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Efficacy of Calcium Borogluconate and Lime Water Therapy in *Anagallis arvensis* Intoxicated Cattle

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ABSTRACT

Anagallis arvensis is a common weed seen in agricultural fields in Marathwada region of Maharashtra state. This weed contains different poisonous principles like glycosides, volatile oil, saponin (anagallin), tannin, and oxalates. In scarcity of fodder, cattle in some regions had exposure to *Anagallis arvensis* weed causing oxalic acid toxicity and nephrotic syndrome. Twenty *Anagallis arvensis* affected cattle were randomly distributed in two groups (Group-I; n=10 and Group-II; n=10). *Anagallis arvensis* intoxicated cattle of Group-I were subjected to primary therapy of calcium borogluconate @ 450 ml per cattle slow I/V, whereas, Group II cattle were treated with combination of calcium borogluconate @ 450 ml per cattle and lime water (1 kg of lime mixed to make 10 litres of water and one litre of this solution was administered twice a day) therapy. Based on improvement in clinical, haematological, biochemical parameters and recovery; parentral calcium borogluconate and oral lime water therapy along with standard supportive regimen was found highly efficacious in the treatment of *Anagallis arvensis* toxicity.

Keywords: *Anagallis arvensis,* toxicity, therapeutic, calcium borogluconate, lime water

Anagallis arvensis (Family-Primulaceae) commonly called blue pimpernel is an intercrop weed observed particularly in turmeric, wheat, maize, cotton etc crops in Marathwada region of Maharashtra state. It is a small perennial herb with square stems, branching from the base, leaves opposite, shiny, stalkless and grows in damp soil, gardens, waste places and in semi-exposed sites. This weed contains different poisonous active principles like glycosides, volatile oil, saponin (anagallin), tannin and oxalates in their edible parts (Everist, 1981).

The literature on toxic effects of *Anagalis arvensis* feeding to cattle and its therapeutic management is scanty. The present paper, therefore, reports toxicity of *Anagalis arvensis* feeding to cattle and its therapeutics.

MATERIALS AND METHODS

The present clinico-therapeutic study was conducted on twenty (20) clinical cases of cattle affected with oxalic acid toxicity due to *Anagallis arvensis*. The cattle were randomly distributed in two groups. The affected cattle of Group-I (n=10) were subjected to calcium borogluconate @ 450 ml per cattle slow IV, as a primary therapy while as Group- II (n=10) cattle were treated with combination of calcium borogluconate @ 450 ml slow IV per cattle and oral lime water therapy (1 kg of lime mixed to make 10 liters of water and one liter of this solution (10%) was administered twice a day). In addition to primary therapy the affected cattle of both the groups were subjected for common supportive therapeutic regimen comprising DNS @ 25 ml/kg b wt. IV, Inj. Bivinal plus (Alembic pharmaceuticals) 10 ml IM, Inj. Flunimec (Zydus animal health) @ 1.1 mg/kg body weight IM, and Inj. Furosemide @ 2 mg/kg BW; IM. Ten (n=10) apparently healthy cattle of similar age group with similar managemental practices from same area were selected and served as healthy control (Group–III) and were utilized for comparative study. The clinical, haematological and biochemical parameters were analysed on day 0 (before therapy) and on day 5th and 10th after therapy. The comparative efficacy of the treatment regimen was evaluated based on the improvement seen in clinical, haematological and biochemical parameters.

RESULTS AND DISCUSSION

The clinical signs exhibited in *Anagallis arvensis* intoxicated cattle were anorexia, depression, staggering gait, lameness of hind limbs with recumbency, swelling at perineal region, dehydration, impaction of rumen, constipation with hard faeces, dilated pupil, frequent attempts to urinate and frothy urination (Fig. 1-4). There was flaccidity of smooth muscles, and few cases exhibited sternal recumbency with head turned into their flank (Fig. 3). The affected cattle manifested significant (p<0.01) hypothermia, tachycardia, tachypnoea and decreased ruminal motility (Table 1). Similar signs where recorded by Dhoot *et al*. (1995), Sadekar *et al*. (1995), Singh *et al*. (1995), Rivero (2001), Al-Sultan *et al*. (2003), Knight and Walter (2003), Al-Mujalli (2008), Al-Snafi (2015), in oxalate poisoning in ruminants. These general clinical signs observed in the present *Anagallis arvensis* intoxicated cattle could be attributed to hypocalcaemia and depressed cellular energy metabolism.

