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Research Article

CHEMOTHERAPEUTIC EFFECT OF TRYPANOCIDAL DRUGS IN NATURALLY OCCURING EQUINE TRYPANOSOMOSIS

VIJENDRA KUMAR PAL[1], AMIT SINGH[1], SANJAY KUMAR[2] and RAJENDER KUMAR[2]*
[1]Department of Veterinary Parasitology, College of Veterinary Science and Animal Husbandry, ANDUAT, Kumarganj, Ayodhya-224229 and [2]ICAR-National Research Centre on Equines, Sirsa Road, Hisar-125001, India

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ABSTRACT

The chemotherapeutic effect of three commercially available trypanocidal drugs was evaluated by analyzing the level of haematological and serum biochemical indices between *Trypanosoma evansi* naturally infected and naturally reared infection free equines. Eighteen microscopically positive (micro haematocrite centrifugation technique and Giemsa stained) indigenous breed of horses and showing clinical signs were randomly divided into three treatment groups of six animals each. They were compared with six others apparently healthy animals which came negative for trypanosomosis by microscopic examination.The infected animals of group T1 treated with quinapyramine di-methylsulphate @ 5.0 mg/kg body weight subcutaneously, group T2 with isometamidium chloride @ 1.0 mg/kg body weight intramuscularly and group T3 with diminazene aceturate @ 7.0 mg/kg body weight intramuscularly, respectively. A significant ($P < 0.05$) alteration in haematological and biochemical parameters *viz.*, Hb, PCV, TEC, lymphocytosis, ALT, GGT, TSP, albumin and blood glucose were noticed in all infected groups when compared to infection free control group T4 in the commencement of the present study. Haemato-biochemical parameters were reverted significantly in both the groups T1 and T2 except group T3 on day 7 to day 14 post-treatment. Outcome of the present study depicted that quinapyramine di-methylsulphate and isometamidium chloride were found more effective in treatment of *T. evansi* infection than diminazene aceturate in naturally infected equines.

Keywords: Diminazene aceturate, Isometamidium chloride, Quinapyramine di-methylsulphate, *Trypanosoma evansi*

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Trypanosomosis (Surra) caused by *Trypanosoma evansi* is one of the most important disease of equines and other domestic mammals. It is transmitted mechanically by different species of Tabanid flies (Desquesnes *et al.*, 2013). In India, it is found as endemic mostly in north and north-western region with seroprevalence (19.69%) of the parasite in Uttar Pradesh state (Kumar *et al.*, 2013). This disease causes significant economic losses to farmers in terms of morbidity, mortality and infertility in animals etc. Annual economic deprivation associated with animal trypanosomosis in India was estimated as INR 44,740 million (Kumar *et al.*, 2017). In India, treatment of animal trypanosomosis depends predominantly on the commercially available trypanocidal drugs i.e. diminazene aceturate, quinapyramine and isometamidium chloride (Ponnudurai *et al.*, 2015). For implementation an effective control of equine trypanosomosis through therapeutic or prophylactic compounds, there is a need to evaluate the efficacy of available antitrypanosomal drugs in equines. Therefore, the objective of the present study was to evaluate the therapeutic efficacy of commercially available drugs against naturally infected equine trypanosomosis recruited from eastern part of Uttar Pradesh, India.

MATERIALS AND METHODS

The present study was conducted in the Department of Veterinary Parasitology, College of Veterinary Science and Animal Husbandry, Kumarganj, Ayodhya, Uttar Pradesh.

Recruitment of study animals: An Institutional Animal Ethics Committee (IAEC) for animal experiment of Acharya Narendra Deva University of agriculture and Technology, Kumarganj, Ayodhya, Uttar Pradesh granted an approval (IAEC/CVSc/2/P-32/2020/26) for the present study. Subjects were recruited at one point (September, 2020) from five villages in the District Gazipur situated in the Eastern plain zone, Uttar Pradesh, India. Based on the clinical sign like pale mucus membrane, dull, lymphadenopathy, high rectal temperature (>104 °F), petechial haemorrages, oedema and anaemia the animals were suspected for surra. Blood samples of the suspected animals collected separately in EDTA vials and in clot activator vials and were examined by blood smear and microhaematocrit centrifugation technique (MHCT). A total of 18 animals suspected to be suffering from trypanosomosis on the basis of clinical signs were found positive for trypanosomosis by blood smear examination and MHCT.

