasma, of rats were decreased very high significantly. Alkaline phosphatase is the marker enzyme for plasma and endoplasmic reticulum (Wright and Plummer, 1974; Shahjahan *et al.*, 2004) and its decrease indicates

the improved synthetic activity of liver, from the toxicological report of the serum parameters proved the safety of the drug. The toxicity of gold, mercury and sulphur was completely removed and the potency of the metals was only enhanced during the preparation on the drug. The detoxifying property is also attributed by the *Alovevera brobadensis* and Hibiscus extracts added during the preparation of the drug,

### KIDNEY FUNCTION

Kidneys are the chief organs for the excretion of wastes. Besides their excretory function, kidney function in a significant manner in the maintenance of internal environment of the body. The damaged kidneys cause an elevated Urea because the kidneys are less able to clear urea from the blood stream. Urea measures the amount of urea nitrogen, a waste product of protein metabolism in the blood. It is also useful to detect the function of kidney tissue. Urea is typically measured to assess kidney function (Mitchell *et al.*, 1972). Creatinine is also used to measure the filtration rate of the kidney. It is the indicators for the function of kidney (Gyton, 1991). Uric acid is a major contributor to total radical trapping capacity (TRAP) (Kharb and Singh 2004)

Table 2. represents the effect of PCM on kidney Creatinine, Urea and Uric acid levels in control and Drug treated rats. Creatinine and Urea levels were significantly decreased PCC treatment the decrease levels were 21% for creatinine and 27% for urea when compared to control rats. But the Uric acid levels was significantly increased 12% in PCM treatment.

Creatinine and urea content, major kidney function parameter, in the male plasma was decreased significantly but the content of uric acid were slightly changed in significant manner. This reduced creatinine and urea level might have results from the decreased synthesis or increased functional capacity of tubular excretion (Mitchell *et al.*, 1972; Zilva *et al.*, 1991)

There are significant changes in serum urea, creatinine and uric acid .Yet these values were proving the safety of the drug. There is an increase in uric acid levels which aids to the safety of the drug. Renal function test credits the safety of the drug. PCC did not accumulate in renal tissues which could be evidently seen by the urea creatinine and uric acid in serum.

### LIPID PROFILE

Atherogenicity with subsequent cardiovascular manifestations is one of the major causes of death and morbidity in the world (Raju and Binda, 2005). The important lipids whose elevations are implicated in these disease conditions are cholesterol and triacylglycerols. Lipids are transported as lipid-protein complexes called lipoproteins, which are classified based on their density and charges. The High-density Lipoprotein cholesterol (HDL) transports lipids out of blood cells to the liver, while the Low Density Lipoproteins cholesterol (LDL) mobilizes lipids against the cells and blood vessels. Triacylglycerols have been found to be elevated along with total cholesterol elevation. Therefore, elevated low-density cholesterol, triacylglycerols and total cholesterol with reduced HDL will enhance the development of atherosclerosis and related cerebrovascular disorders (Nwanjo, 2004). Figure.1. represents the level of lipid profile in PCM treated rats & Normal control rats. The value of TG, TC and LDL were significantly decreased. The decrease levels were 32.23 % for Triglycerides, 27.58 % for Cholesterol and 19.29 % for Low density lipoprotein in PCC treatment than normal control. But the HDL level was significantly increased. The increase level was 44 % in PCM treatment than normal control rat's serum.

The plants constituents (Lee *et al.*, 2000) reduced TG level and it could be suggested that PCM increased lipase activity which hydrolyzed TG. Among the lipids, increased blood level of TC and LDL as well as lowered level of HDL has been identified as contributors in the development of hyperlipidemia (Ross, 1999) which is the consequences of, in majority of the cases, diabetes mellitus (Pushparaj *et al.*, 2000; Pepato *et al.*, 2003; Sharma *et al.*, 1983). The elevation of lipid components is a risk factor for coronary heart disease (Mironova *et al.*, 2000). PCM may act as inhibitor for enzyme such as hydroxyl-methyl-glutaryl-CoA reductase, which is the key enzyme in de novo cholesterol biosynthesis as has been suggested for some plants earlier (Gebhardt and Beck, 1996; Eidi *et al.*, 2006). This reduction could be beneficial in improving lipid metabolism and complications in diabetes (Cho *et al.*, 2002) Abnormalities in serum lipids are associated with diabetes (Virella-Lopes and Virella, 2003; NCEP, 2002).

