

EFFECT OF MONOCROTALINE AND SOME ANTIOXIDANTS ON PROTEIN CONTENT IN FEMALE ALBINO RATS

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ABSTRACT : The effect of monocrotaline and antioxidants (α -Tocopherol and ascorbic acid) on the protein content of the ovary and uterus. It shows that the administration of ascorbic acid and α -tocopherol per se even for 60 days did not cause significant change in the protein content of the ovary and uterus. When monocrotaline was administered at 10mg/kg for 10 and 20 days, no change was observed in the protein content of the ovary while the protein content of the uterus decreased significantly ($P<0.05$) in 20 days of treatment. When the administration of monocrotaline was increased for 40 and 60 days, protein contents were increased significantly ($p<0.05$). When ascorbic acid was administered alongwith monocrotaline, no significant changes were observed in the protein content of the ovary from 10 to 60 days and in uterus from 10 to 40 days.

Key words : Albino rat, monocrotaline, antioxidant, protein, ovary, uterus.

INTRODUCTION

Monocrotaline is one of the pyrrolizidine alkaloids that cause hepatotoxicity and also known for its fetotoxic effects (Amin *et al*, 2014). Earlier studies in the laboratories have shown that plants containing monocrotaline have been found to induce abortion and also induce severe reproductive disorders in cyclic and pregnant rats. To overcome these toxic effects, use of some antidotes or a protective agent is considered. (Patrick *et al*, 2012). Antioxidants like Vitamin-C (ascorbic acid) and Vitamin-E (α -tocopherol) are well known natural antioxidants, which are present in the body as such.

Although some elements like selenium, copper, lead and zinc have been used to encounter toxic effects (Goyer and Cherian 1979; Jamai *et al*, 2007) but α -tocopherol and ascorbic acid have shown very promising results. Agostino *et al* (2004) observed pulmonary hypertension and fibrosis in mammal. Therefore, it has been planned in the present study to select ascorbic acid and α -tocopherol as the antioxidants. Therefore, looking into the toxic nature of monocrotaline, it has been planned in the present proposal to find out the role of α -tocopherol and ascorbic acid in the restitution of induced toxicity by monocrotaline.

MATERIALS AND METHODS

Monocrotaline (Roth GmbH, Germany) was purchased. Antioxidants viz. α -Tocopherol and ascorbic

acid of AR grade were used.

LD₅₀ as observed in the present was found to be 500mg/kg (oral route). Various standard doses of monocrotaline were prepared on the basis of LD₅₀. Two different doses viz. 1/20th and 1/50th of LD₅₀ were prepared for the present study.

After 24 hours of last treatment the animals were sacrificed using light ether anesthesia and ovary and uterus from each rat were excised, freed from adhering tissue. These two organs are used for uterus and ovary biochemical estimation of protein (Lowry *et al*, 1951).

Adult cyclic female rats of Wistar strain weighing 120 -130g were collected from the departmental animal facility and they were caged separately in cages and were kept under standard uniform husbandry conditions of light (14L: 10D) and temperatures ($26 \pm 1^\circ\text{C}$). These animals were supplied with rat pelleted diet (Amrut Pvt. Ltd.) and water *ad libitum*.

Vaginal smear of each rat was examined daily in the morning at 10:00hrs. using methods of long and Evans (1992) with modifications. The animals which showed two consecutive normal regular cycles were selected and were divided into eight groups as shown in the Table 1. Animals under control group received vehicle only, whereas, animals under group-2 and 3 received ascorbic acid and α - tocopherol *per se* as mentioned in the table 1. Group-4 and 5, received monocrotaline at two different

Table 1 : Effect of monocrotaline and some antioxidants on protein content of the ovary and uterus in female albino rats.

Group	Treatment	Duration of treatment (Days)	Ovary mg/100mg	Uterus mg/100mg
1-	Control (Vehicles only)	-	11.5±0.77	12.0±0.62
2-	Ascorbic Acid(per se)	60	12.8±0.88	11.4±0.92
3-	α-Tocopherol(per se)	60	12.7±0.78	13.4±0.96
4-	Monocrotaline (25mg/kg)	10	7.92±0.45*	6.22±0.56*
		20	6.68±0.37*	4.80±0.52*
		40	5.10±0.34*	4.19±0.45*
		60	5.81±0.28*	4.70±0.43*
5-	Monocrotaline (10mg/kg)	10	9.1± 0.58	9.30±0.63
		20	7.79±0.74	7.81±0.35*
		40	7.16±0.49*	6.02±0.46*
		60	7.19±0.55*	6.50±0.51*
6-	Ascorbic Acid + Monocrotaline	10	8.53±0.64	7.41±0.63
		20	6.22±0.61	6.30±0.64
		40	5.30±0.40	6.10±0.41
		60	6.04±0.33	9.20±0.51*
7-	α-Tocopherol + Monocrotaline	10	6.50±0.50	8.88±0.57*
		20	6.95±0.62	8.10±0.68*
		40	8.2 ±0.53*	8.40±0.66*
		60	10.0±0.49*	9.80 ±0.74*
8-	Ascorbic Acid + α-Tocopherol + Monocrotaline	10	9.2±0.32	8.92±0.63*
		20	7.02±0.42	8.00±0.66*
		40	9.4±0.61*	8.50±0.70*
		60	11.1±0.75*	10.10±0.76*

Statistical analysis

* P value vs respective control < 0.05

doses for four different periods as shown in the table 1. Other group received conjoint treatment of monocrotaline and antioxidants.