Experimental design: Animals were randomly divided into three groups (T1, T2 and T3) and treated with quinapyramine di-methylsulphate (Triquin, vetoquinol) @ 5.0 mg/kg body weight subcutaneously, isometamidium chloride (Surral, Alembic) @ 1.0 mg/kg body weight intramuscularly and diminazene aceturate (Berenil, Intervet India Pvt. Ltd.) @ 7.0 mg/kg body weight intramuscularly, respectivelyon day 0 (pre-treatment). Treated groups were compared with control group (T4) of six apparently healthy animals and found negative by microscopic examination. Treated animals were also administered with injection Inferon and Vitamin B complex as supportive therapy and monitored clinically after treatment. Blood

*Corresponding author: rkg.nrce@gmail.com

samples on day 0, 3,7,14 and 21 post treatment were collected to observe haematological and biochemical alterations to monitor the efficacy of the drugs.

Among haematological values total erythrocytes count (TEC $\times 10^6/\mu l$), total leukocytes count (TLC $10^6/\mu l$), haemoglobin (Hb %), packed cell volume (PCV %) and differential leucocytes count (DLC %) was performed by standard procedure.

Serum was separated from the blood and used for the estimation of total serum protein (TSP), serum albumin and serum glucose. Several enzymes and biochemical metabolites like aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyl transferase (GGT), creatinine, bilirubin total (BIT), bilirubin indirect (BID), serum cholestrol, serum urea nitrogen and uric acid (UA) were estimated at different time intervals of both treated and untreated healthy animals at standard wavelength.

Stained blood smear examination: Giemsa stain blood smear examination was done as per the standard method. Finally, slide was examined under microscope (Olympus) for the presence of *T. evansi*.

Statistical analysis: The results generated as mean±SE from present study was calculated for each parameter and significant difference between means evaluated by analysis of variance (ANOVA) using Graphpad prism software (version 8.0.2) for Turkey's multiple comparison test and the value of $P< 0.05$ was considered as statistically significant.

RESULTS AND DISCUSSION

The therapeutic efficacy of three trypanocidal drugs was evaluated by analyzing the levelof haematological and biochemical parameters between *T. evansi* naturally infected and naturally reared infection free equines. Animals from groups T1, T2 and T3 were established microscopic positive (MHCT, Giemsa staining) for *T. evansi* (Fig. 1) prior to the administration of drugs. They were observed microscopically for *T. evansi* on day 3 onwards post treatment for clearance of infection (Fig. 2). This was depicted that selected trypanocidal drugs have good therapeutic efficacy against equine trypanosomosis. Singh *et al.* (2012) reported that the *T. evansi* was not seen in equine blood (MHCT, Giemsa staining) after administrating single dose of quinapyramine di-methylsulphate and isometamedium chloride on day 7 post-treatment.

Haematological observations: Haematological estimations revealed that the mean value of Hb, PCV, TEC and lymphocyte count were significantly ($P < 0.05$) lower on day 0 in all infected groups as compared to control group (Table 1). The overall mean values of these variables in T1 and T2 were found to be continuously increased significantly ($P < 0.05$) and attained normal values from day 14 PT (Table 1). However, in T3, the haematocrit values remained significantly lower than the control group. The lower level of Hb, PCV, and TEC variable were observed because of release of haemolysin by the parasite, erythrophagocytosis and mechanical destruction of RBCs by the parasite flagellar movement (Bal *et al.*, 2014). Damaged cell membrane has also been combined with adhesion of erythrocytes, platelets and reticulocytes to the body surface of *trypanosoma* via sialic acid receptors leading to damages to erythrocytic cell membranes (Shehu *et al.*, 2006). Previous analysts reported lymphocytosis occurs in animal trypanosomiasis (Abd El-Baky and Salem, 2011). In addition, Sulaiman and Adeyemi (2010) suggested to lymphocytosis may be due to hyperplasia of lymphoid tissue in the acute phase of trypanosomosis, while in chronic infection, the immune system becomes depleted of lymphoid cells.

