Clinico-therapeutic studies on sarcopticosis in camels (*Camelus dromedarius*)

ऊँटों में सारकोप्टिकोसिस पर शयनिक – चिकित्सीय अध्ययन

THESIS

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INTRODUCTION

Camel belongs to genus Camelus in the family camelidae under the suborder Tylopoda and order Artiodactyla. There are two types of camels, the one humped or dromedary camel, (Camelus dromedarius) the two humped camel or bactrian camel (Camelus bactrianus). Camelus dromedaries means "running camel". Camelus is Latin word for camel and Dromeus is Greek word for runner. Camelus dromedarius also has many common names such as Camel, one-humped camel, dromedary camel and Arabian camel.

The developing countries have 98.1 per cent of the total world camel population. India has third highest camel population in the world (1.52 million, FAO, 2004). Indian camel population is mostly confined to the North Western part of the country. According to state Animal Husbandry 18th tentative census of 2007, the camel population is 4.30 lac, out of which, Bikaner possesses 0.54 lac camels.

Camel is an important component of the desert ecosystem where the flora of usually marginal land can meet the need of human food and energy. Recurring drought in arid and semiarid areas have decimated many livestock species, however dromedary is still surviving in large numbers due to its outstanding tolerance to the rugged climate of high temperature, water deprivation, endurance for hunger and scarcity. As far as utility of this ungulate in arid desert tract is concerned, it is referred, as excellent means of carrying load, transportation, agriculture and defence services in condition where other animals are scared and failure. Besides, these its hide, wool and even meat are important by-products which contribute significantly towards the rural economy.

The skin surface acts as anatomic and physiologic barrier between the animal and environment. During lifetime skin has its own functions and even after death the utility of skin remains. Camelids like other livestock are exposed to a range of skin affections. Bacteria, Viruses, Parasites and fungi cause the skin affections. The ectoparasite directly or indirectly causes a great diversity of health problems. The mite Sarcoptes scabiei *is the cause of sarcoptic mange, which is regarded as one of most prevalent and serious camel disease (Lodha, 1966; Higgins, 1983).*

*Camel mange is sometimes considered the most important disease of dromedaries after trypanosomiasis. It is often ranked second to trypanosomiasis in importance to all the disorders in dromedary camels (Pegram and Higgins, 1992). There are several skin diseases which may mimic sarcoptic mange. These are ringworm, contagious skin necrosis (*Dermatophilus congolensis*), infestation with other ectoparasites causing skin problems including chorioptic mange (*Chorioptes spp.*), demodectic mange (*Demodex spp.*) and psoroptic mange (*Psoroptes spp.). *The skin affections may be* Staphylococcus *dermatitis, endocrinal dermatopathy, inhalant or food allergies (Rosychuk,1989),*

irritant dermatitis associated with contact to abrasive surfaces when lying down (Rosychuk,1989), camel pox, particularly the papule and scab formation stages, and idiopathic hyperkeratosis (associated with zinc responsive dermatoses in Bactrianus camel).

Mange is highly contagious and debilitating skin disease of camels, which can spread to herdsmen, or others associated with infected animals. The mite may be transmitted directly by contact or indirectly through objects, such as saddles, harnesses, utensils, bedding and even tree trunks. It tends to spread more quickly during cold weather, when hair coat usually grow long and animals huddle together more often.

The incidence and mode of transmission and disease pattern vary with season and location. Some workers have reported higher incidences during winter months (Lodha 1966, Rathore and Lodha 1973). On the other hand, Higgins (1984) found a higher prevalence in Saudi Arabia during hot summer months. Raisinghani and Kumar (1990) recorded infection throughout the year with comparatively higher incidences between December to April.

Any camelid regardless of sex and age may be affected by Sarcoptes scabiei*. However, young and aged camels are more prone probably due to lowered body defences (Kumar et al 1992). Animals in poor condition are more prone to infection (Lodha 1966; Higgins, 1983 and 1984). Stress, age, malnutrition, overcrowding, poor skin condition, long hair coat and worm burden have also been suggested as important predisposing factors. Debilitating conditions like Surra or Tuberculosis also precipitate the disease. Low plane of nutrition plays an important predisposing role (Kumar et al. 1992). The lesions of mange mainly occur on the head, neck, flank and in the inguinal region (Mathur, 2000).*

Against sarcopticosis many drugs viz. organochlorines, organophosphorous compounds and synthetic pyrethrins (Singh and Gahlot. 2000) have been tried and evaluated with variable degree of success. But repeated use of these drugs sometimes leads to various toxic and very often residual effects. Drug resistance contributes significantly and collectively to the problems to a degree of leaving only a limited choice of drugs for use. The disease is obstinate, not easily amenable to common acaricides and needs repeated application. Ivermectin, due to its potency, safety and ease of administration, is considered an efficient alternate drug (Raisinghani et al; 1989). Recently another long-acting new endecticide, doramectin was found to exert wide-spectrum of activity against ectoparasites (Logan et al;1993) and is successfulll in single dose treatment of mange mites in sheep (Bates et al; pigs (Arends et al; 1999).