After 24 hours of last treatments the animals were sacrificed using light ether anesthesia and uterus from each rat were excised, freed from adhering tissue. These two organs were studied for biochemical estimation of protein.

RESULTS

Table 1 shows the effect of monocrotaline and antioxidants (α-Tocopherol and Ascorbic acid) on the protein content of the ovary and uterus. It shows that the administration of ascorbic acid and α-tocopherol *per se* even for 60 days did not cause significant change in the protein content of the ovary and uterus, when compared with their respective control (P<0.05). Monocrotaline, when administered orally at 25mg/kg dose for 10 days protein content of the ovary and uterus shows significant decrease. Further administration of monocrotaline for 20, 40 and 60 days, significantly reduced protein content of the ovary and uterus when compared with their respective control (P <0.05). Similarly, when monocrotaline was administered at 10mg/kg for 10 and 20 days, no change

was observed in the protein content of the ovary while the protein content of the uterus decreased significantly (P<0.05) in 20 days of treatment. When the administration of monocrotaline was increased for 40 and 60 days, protein contents were increased significantly (p<0.05). When ascorbic acid was administered along with monocrotaline, no significant changes were observed in the protein content of the ovary from 10 to 60 days and in uterus from 10 to 40 days, while at 60 days protein content of the uterus increase significantly when compared with monocrotaline.

When α-tocopherol was supplemented in the monocrotaline induced rats, no recoupment was observed in the protein content of the ovary in 10 and 20 days, however when period of treatment was increased to 40 to 60 days, significant recoupment was observed. Protein content of the uterus increased significantly from 10 to 60 days in the monocrotaline induced rats supplemented with α-tocopherol. Conjoint treatment of ascorbic acid, monocrotaline and α-tocopherol showed better results as uterine protein content was recouped even in 10 days treatment (P vs respective control <0.05).

DISCUSSION

Barethia (1996) and Pathak (2003) have reported that extract of *Crotalaria juncea* when administered to cyclic adult rats causes severe alterations in the biochemical constituents of the ovary and uterus of rats protein contents.

Administration of monocrotaline has been reported to decrease RNA and DNA synthesis therefore, it is expected in the reproductive organs to the administration of monocrotaline inhibit the key enzyme involved in the protein synthesis. Administration of antioxidants per se could not show any change but when these are conjointly administered the protein contents recouped to normal. Thus, it is apparent that antioxidants in combination act synergistically and through its inherent property they regulate generation of free radicals and prevent further deterioration.

Protein reduction may also be due to degradation of tissues protein to amino acids which in turn were fed into tricarboxylic acid (TCA) cycle through amino-transference pathway to cope us with high enevary demands for the detoxification and elimination of toxic substances (Syversan, 1981). Protein is mobilized to produce glucose. This instant energy is made available animals by process of gluconeogenesis.

REFERENCES

- Agostino Molteni, Betty L. Herndon, Arif Kamal, Wiliam J. Castellani, Sarah Reppert, Yuan Xue, Joshua Humbehr and Richard C. Baybutt (2004) Effect of the antioxidant α -tocopherol in an experimental model of pulmonary hypertension and fibrosis: Administration of monocrotaline. *Nutrition Research*. **24**, 707-720.
- Amin K A, Hashem K S, Al-muzafor H M and Taha E M (2014) Oxidative hepatotoxicity effects of monochrotaline and its amelioration by lipoic acid. S-adenosyl Methionine and Vitamin E.J. *Complement Integr. Med.* **11**, 35-41.
- Barethia R (1996) Antifertility effect of *Apium graveolens* and *Cratolaria juncea* Linn. In female rats. *Ph.D. Thesis*, Jiwaji University, Gwalior.
- Goyer R A and Cherian M G (1979) Ascorbic acid and EDTA treatment of lead toxicity in rats. *Life Sci.* **24** (5), 433-438.
- Jemai H, Mesaoudi I, Chaouch A and Kerkeni A (2007) Protective effect of zinc supplementation on blood antioxidant defence system in rat exposed to cadmium. *J. Trace Elem. Med. Biol.* **21**, 269-273.
- Long J A and Evans H M (1922) *The oestrous cycle in the rat and its associated phenomena*. University of California Press, Barely.
- Lowry O H, Rosenbaugh N J, Farr A L and Randall R J (1951) Protein measurement with folin-Phenol reagent. *J. biol. Chem.* **193**, 265 - 275.
- Pathak L (2003) Reproductive toxicity of seed extract of *Crotalaria juncea* in female rats. *Ph. D. Thesis*, Jiwaji University, Gwalior.
- Patrick J D, Sally D P and Ulrike L (2012) Role of oxidative stress and antioxidants in ovarian toxicity. *Biol. Repro.* **36**, 27-29.
- Syversen T L M (1981) Effects marcy on protein synthesis in vitro. *Acta. Pharmacol. Toxicol.* **49**, 422-426.