As per previous investigations, all selected trypanocides have the capability to *T. evansi* clearance from the peripheral blood of infected animal within 3 days post treatments (Singh *et al.*, 2012; Aregawi *et al.*, 2015). In the current investigation, haematological parameters were reverted to normallevels on day 7 to day 14 post-treatment in both T1 and T2 groups indicated that the quinapyramine di-methylsulphate and isometamidium chloride were capable to complete clearance of *T. evansi* from peripheral blood circulation. Similar monitoring was made by Singh *et al.* (2012) in equine trypanosomosis. Contrary to above observations, the haematological variables of the animals treated with diminazene aceturate were not reverted to normal level on post treatment.

This have happened because of the drug would not be able to complete parasites clearance. The status of haematological variables was indicated that diminazene aceturate @ 7.0 mg/kg body weight arrived to be effective up to 7 days post-treatment. Earlier investigators were reported that single dose of diminazene aceturate was not effective against trypanosomosis in horses, mule, dog, cat and buffalos (Howes *et al.*, 2011). The reason for inefficacy of diminazene aceturate was proposed either due to significant levels of resistance developed (Zhang *et al.*, 1992) or inadequate doses to clearance the parasites (Masocha *et al.*, 2007).

Changes in biochemical indices: The result of different serum enzymes appears from variety of tissues especially hepato-biliary system such as serum ALT and GGT levels were recorded significantly high ($P < 0.05$) in all infected groups on pre-treatment when compared to control (Table 1). A significant ($P < 0.05$) higher level of ALT enzyme could be due to tissue breakdown and inflammation in the host

Table 1

Comparative analysis of haemato-biochemical variables in horses pre and post treatment against *T. evansi* infection