Looking to the severity of sarcopticosis in camels in this area clinical studies have been proposed to give emphasis especially on therapeutics along with haemato-biochemical parameters. The objectives of the proposed investigation were as under:

1. *To study the clinical manifestations in camels suffering from sarcopticosis.*
2. *To study the pre and post- treatment hematological parameters viz. Haemoglobin, packed cell volume, total erythrocyte count, total leukocyte count, differential leukocyte count and total eosinophilic count in mange affected camels.*
3. *To study pre and post- treatment biochemical parameters, viz. serum glucose, serum total protein, serum albumin, serum globulin, A: G ratio, serum zinc, serum creatinine and serum urea in mange affected camels.*
4. *To compared therapeutic efficacy of ivermectin and doramectin in camels suffering from sarcopticosis.*

REVIEW OF LITERATURE

Pertinent literature regarding various parameters in relation with sarcopticosis has been reviewed under the following main headings.

Introduction

Kouba (1964) reported various animal disease problems in the Mongolians Peoples Republic. He found that mange and ectoparasitism were common skin diseases in camels.

Lodha (1966) reported that sarcoptic mange is a highly contagious skin disease of camels and occur thought the state of Rajasthan. The infected camel spends most of the part of their time in biting and scratching the affected parts, stops feeding and is thrown out of work. Thus, this disease is of great economic importance.

Rathor and Lodha (1973) studied seasonal, age-wise incidence and intensity of sarcoptic mange in camels of Rajasthan. They studied 540 camels in three districts over a period of three years. Sarcoptic mange was diagnosed in 11 per cent of camels kept under good conditions (in army) and in 95 per cent of village camels kept under poor conditions. The highest incidence were found in the camels of 16 years of age and above (95.45 per cent). In age group of 1-5 years, the incidences were 85 per cent. Likewise, the incidence was reported to be 75 percent, in the camels of 11-15 years age group. The minimum seasonal incidence of mange was 17.45 per cent in winter and 19.12 per cent in rainy season was also an important finding. Average number of mites per square centimeter of skin was 330, while their eggs were 264.

Higgins (1983) reported that after surra, mange is the most feared and widespread disease affecting the Arabian camel. The disease is highly contagious and debilitating. He surveyed 28 nomadic herds of dromedaries in north-eastern Saudi Arabia in winter; not a single animal showed signs of acute mange, 98 showed inactive mange lesions of which seven had widespread chronic mange. Lesions were most common on the neck, in the axillae and inguinal region and around the tail and head. The disease reaches its peak in summer when temperature is highest and grazing is poor; while in winter mites are less active in feeding and egg laying. Spread of the disease in summer is aided by the fact that animals are in close contact, herded around water holes.

Rutagwenda (1984) studied important camel diseases in Northern Kenya. Trypanosomiasis was detected by parasitaemia in 7.7 per cent camels in a herd of 174 dromadaries, while serum antibodies were detected by indirect haemagglutition in 79 per cent camels. It was caused by a member of the brucei group probably T. evansi*. Tick burdens were high on camels not treated with acaricides.* Hyalomma dromedarii*,* Rhipicephalus *and* Hyalomma truncatum *were mainly confined to caudal and perineal regions. Sarcoptic mange was common in calves.*

Higgins (1995) reported that sarcoptic mange caused by Sarcoptes scabiei var. cameli *is a serious contagious and debilitating disease affecting both dromedary and bacterian camels.*

Chauhan (1986) surveyed 283 apparently healthy dromedaries in Rajasthan and Haryana. In a skin examination of 150 camels which showed symptoms of pruritus, 32 (21.3 per cent) were positive for sarcoptic mange (caused by Sarcoptes scabiei).

Nayel and Abu Sharma (1986a) examined 33,000 dromedaries during a 2 year survey in Kassala and in the Red Sea, Nile and Northern province of Sudan.18,090 had sarcoptic mange. It was not found in riding or racing animals, but in working dromedaries the incidence was as high as 80 per cent, while in moving herds it was 52.6 per cent.The incidence was highest at the times of the year when dromedaries crowed together; 59 per cent in winter and 55 per cent in the rainy season; in summer it fell to 43 per cent. Most infected animals were well fed and in good condition.

Fassi–Fehri (1987) found that gastrointestinal helminthosis, mange and trypanisomiasis are, by far, the wide spread diseases in camel. Whereas, Schillinger (1987) described transmission of mange from camel to man.

Arlian et al. *(1989) described that sensory physiology and behavior experiments have indicated that all life stages of* S. scabiei *perceive and respond to host and non-host environmental stimuli. Mites perceived host body temperature and body odour and sought their sources to burrow when dislodged from the host. These responses were most pronounced in adults and least pronounced at the larval stage. Therefore, direct contact between hosts is not the only means of transmission. In addition, all life stages were strongly photosensitive and visible to ultraviolet light. In a light gradient, mites sought the most intensely illuminated area. The mite body, mite secretions and faecal material were antigenic and stimulated the host's T and B cell immune system. SDS-PAGE profiles of extracts produced from mite's bodies contained many soluble proteins. The sera of some scabies-infested patients contained IgE type antibodies specific for some of these antigens.*

Tika Ram et al. *(1991) reported 8 clinical cases of human scabies contacted from camels possibly for the first time. Examination of mites recovered from human lesions revealed* Sarcoptes scabiei *morphologically identical to* S. scabiei var. cameli*. The history, epidemiological findings, clinical pictures and lesions indicated zoonotic potential of the camel mite.*

Dioli and Stimmelmayer (1992) reported that sarcoptic mange caused by the itch mite Sarcoptic scabiei var. cameli. *The infection is recognized both as an acute and chronic debilitating disease causing the affected animals a lot of stress and discomfort. Infected camel may stop grazing and milk production may show a rapid fall.*