Analysis of haematological parameters in cattle affected with *Anagallis arvensis* intoxication revealed significant (p<0.01) decrease in haemoglobin (Hb), Packed Cell Volume (PCV), Total Erythrocyte Count (TEC), while significant (p<0.01) increase in Total Leucocyte Count(TLC), Neutrophil Count and eosinophil count. There was significant (p<0.01) reduction in lymphocyte count of affected cattle. Thus neutrophilic leucocytosis with eosinophilia and lymphopenia was observed in the present oxalate toxicosis. Following therapy, all the haematological parameters were improved on day 5th and 10th in corresponding group I and II respectively (Table 2). However magnitude of improvement and extent towards normalcy was more in calcium borogluconate - lime water group (Group II). Similar observations were reported by Al-Sultan *et al*. (2003), Al-Mujalli (2008), Radostits e*t al*. (2007), Digraskar *et al*. (2010), Al-Snafi, (2015). Decrease in red cell parameters could be attributed to reduced cell energy metabolism (Knight and Walter, 2003), and cytotoxic effects of *Anagallis arvensis* (El-Garieb, 1990; Al-snafi 2015) and depressed haemopoietic activity due to oxalate toxicosis. As regard to alterations in white cell parameters, elevated TLC count observed in the present investigation (Table 2) has been also reported by Singh *et al*. (1995), Digraskar *et al*. (2010) and Mohammad *et al*. (2011) and may be attributed to body's response to oxalate toxaemia, and metabolic disturbances caused by uraemia.

The blood biochemical profile of Anagallis arvensis toxic cattle indicated significant (p<0.01) hypoproteinemia, hypoalbunemia, hypoglobulinaemia, reduced A:G ratio, hypocalcemia, moderate increase in inorganic phosphorus and significant (p<0.01) increase in AST and ALT activities (Table 3). The dysproteinemia observed in the present study is in agreement with the observations of Dhoot *et al*. (1995), Smith (2011), Digarskar *et al*. (2010), Radostits *et al*. (2007), Rood *et al*. (2014). and could be attributed to leakage of protein through glomuruli filtration and its disintegration through degenerating tubules consequent to oxalate nephrosis and damage to hepatic tissues by calcium oxalate crystals. Hypocalcemia is a characteristic and consistent biochemical finding on oxalate toxicosis and has been documented by several earlier authors (Littledike *et al*., 1976; Dhoot *et al*., 1995; Singh *et al*., 1995; Kerr and Kelch, 1998; Radostits *et al*., 2007; Al-Mujali, 2008; Rahman *et al*., 2011; Rood *et al*., 2014). The increased activities of AST and ALT could be attributed to cell necrosis of different tissues including liver and kidney (Singh *et al.,* 1995) marked damage to vascular tissues (Radostits *et al*., 2007). Elevated ALT specifically confirmed hepatopathy due to Anagallis arvensis toxicity (Al-Mujali, 2008).

Anagallis arvensis toxicity caused higher concentration of calcium oxalates in blood and subsequent characteristic oxalate nephrosis which resulted in renal insufficiency and accumulation of sensitive renal indicators creatinine and urea nitrogen in the blood, observed in current investigations (Table 4). Significant increase in the level of creatinine may be regarded to glomerular damage and excessive muscular catabolism due to calcium oxalate crystals (Al-Sultan *et al*., 2003) calcium oxalates are practically insoluble and when present in higher concentration has propensity to precipitate in renal tubules, causing nephrosis and hence kidney is the first target organ affected due to oxalic acid toxicity. Similar to the present observations Singh *et al*. (1995), Dhoot *et al*. (1995), Kerr and Kelch (1998), Radostits *et al*. (2007), Rood *et al*. (2014) authors documented significant azotemia in oxalate