**Table 1: Effect of oral administration of PCM (3 mg/ kg body weight) on various parameters of liver Functions of rats' plasma**

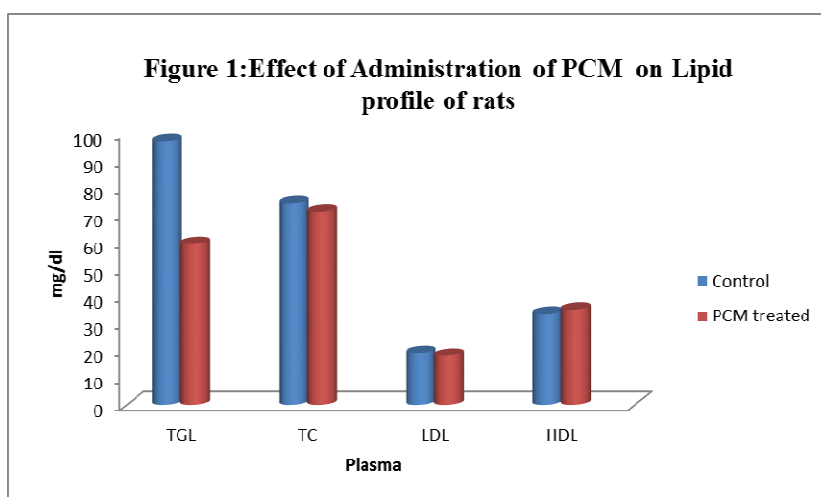
Parameters	Control	PCM Treated
Total protein	7.27±0.45	8.25±0.56**
Albumin	4.25±0.39	5.29±0.45**
Bilirubin	0.13 ± 0.01	0.02 ± 0.01***
SGPT	60.27 ± 0.13	55.54 ± 0.12***
SGOT	101.73 ± 0.31	93.75 ± 0.24***
ALP	43.56 ± 0.11	40.12 ± 0.08***

In the tables the statistical data are shown as: \* = p<0.05 = Significant, \*\* = p<0.01 = High Significant, \*\*\* = p<0.001 = Very High Significant

Table 2: Effect of administration of Poorna chandrodayam chendooram (3mg/ kg body weight) on various parameters of kidney functions of rats' plasma

Parameters	Control	
Creatinine	<b>0.95±0.12</b>	<b>0.75 ± 0.02*</b>
Urea	<b>65.86 ± 1.04</b>	<b>47.35 ± 0.20*</b>
Uric acid	<b>2.58 ± 0.06</b>	<b>2.90 ± 0.08*</b>

Values are expressed as mean ±S.D. for six rats. Comparisons were made between group I with II P<0.05 = Significant



## CONCLUSION

Interestingly, it is seen that PCC has steady decreased levels of urea, creatinine, SGOT, SGPT, ALP levels which reveal that fact that this drug may also be useful treatment of hepatic disorders and renal diseases. The myth that heavy metal cause toxicity is broken out in this study when the drug is properly prepared and given safe dosage during the duration of treatment. It is confirmed that Metal base drug PCC is a safe and effective drug .It is evident that the trial drug eliminate the toxic substances from the body and enhances the longevity of life.