Basu et al. *(1996) conducted a survey on the incidence of* Sarcoptes scabiei var. cameli *infection in 200 camels (dromedaries) in an abattoir in Nigeria, demonstrated an infection rate of 72 per cent. It was noticed that 5 boys aged 13- 15 years who processed the camel meat also got infected.*

Egbe Nwiyi and Chaudhari (1996) examined sample of 469 adult camels (dromedaries) from Maiduguri, Nigeria between January 1991 and December 1993 for infestations of Sarcoptes scabiei var. cameli*. Mites were recorded in 62 per cent of camels in the hot seaon (March- October) and 32 per cent in the cold season (November-February).*

*Agab and Abbas (2001) investigated 15 camel herds comprising a total of 3731 dromedaries in Sudan for the most common diseases and health problems. It was found that mange (*Sarcoptes scabies*) was the most prevalent disease in the study area (31.36 per cent). Most of the camels found positive during summer (63.2 per cent) and winter (58.2 per cent) were lightly parasitized, whereas most found to be infected during autumn (58.6 per cent) were heavily parasitized.*

Mehta et al. *(2002) observed that camel is a major source of biological energy in the desert and is still being used by the poor people in the villages and cities to earn their livelihood. So they conducted a survey to see the socioeconomic condition of the camel keeper and health problems of Jaisalmeri camel. Mange was reported to be the major health problem (39.73 per cent) in Jaisalmeri camel.*

Mathur (2004) recorded sarcopticosis in 11.76 per cent cases in camels of either sex, age and breeds in 5 districts of Rajasthan. The mites were identified as Sarcoptes scabiei var. cameli *from the skin scrapings of affected camels.*

Muhammad et al. *(2006) reported that the commonest parasites, which affected dromedary camels, are sarcoptic and psoroptic mites.*

*Mange in camel (*Camelus dromedaries*) is almost invariably sarcoptic type, which is caused by* Sarcoptes scabiei var. cameli*. It is a widespread contagious debilitating disease, which causes serious economical losses to camel owners. As far as treatment of the disease is concerned, a lot of work has been done in India and abroad. The mange infestation has been routinely treated by different organochlorine and organophosphate compounds (Radstits et al. 2007)*

Parsani et al. *(2008) reported that among ectoparasitic infestations, sarcoptic mange caused by* Sarcoptes scabiei var. cameli *is an emerging and serious problem in camels in India.*

Mouchira and Khalid (2009) reported that sarcoptic mange is an important disease engendering significant morbidity and mortility in wild, domestic and farmed animals. Scabies is caused by the ectoparasitic mite Sarcoptes scabiei *which burrows in to the host epidermis. Scabies in dromedaries is caused by* Sarcoptes scabiei var. cameli.

Temperature, pulse and respiration

Lease (1927) reported the mean basal body temperature of one humped camels at morning hours 36.52^{0}C as against 37.96^{0}C at evening hours. Normal pulse rate in camel ranged between 32-44 per minute in the morning and 26-50 per minute in the evening. The frequency of respiration In normal healthy camel was 5-12 per minute.

Schmidt-nielson et al. *(1957 and 1963) observed diurnal variations in rectal temperature of one humped camels during summer and winter month. During summer months, the body temperature may exceed by 6 ^{0}C in the evening hours (40.6 ^{0}C) than a minimum of 34.2 ^{0}C in cool morning hours. In winter, when the external temperature was more moderate, the diurnal variations were about 2 ^{0}C only. These variations in body temperature were considered to be significant in the water economy of the camel, like other species.*

Bhatt et al. *(1960) recorded diurnal variations in rectal temperature, respiration frequency and heart rate of camels during the month of May in the morning and evening. Values for these parameters were 97.9 ^{0}F and 100.0 ^{o}F, 7.5 and 9.0 rate/minute and 33 and 36 rate/minute, respectively.*

Schmidt-Nielson et al. *(1967) reported that the respiration rate of the camel increases as its body temperature elevated from 35 to 41^{o}C.*

Joshi et al. *(1981) studied the rectal temperature, pulse and respiration rates in relation to estrus in female camels and found that the average body temperature, respiration and heart rate were 36.9 ^{o}C, 7.2 and 45.1 rate/minute, respectively during oestrus.*

Sardari Lal (1988) reported mean ± SE values of temperature, pulse and respiration rate in 5 apparently healthy camels. The values recorded during morning were 97.04 ± 0.37 (^{0}F), 33.8 ± 1.85 (rate/ minute) and 5.8 ± 0.37 (rate/min), respectively. While the values recorded during evening were 100.07 ± 1.10 (^{0}F), 41.6 ± 1.21 (rate/minute) and 7.8 ± 0.37 (rate/minute), respectively.

According to published annual report of National Research Centre on camel, Bikaner (1990-91), there was increase of rectal temperature by 7.32 per cent between the two ploughing i.e. before (37.08 ^{o}C) and after (39.76 ^{o}C) ploughing.

According to Pandey (1993) the physiological characteristics like rectal temperature, pulse and respiration are the most important indices of health and disease and are easily affected with change in the climatic conditions, physiological state of the animal, condition of internal and external stress and disease. Knowledge about the normal basal values of these parameters under different physiological state is essential to distinguish a healthy animal from the diseased one.

El-Hassan and Assad (1996) reported that dromedaries tend to maintaine their body temperature under muscular activity stress relatively close to that at rest by activation of evaporated cooling mechanism through the respiratory tract and skin.