Fig. 1: Hard constipated faeces

Fig. 2: Dilatation of Pupil

Fig. 3: Sternal recumbency

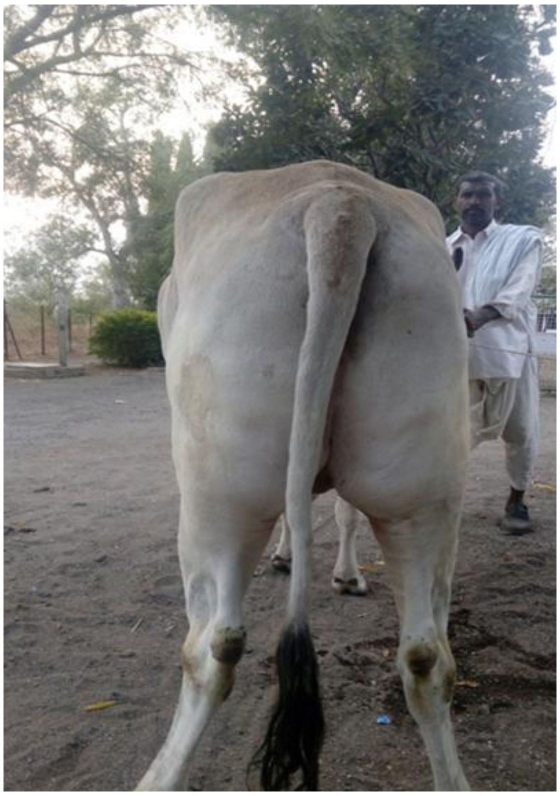

Fig. 4: Perirenal oedema

Table 1: Clinical observations of healthy and cattle affected with *Anagallis arvensis* toxicity before (BT) and after therapy (AT)

Sl. No.	Parameters	Gr. I (Calberol)				Gr. II (Calberol and lime water)				Gr.III (Healthy Control)
		BT (0 day)	AT (5^{th} day)	AT (10^{th} day)	CD	BT (0 day)	AT (5^{th} day)	AT (10^{th} day)	CD	
1	Temperature (°F)	98.93^{c} ± 0.23	99.23^{bc} ± 0.31	99.75^{b} ± 0.21	**At 5%** - 0.67 **At 1%** - 0.90	98.17^{r} ± 0.11	99.55^{q} ± 0.20	99.95^{q} ±0.19	**At 5%** - 0.48 **At 1%** - 0.64	101.11$^{a\,p}$ ± 0.15
2	Heart rate (bpm)	74.9^{a} ± 0.95	71.0^{b} ± 0.71	67.7^{c} ±0.55	**At 5%** - 2.06 **At 1%** - 2.77	75.9^{p} ± 0.50	70.0^{q} ± 0.76	67.3^{r} ± 1.03	**At 5%** - 2.14 **At 1%** - 2.87	68.7$^{b\,r}$ ±0.57
3	Respiratory rate (no./min)	44.0^{a} ± 0.7	34.4^{b} ± 0.88	30.7^{c} ±0.55	**At 5%** - 1.88 **At 1%** - 2.52	45.0^{p} ± 0.61	33.0^{q} ± 0.57	28.8^{r} ± 0.55	**At 5%** - 1.59 **At 1%** - 2.14	24.6$^{d\,s}$ ± 0.47
4	Ruminal motility (no./5min)	1.8^{c} ± 0.2	2.9^{b} ± 0.23	4.3^{a} ± 0.21	**At 5%** - 0.56 **At 1%** - 0.76	1.5^{r} ± 0.16	3.2^{q} ± 0.2	4.4^{p} ± 0.16	**At 5%** - 0.48 **At 1%** - 0.64	4.8$^{a\,p}$ ± 0.13

a, b, c superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group I and III

p, q, r, s superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group II and III

Table 2: Haematological observations of healthy and cattle affected with *Anagallis arvensis* toxicity before (BT) and after therapy (AT)