Variables	Day	Group C (n=6)	Group T1 (n=6)	Group T2 (n=6)	Group T3 (n=6)
Haemoglobin (g %)	0	$^{A}10.73 \pm 0.31$	$^{B}4.80 \pm 0.29^{a}$	$^{B}4.73 \pm 0.21^{a}$	$^{B}4.63 \pm 0.18$
	3	$^{A}10.33 \pm 0.48$	$^{B}5.00 \pm 0.16^{a}$	$^{B}4.93 \pm 0.22^{a}$	$^{B}5.00 \pm 0.31$
	7	$^{A}10.53 \pm 0.24$	$^{AB}9.33 \pm 0.56^{b}$	$^{BC}8.16 \pm 0.63^{b}$	$^{C}6.36 \pm 0.32$
	14	$^{A}9.90 \pm 0.42$	$^{A}9.56 \pm 0.42^{b}$	$^{A}8.70 \pm 0.44^{c}$	$^{B}6.06 \pm 0.19$
	21	$^{A}10.16 \pm 0.39$	$^{A}9.86 \pm 0.30^{b}$	$^{A}8.93 \pm 0.5^{d}$	$^{B}5.86 \pm 0.15$
Packed cell volume (%)	0	$^{A}35.16 \pm 1.01$	$^{B}20.66 \pm 0.88^{b}$	$^{B}19.33 \pm 0.88^{b}$	$^{B}20.00 \pm 1.12^{b}$
	3	$^{A}33.16 \pm 0.47$	$^{B}27.00 \pm 0.85^{c}$	$^{B}25.00 \pm 1.46^{b}$	$^{B}23.16 \pm 1.68^{ba}$
	7	$^{A}34.33 \pm 0.55$	$^{A}35.83 \pm 0.54^{a}$	$^{AB}32.16 \pm 1.77^{a}$	$^{B}28.16 \pm 1.07^{ac}$
	14	$^{A}33.66 \pm 0.88$	$^{A}34.33 \pm 0.80^{a}$	$^{A}33.83 \pm 1.85^{a}$	$^{B}23.83 \pm 0.65^{bc}$
	21	$^{A}32.66 \pm 1.47$	$^{A}34.66 \pm 0.88^{a}$	$^{A}32.83 \pm 0.87^{a}$	$^{B}23.33 \pm 0.98^{b}$
Total erythrocyte count ($\times 10^{6}/\mu l$)	0	$^{A}7.30 \pm 0.35$	$^{B}2.61 \pm 0.24^{b}$	$^{B}2.70 \pm 0.27^{c}$	$^{B}3.05 \pm 0.13^{a}$
	3	$^{A}7.58 \pm 0.87$	$^{B}4.21 \pm 0.22^{b}$	$^{B}3.55 \pm 0.15^{c}$	$^{B}3.81 \pm 0.21^{ab}$
	7	$^{A}B7.99 \pm 0.44$	$^{A}8.43 \pm 0.47^{a}$	$^{B}C6.28 \pm 0.51^{b}$	$^{C}5.11 \pm 0.24^{bc}$
	14	$^{A}7.76 \pm 0.48$	$^{AB}6.76 \pm 0.56^{a}$	$^{A}8.46 \pm 0.32^{a}$	$^{B}4.95 \pm 0.20^{ac}$
	21	$^{A}7.38 \pm 0.19$	$^{A}7.63 \pm 0.29^{a}$	$^{A}7.90 \pm 0.49^{ab}$	$^{B}5.06 \pm 0.31^{bc}$
Absolute lymphocyte count ($\times 10^{3}/\mu l$)	0	$^{A}4.06 \pm 0.49$	$^{B}2.46 \pm 0.23^{b}$	$^{A}B2.84 \pm 0.36^{b}$	$^{A}B2.78 \pm 0.20$
	3	$^{A}4.79 \pm 0.037$	$^{AB}3.36 \pm 0.25^{ba}$	$^{B}3.21 \pm 0.26^{ba}$	$^{B}2.94 \pm 0.25$
	7	$^{A}4.49 \pm 0.29$	$^{A}4.12 \pm 0.22^{a}$	$^{A\,B}3.64 \pm 0.21^{ba}$	$^{B}2.22 \pm 0.09$
	14	4.24 ± 0.34	$3.60 \pm 0.64b^{a}$	4.65 ± 0.31^{a}	3.24 ± 0.33
	21	$^{A}4.51 \pm 0.21$	$^{A}4.29 \pm 0.14^{a}$	$^{A}4.38 \pm 0.30^{a}$	$^{B}2.64 \pm 0.20$
Alanine aminotransferase (U/L)	0	$^{D}32.95 \pm 0.52$	$^{C}56.61 \pm 3.03^{a}$	$^{B}80.15 \pm 1.17^{a}$	$^{A}93.43 \pm 1.73^{a}$
	3	$^{D}35.48 \pm 2.14$	$^{BC}47.25 \pm 3.15^{c}$	$^{B}53.68 \pm 3.2^{c}$	$^{A}71.13 \pm 2.33^{b}$
	7	$^{B}34.73 \pm 1.39$	$^{B}38.56 \pm 0.90^{bc}$	$^{AB}40.85 \pm 1.77^{b}$	$^{A}51.33 \pm 4.36^{c}$
	14	$^{B}35.96 \pm 0.81$	$^{B}31.31 \pm 1.56^{b}$	$^{B}34.51 \pm 1.52^{b}$	$^{A}67.81 \pm 2.93^{b}$
	21	$^{B}33.10 \pm 1.56$	$^{B}32.21 \pm 1.40^{b}$	$^{B}40.85 \pm 1.77^{b}$	$^{A}70.76 \pm 1.36^{b}$
Gamma glutamyl transferase (U/L)	0	$^{B}21.81 \pm 3.93$	$^{A}36.18 \pm 1.64^{a}$	$^{A}33.10 \pm 1.00$	$^{AB}30.63 \pm 0.92$
	3	$^{B}22.05 \pm 3.46$	$^{A}36.95 \pm 2.20^{a}$	$^{AB}30.76 \pm 0.59$	$^{AB}31.13 \pm 0.91$
	7	25.51 ± 1.89	25.30 ± 1.75^{b}	28.16 ± 1.75	25.68 ± 2.20
	14	21.15 ± 0.88	21.30 ± 0.84^{b}	27.51 ± 1.68	25.80 ± 0.61
	21	20.88 ± 2.27	22.43 ± 0.61^{b}	24.3 ± 0.79	30.03 ± 1.31
Total serum protein (g/dl)	0	$^{A}5.95 \pm 0.20$	$^{B}1.61 \pm 0.67^{d}$	$^{B}2.56 \pm 0.44^{c}$	$^{B}2.94 \pm 0.92^{c}$
	3	$^{A}5.72 \pm 0.13$	$^{B}1.84 \pm 0.30^{dca}$	$^{B}2.83 \pm 0.47^{c}$	$^{B}2.87 \pm 0.25^{c}$
	7	$^{A}5.95 \pm 0.38$	$^{B}3.75 \pm 0.3^{cb}$	$^{AB}4.44 \pm 0.26^{cba}$	$^{AB}3.92 \pm 0.22^{cba}$
	14	6.06 ± 0.18	5.11 ± 0.24^{ba}	5.12 ± 0.11^{ba}	3.73 ± 0.10^{ba}
	21	5.65 ± 0.66	5.84 ± 0.88^{a}	5.21 ± 0.27^{a}	3.42 ± 0.16^{a}
Albumin (g/dl)	0	$^{A}2.66 \pm 0.08$	$^{B}0.52 \pm 0.16^{b}$	$^{B}0.95 \pm 0.14$	$^{B}1.03 \pm 0.22$
	3	$^{A}2.47 \pm 0.12$	$^{B}0.95 \pm 0.17^{b}$	$^{B}1.02 \pm 0.03$	$^{B}1.21 \pm 0.08$
	7	2.37 ± 0.17	1.53 ± 0.10^{a}	1.66 ± 0.06	1.69 ± 0.15
	14	2.07 ± 0.15	1.75 ± 0.13^{a}	1.68 ± 0.13	1.71 ± 0.20
	21	2.34 ± 0.16	1.93 ± 0.17^{a}	1.61 ± 0.60	1.50 ± 0.10
Serum glucose (mg/dl)	0	$^{A}71.51 \pm 2.77$	$^{B}30.53 \pm 1.61^{b}$	$^{B}24.85 \pm 0.69^{b}$	$^{B}32.28 \pm 1.99^{b}$
	3	$^{A}68.01 \pm 1.60$	$^{B}26.60 \pm 3.71^{b}$	$^{B}23.56 \pm 1.23^{b}$	$^{C}37.41 \pm 1.76^{ba}$
	7	$^{A}66.91 \pm 1.73$	$^{B}46.01 \pm 1.26^{a}$	$^{B}39.90 \pm 1.28^{a}$	$^{B}44.48 \pm 2.88^{a}$
	14	$^{A}65.16 \pm 1.48$	$^{B}50.46 \pm 0.93^{a}$	$^{B}44.66 \pm 2.57^{a}$	$^{B}42.21 \pm 3.24^{ba}$
	21	$^{A}70.03 \pm 1.89$	$^{B}51.33 \pm 1.27^{a}$	$^{B}C43.78 \pm 1.83^{a}$	$^{C}38.88 \pm 2.29^{b}$