Raghbendra et al. (1997-98) recorded respiration, pulse rate and rectal temperature before and after giving race of 1km in a desert kaccha tract. Respiratory frequency, pulse rate and temperature before race was 7.66 ± 0.21 per minute, 57.67 ± 1.36 per minute and 37.08 ± 0.04 ^{o}C, respectively. The respiratory frequency and pulse rate on racing increased by 21.5 per cent and 57 per cent, respectively.

Sarwar et al. *(1998) studied the influence of sex, age and location and/or pregnancy on some physio-chemical characteristics of dromedaries in summer and reported that irrespective of sex and age, the 56 camels had the mean ± SE values for rectal temperature, pulse and respiration rate as 99.63 ± 0.14 (97.8 –101.8^{o}F), 43.46 ± 1.09 (31-72 beats/min) and 11.00 ± 0.03 (7-16/min), respectively. It was concluded that location and /or pregnancy did not have a significant affect on the parameters studied.*

Dongre (2000) reported mean ± SE values of temperature, pulse and respiration rate in 10 apparently healthy adult male dromedaries as 37.28 ± 0.17 ^{o}C, 43.40 ± 0.59 rate/min and 6.80 ± 0.19 rate/min, respectively.

Mali (2002) reported mean ± SE values of temperature, pulse and respiration rate in apparently healthy control camels as 98.50 ± 0.20 ^{o}F, 44.30 ± 0.74 rate/min and 6.9 ± 0.48 rate/min, respectively.

Rathore (2006) recorded mean ± SE temperature, pulse and respiration at the 0 day of the experiment in 8 apparently healthy camel to be 96.97 ± 0.19 (96.4 - 97.8)°C, pulse 43.75 ± 0.95 (41 - 49) rate/minute, and 12.37 ± 0.49 (10 - 14) rate/minute.

Kumar (2007) recorded mean ± SE temperature, pulse and respiration in 15 apparently healthy camels to be 97.36 ± 0.23°F, 41.86 ± 0.35 rate/min and 8.4 ± 0.28 rate/minute.

Chaudhary (2008) recorded mean ± SE values of temperature 98.94 ± 0.03 ºF, pulse 44.10 ± 0.27 rate/minute and respiration 7.10 ± 0.18 rate/ minute, in 10 apparently healthy camels, respectively, while working during clinical studies on digestive disorders in camels.

Clinical manifestations

The incubation period of sarcoptic mange in camel is believed to be around 2-3 weeks (Lodha, 1966; Higgins 1983). Rathore (1971) reported that the initial lesions of sarcopticosis usually start on the ventral surface but sometimes at the leg or just above the foot pad. The camel starts scratching and biting at the lesions, which often spread rapidly to the sheath or flank. This results in spread of infection to lips and face. Lesions may also start around the chest pad and axillary regions. The camel endeavors to scratch with the toes of the hind limb, thus spreading the infection to the toes and upwards on the limbs. The hump region is rarely affected. Similar lesions on most of these sites have been reported by Hafez (1994).

Higgins (1983) reported that disease is often sudden in onset. Small nodules first appear and these can easily passed unnoticed. As the mites pierce the skin to suck lymph and to feed on young epidermal cells, intense irritation develops. Biting, scratching and nibbling are present. The irritation leads to an exudative, dermatitis, crusting and hair loss. The disease often starts on the medial aspects of the thighs, inguinal rigion, neck and flank and in severe cases

may spread all over the body. Pruritis may become severe and in such cases, the camel rapidly loses condition within 2-3 weeks in untreated cases, the acute inflammatory reaction gives way to the more chronic state. Excess keratinisation and proliferation of connective tissue leads skin to become thick and wrinkled, often covered with a fine powdery coating. Cracks with some haemorrhages may also be found.

Raisinghani and Kumar (1990) reported that in chronic cases loss of hair, scab formation keratinisation and proliferation of connective tissue lead to thickening of the skin, which became corrugated with mites deeply penetrated in the skin tissues. At this stage the skin gave a sandy appearance with a chalk like covering.

Tuzer et al. *(1991) diagnosed* S. scabiei var. cameli *infestation in three dromedaries and a donkey housed together in winter of 1986-87. The lesions occupied whole surface of body of one of the dromedaries and the head, neck, leg and some parts of bodies of two other dromederies. In the camel, intense itching, anorexia, weight loss, scurf, loss of hair, sin cracks, wrinkles and skin encrustations upto 1cm. thick were observed.*

Kumar et al. *(1992) reported that stress, age, malnutrition, overcrowding, poor skin condition long hair coat and worm burden all are important predisposing factors for camel mange. Young or aged camels are more prone to infection, probably reflecting lowered body defences. Nomadic camels appear particularly more susceptible which may be due to a low plane of nutrition and high worm burden. Debilitating conditions such as surra or tuberculosis also precipitate the disease.*

Gorakhmal et al. *(2000) observed clinical sings of mange-infested animals, which include pruritus, alopecia, scab formation and cutaneous hypersensitivity with extreme lesions on the ventral surface of the body, facial region and limbs.*

Al-Rawashdeh et al. *(2000) reported that sarcoptic mange is a contagious parasitic skin disease, it include clinical signs resulting from the development, multiplication and pathogenic action of the mites at the skin surface and in the stratum corneum.*

Mathur (2000) studied cutaneous ectoparasitoses in camels in 5 districts of Rajasthan. In camels suffering from sarcopticosis grossly papules, crust and eruptions were observed on the head,neck, flank, and in the inguinal region. The affected area showed alopecia, corrugation and grayish colouration of skin.