Sl. No.	Parameters	Gr. I (Calberol)				Gr. II (Calberol and lime water)				Gr. III (Healthy Control)
		BT (0 day)	AT (5^{th} day)	AT (10^{th} day)	CD at 5%/1%	BT (0 day)	AT (5^{th} day)	AT (10^{th} day)	CD at 5%/1%	
1	Hb (g %)	8.19 ± 0.13^{c}	8.89 ± 0.09^{bc}	9.22 ± 0.16^{b}	0.58/ 0.78	8.65 ± 0.09^{r}	9.18 ± 0.14^{r}	9.7 ± 0.17^{q}	0.56/ 0.75	12.09$^{a\,p}$ ± 0.28
2	PCV (%)	27.9 ± 1.01^{b}	28.35^{b} ± 0.49	29.18^{b} ± 0.56	1.95/ 2.61	27.4 ± 0.47^{r}	28.3 ± 0.3^{r}	31.6 ± 0.49^{q}	1.28/ 1.72	36.6$^{a\,p}$ ± 0.49
3	TEC($\times 10^{6}$/µl)	3.78 ± 0.08^{d}	4.11 ± 0.11^{c}	4.51 ± 0.14^{b}	0.33/ 0.44	3.88 ± 0.04^{s}	4.27 ± 0.10^{r}	4.87 ± 0.16^{q}	0.30/ 0.41	6.43$^{a\,p}$ ± 0.08
4	TLC(10^{3}/µl)	10.19 ± 0.24^{a}	9.79 ± 0.09^{a}	8.83 ± 0.10^{b}	0.48/ 0.64	11.46 ± 0.20^{p}	10.13 ± 0.31^{q}	9.18 ± 0.11^{r}	0.38/ 0.51	8.34$^{c\,s}$ ± 0.08
5	N (%)	59.1 ± 1.61^{a}	55.4 ± 2.39^{a}	45.00 ± 0.78^{b}	4.39/ 5.88	61.30 ± 0.97^{p}	55.5 ± 0.58^{q}	44.5 ± 0.85^{r}	2.23/ 3.00	43.4$^{b\,r}$ ± 0.63
6	L (%)	34.4 ± 0.88^{d}	38.7 ± 1.35^{c}	50.1 ± 0.56^{b}	2.72/ 3.65	32.10 ± 0.72^{s}	37.0 ± .0.66^{r}	48.9 ± 0.67^{q}	2.07/ 2.77	56$^{a\,p}$ ± 0.81
7	M (%)	2.80 ± 0.35	2.8 ± 0.48	2.1 ± 0.40	—	3.0 ± 0.44	4.2 ± 0.35	3.7 ± 0.51	—	3.1 ± 0.43
8	E (%)	4.2^{a} ± 0.82	3.0^{ab} ± 0.73	2.8^{b} ± 0.48	1.58 (5%)	3.6 ± 0.68	3.3 ± 0.53	2.9 ± 0.37	—	2.1^{b} ± 0.34

a, b, c superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group I and III

p, q, r, s superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group II and III

Table 3: Biochemical observations of healthy and cattle affected with *Anagallis arvensis* toxicity before (BT) and after therapy (AT)