## REFERENCES

1. Austin A., M. Jagadeesan and S. Subramanian. (2002) Toxicological studies of *Linga chendooram*: a Siddha Drug. Indian J.Pharm.Sci.; 64:53-58.
2. Doumas, B.T., W. A. Watson and H. G. Biggs, (1971). Albumin standards and measurement of serum-albumin with bromocresol green. Clin. Chim. Acta. 31(1): 87- 96.
3. Eidi, A., M. Eidi and E. Esmaeili, (2006). Antidiabetic effect of garlic (*Allium sativum L.*) in normal and streptozotocin-diabetic rats. Phytomedicine, 13 (9-10): 624-629.
4. Evelyn, K.A. and Malloy H. T.( 1938). Microdetermination of Oxyhemoglobin, Methemoglobin and Sulfhemoglobin in a single sample of Blood. J. Biol. Chem. 126, 655 -662.
5. Flora, S.J.S., Mathur, S., Mathur, R (1995). Effects of meso 2,3 -dimercapto succinic acid or 2,3 -dimercapto 1-sulfonate on beryllium induced biochemical alteration and metal concentration in male rats. *Toxicology*, 95, 167.
6. Friedewald, W.T., R.I. Levy and D.S. Fredrickson, (1972). Estimation of the concentration of Low-Density Lipoprotein Cholesterol in plasma, without use of the preparative ultracentrifuge, Clinical Chemistry, 18(6): 499-502.
7. Gebhardt, R. and Beck H. (1996). Differential inhibitory effects of garlic-derived organosulfur compounds on cholesterol biosynthesis in primary rat hepatocyte cultures. *Lipids*, 31(12):1269- 1276.
8. Guyton, C. (1991) Ed. Text book of Medical Physiology. Saunders. New York.
9. Halliwell, B. (1998). Albumin: an important extracellular antioxidant, *Biochem Pharmacol.*, 37, 569-571.

11. Hardy AD., H.H. Sutherland, R. Vaishnav and M.A. Worthing. A report on the composition of mercurials used in the traditional medicines in Oman. *J. Ethnopharmacol.* 1995; 49:17-22. 13.
12. Harper, H.A. (1991). Harper's review of biochemistry In: Murray, R.K., Mayes, P.A., Granner, D.K., and Rodwell, V.W. (eds). Large medical publication. London pp. 450-453.
13. Hazeena Begum V, Muthukumar P(2014). Phytochemical and free radical scavenging activity of Poorna chandrodayam chendooram (metallic herbal based drug). *The Journal of Phytopharmacology*, 3(6): 418-422
14. Zilva, JF Panmall P. R. and Mayne, P. D. (1991). *Clinical Chemistry in Diagnosis and Treatment*, England Clays Ltd., St. Ives Plc., England, 5th edition.
15. Kaplan, A.P. 1965. Urea nitrogen and urinary ammonia. In: *Standard Method of Clinical Chemistry*, Meites S., Academic Press Inc., New York, pp: 245 – 256.
16. Kharb, S. and Singh, V. (2004). Total free radical trapping antioxidant potential in normal pregnancy. *J Obstet Gynecol Ind.* 54(3), 249-50.
17. Kumar, A., A.G. Nair, A.V. Reddy and A.N. Garg. .Bhasmas: Unique ayurvedic metallic herbal preparations, chemical characterization. *Biol. Trace Elem. Res.* 2006; 109:231-254.
18. Lee, K.T., I.C. Sohn, D.H. Kim, J.W. Choi, S.H. Kwon and H.J. Park, 2000. Hypoglycemic and hypolipidemic effects of tectorigenin and kaikasaponin III in the streptozotocin-induced diabetic rat and their antioxidant activity in vitro. *Arch. Pharm. Res.*, 23: 461-466.
19. Mahdihassan, S. Cinnabar. Gold as a best alchemical drug of longevity called Makaradhwaja in india. *Am J Chin Med.* 1985; 13:93-108.
20. Martin, D.W. Jr., Mayes, P.A and Rodwell, V.W. (1981). Harper's review of biochemistry. 18th edn. (Lange medical publication California), 61.
21. Mazumder, U.K., Gupta, N., Chakrabarti, S. and Pal, D. (1999). Evaluation of hematological and hepato renal functions of methanolic extract of *Moringa oleifera* (Lam) root treated mice, *Ind J Expl Biol.*, 37, 612-614.
22. Mironova, M.A., R. L. Klein, G.T. Virella and M.F. Lopes-Virella, 2000. Anti-modified LDL antibodies, LDL-containing immune complexes and susceptibility of LDL to in vitro oxidation in patients with type -2 diabetes. *Diabetes*, 49:1033-1049.
23. Mitchell, F.L., and Watts, R. W. E. (1972). Renal function tests suitable for clinical practice. *Ann. Clin. Biochem.* 9, 1-20.
24. Mitchell, J. M., Jr. (1972), The Natural Breakdown of the Present Interglacial and its Possible Intervention by Human Activities, *Quatern. Res.*, 2, 436– 445.
25. Muthaliar, K.N.K and Uttamarayan K.S. (1987). *Siddha Pharmacopoeia*. Parinilayam, Chennai, pp:167-168.
26. Naganna, B. (1989). Plasma proteins. In: *Textbook of Biochemistry and Human Biology*, Talwar
27. NCEP (Third Report of the National Cholesterol Education Program) (2002). Expert panel on detection, evaluation and treatment of high blood cholesterol in adult (Adult Treatment Panel III) final report. *Circulation*, 106: 3143-3421.
28. Nwanjo, H.U. (2004). *Lipids and Lipoproteins in Biochemistry for Students of Pathology*. 1st Edn., Laudryman, Nigeria
29. Patel NG (1986). *Ayurveda: the traditional medicine of India*, in *Folk Medicine; The Art and the Science*, RP Steiner ed. American Chemical Society, Washington, DC, pp. 41–65.
30. Pepato, M.T., A.M. Baviera, R.C. Vendramini, M.P. Perez, I.C. Kettelhut and I.L. Brunetti, (2003). *Cissus sicyoides* (Princess wine) in the long term treatment of streptozotocin-diabetic rats. *Biotechnol. Applied Biochem.*, 37:15-20.
31. Plummer, D.T. (1971). *An Introduction to Practical Biochemistry*. McGraw-Hill, London, 2nd edn, pp: 144-145.
32. Raju, SM, Bindu M (2005). *Illustrated Medical Biochemistry*, 1st ed. Brjbasi Press Ltd. New Delhi, 152-153.
33. Ronald, L., and Koretz, M.D. (1992). Chronic hepatitis: Science and superposition. In: Gitnick, G (ed). *Current hepatology*. Mosby, Chicago II.
34. Ross, R. N., (1999). Atherosclerosis — An Inflammatory Disease. *Eng. J. Med.*, 340: 115-126.
35. Savrikar SS (2004) Use of metallic/mineral medicinal preparations in the management of disease, in *Proc. Seminar on Metals in Medicine; Ayurvedic and Modern View*, p. 16–18.
36. Shahjahan, M., K. E. Sabitha, J. M. and C. S. Shyamala-Devi, (2004). Effect of *Solanum trilobatum* against carbon tetrachloride induced hepatic damage in albino rats, *Indian J. Med. Res.* 120: 194-198
37. Sharma, M. K., A.K. Khare and H. Feroz, (1983). Effect of neem oil on blood glucose levels of normal, hyperglycemic and diabetic animals. *Indian Med. Gaz.*, 117: 380-383.
38. Srivastava L. M. and Moudgil, K. D., Prentice- Hall of India Private Ltd., New- Delhi, 2nd edn.; pp: 59 – 61.