Ravindran et al. *(2000) reported that the highest prevalence of scabies is in the months of August associated with low atmosphere temperature in camels.*

Al Saad et al. *(2000) examined 30 Arabian dromedaries in laboratory showing signs of mange and identified sarcoptic mange. Animals showed signs of itching, pruritis, loss of hair, dryness of skin and restlessness. Lesions were concentrated mainly on trunk, tail, neck, face and limbs and frequently involved more than one area.*

The most affected part of body appeared to be skin irrespective of it's location. The only difference observed over the body surface was in the occurrence of lesions with relatively more density on root of tail, flank, inner, interior and posterior surface of thighs, axillary and occular region. The symptoms such as itching, reddening, biting, cracks, bleeding, thickening and wrinkling et., were observed. These changes may be attributed to the collective action/reaction of the body to its internally secreted various amines (Chauhan, 2001).

Dixit et al. *(2002) recorded persistence and occurrence of lesions of mange all over body surface. Main affected sites were root of tail, flank, inner, interior and posterior surface of thighs, axillary and occular region etc. hump was less affected part of the body. Occurrence of the symptoms such as itching, reddening, biting etc. may be attributed to the reaction of the body to its internally secreted various amines and movement of mites to different layers of skin, forcing the animal for intermittent rubbing against hard objects resulting in falling of hair and thickening of skin due to keratinization and proliferation of connective tissue.*

Wernery and kaaden (2002) reported that the first sign of mange in camels are small hyperemic papules often appearing on the medial aspects of thighs or inguinal region, the head and neck, medial areas of flanks, udder and shoulder. Within a few weeks the acute disease may develop to a chronic stage, there is hyperkeratosis, thickening, fissured, and corrugated skin appearing like a dried cracked field of clay.

Kinne, J and Wernery, U. (2003) described the experimental introduction of a mange-infected camel into a clean herd, with subsequent observations of the resulting effects on the herd. Twelve days after the introduction, 2 camels developed pruritus, followed by alopecia with papule development. One week later all four trial camels had contracted mange.

Parmar and Singh (2005) studied camels, which are naturally affected with sarcoptic mange-mite. Clinically affected camels exhibited pruritus, restlessness, emaciation, and alopecia with thick, wrinkled skin over the neck, medial aspect of thigh and brisket region and oozing of serous fluid and blood.

Shelly et al. *(2007) reported that acariotic mange due to* sarcoptes scabiei *belong to the family sarcoptidae and are highly contagious and burrowers.*

Mouchira and Khalid (2009) carried out a study to describe the clinical and pathological feature of 14 cases, divided into 10 dromedaries and 4 llamas diagnosed as scabies, caused by sarcoptic scabiei var cameli *in one farm of Western Riyadh, Kingdom of Saudi Arabia. Clinical symptoms like pruritis, pyoderma and Histologically hyperkeratosis, parakeratosis and dermatitis were seen.*

Haemato-biochemical examination

Salahledin et al. *(1979) reported the average values with range of some blood parameters in 96 clinically healthy camels as Hb 11.1 (range 7.8-15.9 g/100ml), PCV 30 (range 25-34%), TEC 7.83 (range 7.12-11.76 × $10^6/mm^3$), TLC 13.26 (range 11.5-16.5 × $10^3/mm^3$) and the values for DLC were neutrophils 51%, lymphocytes 40%, monocytes 4% and basophils 0.5% respectively. According to Ibrahim et al. (1981) decrease total protein, albumin, calcium, inorganic phosphate, iron, copper, zinc, blood glucose, blood urea nitrogen and serum transaminase have been noticed in camel affected with mange.*

Musa and Mukhtar (1982) reported mean ± SE values of haemato-biochemical parameters in 174 healthy dromedaries in quarantine awaiting export. The reported mean ± SE values were haemoglobin 11.6 ± 2.5 g/100ml, packed cell volume 25.9 ± 4.4%, red blood cells 6.1 ± 1.5 × 10^6/µl and WBC 12.6 ± 5.2 × 10^3/ µl. The proportion of various white blood cells were neutrophils 55.1 ± 5.2%, lymphocytes 33.9 ± 11.4%, monocytes 4.5 ± 1.6% eosinophils 1.5 ± 6.8% and basophils 0.16 ± 0.5%, respectively.

Ateeq et al. *(1984) reported haematological values in 88 healthy camels aged 1.5-9 years, including 17 pregnant animals aged 5-9 years. Erythrocyte count ranged from 8.76 × $10^6/mm^3$ in group C (female camels 1.5- 4 years old) to 10.22 × 10^3 in group B (male camels 5-9 years old). Leucocytes count ranged from 14.7 × $10^3/mm^3$ in group A (males 1.5-4 years old) to 15.8 × $10^3/mm^3$ in group D (female camels 5-9 years old). Neutrophils and lymphocytes accounted for the greater part of the leucocytes.*

Mourad et al. *(1987) collected blood samples and skin scrappings from 25 mange suffering dromedaries aged 7-9 years. They reported increased eosinophil count. They found that serum from dromedaries with mange had lower concentration of glucose, protein, albumin, sodium and chloride than healthy dromedaries. There was little difference in potassium, calcium, phosphorus and creatinine concentrations.*