Sl. No.	Parameters	Gr. I (Calberol)				Gr. II (calberol and lime water)				Gr. III (Healthy Control)
		BT (0 day)	AT (5^{th} day)	AT (10^{th} day)	CD at 5%/1%	BT (0 day)	AT (5^{th} day)	AT (10^{th} day)	CD at 5%/1%	
1	TSP (g/dl)	6.18 ± 0.01 ^{d}	6.31 ± 0.02 ^{c}	6.45 ± 0.02 ^{b}	0.10/ 0.14	6.10 ± 0.06 ^{s}	6.30 ± 0.02 ^{r}	6.50 ± 0.04 ^{q}	0.14/ 0.19	6.80 $^{a p}$ ± 0.06
2	Albumin	2.89 ± 0.01 ^{c}	3.09 ± 0.04 ^{b}	3.1 ± 0.04 ^{b}	0.11/ 0.51	2.90 ^{q} ± 0.05	3.01 ^{q} ± 0.05	3.02 ^{q} ± 0.05	0.14/ 0.19	3.33 $^{a p}$ ± 0.04
3	globulin	3.29 ± 0.05^{b}	3.26 ± 0.06^{b}	3.29 ± 0.05^{b}	0.15/ 0.21	3.10 ± 0.09 ^{q}	3.20 ± 0.04 ^{q}	3.51 ± 0.08 ^{p}	0.22/ 0.30	3.52 $^{a p}$ ± 0.07
4	A:G	0.87 ± 0.01	0.95 ± 0.02	0.92 ± 0.02	—	0.92 ± 0.03	0.88 ± 0.02	0.80 ± 0.03	—	0.90 ± 0.02
5	Calcium	5.18 ± 0.41 ^{d}	6.29 ± 0.40 ^{c}	7.63 ± 0.13 ^{b}	0.94/ 1.27	5.54 ± 0.33 ^{s}	7.05 ± 0.26 ^{r}	8.21 ± 0.19 ^{q}	0.78/ 1.04	10.59 $^{a p}$ ± 0.28
6	inorganic Phosphorus	5.14 ± 0.09	5.05 ± 0.08	5.01 ± 0.11	—	5.15 ± 0.06 ^{p}	4.96 ± 0.04 ^{qr}	4.89 ± 0.04 ^{r}	0.15/ 0.20	5.07^{pq} ± 0.05
7	SGOT	114.4 ± 2.17 ^{a}	87.20 ± 2.4 ^{b}	64.00 ± 0.9 ^{c}	5.05/ 6.77	121.09 ± 1.7 ^{p}	80.60 ± 3.3 ^{q}	63.70 ± 0.88^{r}	5.64/ 7.56	60.7 $^{d r}$ ± 0.8
8	SGPT	104.3 ± 2.57 ^{a}	54 ± 3.51 ^{b}	22.5 ± 0.88 ^{c}	6.43/ 8.63	99.1 ± 0.92 ^{p}	41.8 ± 0.8 ^{q}	23.0 ± 1.21 ^{r}	2.69/ 3.61	18.6 $^{c s}$ ± 0.65

a, b, c superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group I and III

p, q, r, s superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group II and III

Table 4: Kidney function test observations of healthy and cattle affected with *Anagallis arvensis* toxicity before (BT) and after therapy (AT)

Sl. No.	Parameter	Group I				Group. II				Gr.III (Healthy control)
		0	5^{th}	10^{th}	CD	0	5^{th}	10^{th}	CD	
1	BUN	176.5 ± 4.41 ^{a}	56.5 ± 1.65 ^{b}	34.3 ± 0.68 ^{c}	At 5% - 9.08 At 1% - 12.18	154.5 ± 13.6 ^{p}	66.74 ± 6.89 ^{q}	30.31 ± 2.36 ^{r}	At 5% - 22.19 At 1% - 29.76	19.41$^{d r}$ ± 1.23
2	CREA	17.64 ± 1.21 ^{a}	9.38 ± 0.24 ^{b}	4.01 ± 0.35 ^{c}	At 5% - 1.84 At 1% - 2.47	16.05 ± 1.27 ^{p}	9.09 ± 0.18 ^{q}	3.32 ± 0.26 ^{r}	At 5% - 1.88 At 1% - 2.52	1.05 $^{d s}$ ± 0.05

a, b, c superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group I and III

p, q, r, s superscripts indicates statistical significance at $p<0.05$ (5%) and at $p< 0.01$ (1%) within the rows of Group II and III

toxicity (James *et al.*, 1967; Dhoot *et al.,* 1995; Knight and Walter, 2003).

CONCLUSION

Based on magnitude of improvement in clinico haematological, biochemical parameters, kidney function parameters and extent of their reversal towards normalcy, parentral calcium borogluconate and oral lime water therapy along with standard supportive regimen was found highly efficacious in the treatment of Anagallis arvensis toxicity in cattle.

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