A, B or a, b to be specifically mentioned for a row or a column.

infected with trypanosomosis (Sivajothi *et al.*, 2015) or centrilobular degeneration as a result of the hypoxia and severe oxidative stress induced by *T. evansi* infection in equines (Abd El-Baky and Salem, 2011). All the treated groups were regained normal level of ALT on day 7 post-treatment except group T3. This was depicted that administered drugs would have positive action and no any toxic effect during clearance of parasites (Ngure *et al.*, 2008). The level of GGT was recorded significantly ($P < 0.05$) higher initially and reverted normal range on day 7 post- treatment in all infected groups (Table 1). Elevation in GGT is suggestive of tissue breakdown and inflammation in the animal's body or might be due to lysed trypanosomes at different stages of the infection (Takeet and Fagbemi, 2009).

The mean value of TSP and serum albumin in control group was recorded significantly higher as compared to all infected groups on pre-treatment (Table 2). Substantial increase inserum albumin was noticed in all treated groups and observed no significant alteration on day 14 which is the compliance with the detecting of Hota *et al.*, 2019.

Blood glucose level was increased in all *T. evansi* infected groups (Table 1). Hypoglycemia recorded due to rapid consumption of the blood glucose by the parasites for their metabolism during infection (Bal *et al.*, 2014). Follow up the treatment, the level of blood glucose was prevailed steadily elevated in the termination of the present study, but no one group was achieved blood glucose collateral to the control group. However, uninterrupted elevation was recorded in group T1 and T2 depicted that both drugs (Quinapyramine and Isometamidium chloride) have yielded better therapeutic efficacy against *T. evansi* infected equines. Similar finding was recorded by Singh *et al.* (2012). All other serum biochemical variables were statistically not significant from the control group.

Outcome of haematological and biochemical observations indicated that quinapyramine di-methylsulphate and isometamidium chloride were found more effective against *T. evansi* than diminazine aceturate in naturally infected equines. Diminazine aceturate did not completely clear *T. evansi* infection at the recommended dose. This might be due to degree of drug resistance developed against this parasite. This field based study contributes preliminary information about the therapeutic efficacy of the commercially available trypanocidal drugs in Eastern Uttar Pradesh.

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