Radwan et al. *(1987) conducted ivermectin trial on 20 camels suffering with sarcoptic mange.He observe lower blood heamoglobin, haematocrit, total RBC count, glucose, total protein, albumin and iron. With exception of iron all haematological and biochemical values returned to normal after treatment.*

Al-Ali et al. *(1988) reported values of some haemeto-biochemical parameters as mean value obtained from 20 healthy dromedaries to be Hb 14.5 ± 1.5 mg/dl, erythrocyte count 9.09 ± 0.35 ×10^9/ml, leucocyte count 21.1 ± 8.3×10^3/ml and glucose 138 ± 17mg/dl.*

Yagoub (1988) reported mean ± SE values of some haemotological parameters of 97 healthy male and female camels of different age groups as Hb 12.5 ± 2.4 g/100ml, PCV 26.4 ± 3.4 per cent, TEC 9.0 ± 1.6×10^6µl,TLC 12.9 ± 2.0×10^3 µl, neutrophils 54.2 ± 9.5 per cent and eosinophils 5.4 per cent.

Raisinghani et al. *(1989) studied 20 camel infected with* sarcoptic scabiei var. cameli *and 5 clinically healthy camels of B.S.F. at Jaisalmer. Haematological values of healthy uninfected control camels were haemoglobin (gm per cent) 11.9 ± 0.24, TEC (10^6/cmm) 8.35 ± o.33, PCV (per cent) 28.25 ± 1.48 and TLC (10^3/cmm) 12.1 ± 0.13 witch almost unchanged on 1st day and 145th day of observation. Haematological findings of infected untreated control animals showed a significant decrease in Hb from 8.62 ± 0.40 to 8.25 ± 0.21(gm per cent), TEC from 5.50 ± 0.18 to 4.20 ± 0.24(10^6/cmm) and in PCV from 26.0 ± 1.87 to 22.5 ± 1.48 (per cent). However, leucocytosis and eosinophilia were evident, increasing from 16.93 ± 0.63 to 17.56 ± 0.52 and from 14.4 ± 1.15 to 18.2 ± 0.54 respectively. Haematological values of camel affected with sarcoptic mange showed subseqent improvement following treatment with Ivermectin. The values improved, Hb from 7.1 ± 0.3 to 10.58 ± 0.34 (gm per cent), TEC from 5.54 ± 0.07 to 8.25 ± 0.13 (10^6/cmm) and that of PCV from 24.43 ± 1.24 to 30.57± 0.54 (per cent).*

Partani et al*. (1995) reported normal biochemical values from apparently healthy camels. The values of total serum proteins, albumin and globulin were 6.13 ± 0.19, 3.32 ± 0.12, and 2.80 ± 0.21 g/dl, respectively. Whereas, these values for haematological parameters were Hb 12.77 ± 0.58 gm per cent, PCV 31.12 ± 2.69 (per cent), TEC 7.71 ± 0.45 (× 10^6/cumm) and TLC 10.15 ± 8.2 (× 10^3/cumm).*

Sarwar and Majeed (1997) studied mean biochemical parameter in 1-15 year old camels. The biochemical values for serum glucose 45.03 ± 2.34 mg/dl (18.75 - 75), total protein 7.78 ± 0.22 mg/dl (4.56 - 11.21), albumin 4.34 ± 0.11 gm/dl (2.45 -7.36), globulin 3.51 ± 0.15 gm/dl, (1.45 - 6.94) and A:G ratio 1.36 ± 0.06 (0.36 -2.96). Further, these values for RBC 7.96 ± 0.20 ×10^6/µl (4.29 - 12.15), PCV 26.10 ± 0.45% (20.32), Haemoglobin 13.14 ± 0.17 gm/dl (10.40 - 16.20), TLC 19.04 ± 0.56 × 10^3/µl (10.40 - 29.68), neutrophils 36.66 ± 1.70 % (10.63 – 40.12), eosinophils 5.63 ± 0.40 % (0-14), basophils 0.61 ± 0.10 % (0-3), lymphocytes 51.79 ± 1.84% (22-76) and monocytes 4.69 ± 7.62% (3-8).

Egve Nwiyi and Chaudhari (1996) examined sample of 469 adult camels for infestation of sarcoptis scabiei var. cameli*. Haematological disorders in infected animals were microcytic hypochromic anaemia and leukocytosis.*

Nyang'ao et al. *(1997) studied the mean value of some Haemato-biochemical parameters of the 21 healthy Somali type dromadaries, aged between 3 and 6 years. The value of Haemoglobin concentration, PCV, TEC and TLC were 11.2 ± 1.7 g/dl, 27.1 ± 3 per cent, 8.5 ± 1.6×10^6/µl and 16.3 ± 6.9 ×10^6/µl, respectively. In the DLC, lymphocytes were predominant (56.5 ± 5 per cent, followed by nutrophils (38 ± 4 per cent). There were very few monocytes (0.5 ± 0.2 per cent), eosinophils (1.0 ± 0.2 per cent) and basophils. The mean blood plasma level of blood glucose was 122.9 ± 22.7 mg/dl.*

Rezakhani et al. *(1997) studied haematological and biochemical parameters in 83 healthy Turkmen camel of different age groups of less than 3 (35), 3-6 (17) and more than 6 years (31). The mean haematological values from all the animals were : haemoglobin concentration 12.79 ± 1.28 g/dl; packed cell volume 28.94 ± 3.94 per cent, erythrocyte count 9.84 ± 0.79 × 10^6 cells/µl. The differential counts were neutrophils 46.41 ± 1.57 per cent, lymphocytes 46.67 ± 2.55 per cent, eosinophils 4.53 ± 1.75 per cent, monocytes 2.07 ± 0.08 per cent and basophils 0.00 ± 0.00 per cent. the highest RBC (10.72 ± 0.94 ×10^6 cells /µl) was in more than 6 years old and the lowest (9.18 ± 1.99 × 10^6 cells /µl) in less than 3 years old animals. Hb and PCV increased with the age and there were significant differences between the three groups WBC decreased with increasing age of the animals. Neutrophils were most prevalent in younger camels. The per cent of lymphocytes making up the leucocytes in the different age groups were*

significantly different but showed no association with age. Eosinophils made up of a greater proportion of lyuococytes in older animals.