39. Sudha, A., V.S. Murty and T.S. Chanda (2009). Standardization of metal based herbal medicines. *Am. J. Infect. Dis.* 5:193-199.
40. Suoboda RE. (1998). *Prakriti; Your Ayurvedic Constitution*, 2nd ed., Sadhana Publications, Bellingham, WA, pp. 169-174.
41. Thiagarajan, R (1992). Directorate of Indian Medicine and Homeopathy .Tamilnadu, Chennai. pp.134-144.
42. Tietz, N.W., E. L. Prude and O. Sirgard-Anderson, (1994). *Tietz Textbook of Clinical Chemistry*. ed. Burtis C. A. and Ashwood, E. R., W. B. Saunders Company, London, 2nd edn; pp: 1354 – 1374.
43. Virella-Lopes, M.F. and G. Virella, (2003). The role of immune and inflammatory processes in the development of macrovascular disease in diabetes. *Frontiers Biosci*, 8:750-768.
44. Wright, P.J. and D. T. Plummer (1974). The use of urinary enzyme measurement to detect renal damage caused by nephrotoxic compounds. *Biochem. Pharmacol.*, 23(1): 65-73.
45. Yeragi, S.G., Koli, A. and Yeragi, S. (2000). Effect of pesticide malathion on protein metabolism of the marine crab, *Uca marionis*. *J. Ecotoxicol. Environ Monot.*, 10, 59-62.

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#### CONFLICTS OF INTEREST

“The authors declare no conflict of interest”.

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