Dongre (2000) studied the haemato biochemical parameters in ten apparently healthy dromedaries. The mean ± SE values of the parameters studied were reported as Hb 12.16 ± 0.17gm per cent, PCV 27.10 ± 0.34 per cent, TEC 10.64 ± 0.42 millions/cmm, TLC 9.86 ± 0.33 thousands/ cmm, neutrophils 33.0 ± 1.62 per cent, lymphocytes 59.30 ±1.63 per cent, monocytes 4.10 ± 0.52 per cent, eosinophils 3.60 ± 0.52 per cent and besophils 0.10 ± 0.08 per cent respectively, while the mean ± SE values for serum biochemical parameters were calcium 8.24 ± 0.45mg/100ml and glucose 81.10 ± 4.27mg/100ml, respectively.

Gorakhmal et al. *(2000) performed haemato-biochemical examination of camel infected with sarcoptic mange which revealed significant decrease in TEC, TLC, Hb, nuetrophils and monocytes and an increase in lymphocytes (46 ± 1.0% from 38 ± 2.0 per cent) in infested animals.*

Raghbendra et al*. (2000) studied some haemoto- biochemical parameters in 6 healthy dromadaries. The mean ± SE values reported were Hb 11.10 ± 1. 39 g/dl, TEC 8.39 ± 0.40 (× 10^{12}/ L), TLC 13.36 ± 0.52, (× 10^{9}/L), neutrophils 51.33 ± 0.88 (per cent), lymphocyte 37.33 ± 0.99 (%), monocytes 5.84 ± 0.48 (per cent), easonophils 4.50 ± 0.43 (per cent) and besophils 1.00 ± 0.26 (per cent).*

Al-sad et al. *(2000) examined 30 Arabian dromadaries in laboratory showing sings of mange and identified sarcoptic mange. Haematology showed decrease PCV, Hb and TEC.*

Sharma (2000) studied some haemoto- biochemical parameters in 15 apparently healthy dromadaries and the mean ± SE values reported were Hb 10.44 ± 0.23 g/ per cent, PCV 28.33 ± 0.61 per cent, TEC 6.26 ± 0.15 (× 10^{6}/cmm), TLC (7.72 ± 0.03) (×10^{3}/cmm), neutrophils 51. 13 ± 1.73 per cent, lymphocytes 44.47 ± 1.81 per cent, eosonophils 1.40 ± 0.24 per cent, monocytes 2.53 ± 0.22 per cent and basophils 0.46 ± 0.13 per cent.

Singh and Gahlot (2000) reported haemato-biochemical values during some drug trials in mangy camels. They observed decrease in percent Hb, serum glucose, total protein, globulin and calcium with increased eosinophil count due to effect of mites. The haemato-biochemical parameters were studied at pre and post treatment stages with an objectives to access the effect of sarcoptis scabiei *infestation on physiological status of health and to record the efficacy of treatment regarding recovery. There were significant differences for Hb; eosinophil count, blood glucose, total protein, globulin and calcium levels among the pre and post treatment values.*

The quantitative biochemical estimations done by Gorakhmal et al*. (2001) on 16 mange infested camel revealed an increase in alanine transaminase, aspartate transaminase, total proteins, globulins, triglycerides and a decrease in albumin and cholesterol.*

Gorakhmal et al. *(2002) studied hematological and mineral status values in mange affected and healthy camels. Authors observe a significant decrease in the Hb content and a significant increase in number of eosinophils in mange affected camels. Generally the mites feed on lymph and cause hypoproteinemia, oedema, hemorrhage and dermatitis.*

Mali (2002) recorded mean ± SE values of Hb, PCV, TEC and TLC of apparently healthy camels which were 10.20 ± 0.48 gm per cent, 27.90 ± 0.82 per cent, 9.32 ± 0.338 millions/cumm and 11.25 ± 0.41 thousand/cumm respectively. The mean ± SE values of neutrophils, lymphocytes, monocytes, eosonophils and basophils recorded in apparently healthy camels were 52.60 ± 0.54 per cent, 39.60 ± 0.42 per cent, 3.30 ± 0.36 per cent, 4.10 ± 0.16, respectively.

Singh Ishwar et al. *(2003) observed twenty camels naturally affected with sarcoptic mange and recorded mean ± SE value of creatinine, urea and zinc in mange affected camels as 2.08 ± 0.19 mg/dl, 20.16 ± 1.50 mg/dl and 68.40 ± 3.51µg/dl respectively and 1.03 ± 0.17 mg/dl, 52.86 ± 1.41 mg/dl and 124.80 ± 2.05 µg/dl respectively in healthy*

camels. There was a significant reduction in serum urea and zinc levels, but significant increase in serum creatinine levels.

Kataria and Kataria (2004) collected blood samples from 32 adult male camels of which 14 were affected with mange and 22 adults female of which 10 had mange. The overall (male + female) Value of IgE (IU/ml) and total eosinophils counts/µl in healthy camels were 2.35 ± 0.05 and 381.33 ± 12.91, these values for healthy males were 2.38 ± 0.05 and 378.88 ± 16.65 and for healthy females were 2.31 ± 0.09 and 385.00 ± 21.33 respectively. The overall values of IgE (IU/ml) and total eosinophils counts/ µl in mangy camels were 7.33 ± 0.98 and 1032.50 ± 47.35 in mangy males were 8.21 ± 1.54 and 1007.14 ± 59.14 and mangy females were 6.11 ± 0.75 and 1068 ± 84.65 respectively.

Mathur (2004) recorded various haemato-biochemical parameters in camels suffering from sarcopticosis which revealed significant increase in TLC, neutrophil count, eosinophil count, blood glucose, total serum proteins, serum albumin, serum globulin with decrease in, TEC, Hb, PCV, MCHC and lymphocytes count. The values of MCV, basophils count, monocytes count and A: G ratio revealed non-significant effect of cutaneous parasitosis in comparison to normal healthy animal

Mahran, et al. *(2004) reported that sarcoptic mange infested camels showed leucocytosis accompanied by lymphocytosis and eosinophilia and hypoproteinaemia consequent to hypoalbuminaemia and hypoglobunaemia.*

Parmar (2005) recorded mean ± SE value of total protein, Albumin, Globulin, A:G ratio, Hb, TEC, TLC and PCV in mange affected camels as 5.93 ± 0.10 g/dl, 4.01 ± 0.07 g/dl, 1.93 ± 0.08 g/dl, 2.30 ± 0.14, 8.79 ± 0.13 gm per cent, 7.61 ± 0.20 millions/cmm, 12.30 ± 0.42 thousand/cmm and 27.20 ± 0.39 per cent respectively and the values for same parameters in healthy camels were 7.23 ± 0.30, 4.30 ± 0.14, 2.93 ± 0.27, 1.72 ± 0.16, 11.52 ± 0.17 , 10.42 ± 0.13, 9.24 ± 0.18 and 31.50 ± 0.18 respectively.

Anju-Bala., Rath, S.S. (2006) studied haemato-biochemical changes in buffalo calves infested with sarcoptic mange. They reported that the TEC, PCV and Hb decreased and the TLC increased in mange affected calves compared to healthy calves. A slight decrease in blood Cu, Zn and Fe concentration was observed in buffalo calves with mange.

Rathod (2006) recorded some biochemical parameters in eight healthy camels. The values for total protein was 7.65 ± 0.32 (6.66-8.9) g/dl, albumin was 4.25 ± 0.16 (3.88-4.96) g/dl, globulin was 3.41 ± 0.18 (2.78-3.94) g/dl and A:G ratio was 1.26 ± 0.04 (0.15-1.39). Further, mean ± SE values for hemoglobin (gm per cent), PCV (per cent), TEC (million/cumm) and TLC (thousand/cumm) at the 0 day of the experiment in eight apparently healthy camel were 11.8 ± 0.44 (10.2-13), 35.5 ± 1.18 (30-38), 9.13 ± 0.15 (8.79-9.8) and 8.66 ± 0.09 (8.46-9.1), respectively.

*Choudhary, B.S. (2008) recorded mean values of serum urea and serum creatinine in the serum of 10 apparently healthy camels and these values were 21.49 ± 1.103 mg/dl and 1.75 ± 0.054 mg/dl respectively during clinical studies of digestive disorder in camels (*Camelus dromedaries*).*

Kamal, A.M. (2008) recorded mean ± SE value of RBC'S, haemoglobin, PCV, total protein, albumin and creatinine in mange affected camels as 13.95 ± 1.20 (10^{12}/L), 120.10 ± 10.90 gm/l, 25.68 ± 2.15 per cent, 6.80 ± 0.53 gm/l, 2.10 ± 0.29 gm/dl and 1.01 ± 0.40 mg/dl respectively and 18.10 ± 1.21 (10^{12}/L), 136.50 ± 12.50 gm/l, 32.70 ± 2.91 per cent, 7.80 ± 0.65 gm/dl, 3.18 ± 0.32 gm/dl and 0.85 ± 0.05 mg/dl respectively in healthy camels.

Abdally, M.H. (2010) reported mean ± SE values of glucose, urea, creatine, creatinine, GOT and GPT in the serum of mange infested camels and these values were 5.20 ± 0.35 mmol / l, 14.50 ± 4.18mmol/l, 11.50 ±4.18, 119.5 ±13.12 mmol/l, 123.5 ±13.12 and 14.5 ± 7 U/L respectively.

Mineral status

Ghosal and Shekhawat (1992) studied 122 dromedaries (82 from University farm and 40 from 23 villages of Bikaner district). The overall reported mean serum levels of zinc, copper and iron were 85.4 ± 2.5 µg/dl (33.3 - 100.0), 94.3 ± 3.2 µg/100 ml (39.9-160.0) and 107.4 ± 3.0 µg/100 ml (40.0-182.0). Organized farm camels showed higher values as compared to village camels.

Baksh Abdulsalam A. (2000) recorded serum zinc in 50 camels belonging to different breeds, age groups and both sexes and mean value in different age groups of 5-7, 8-10 and above 10 years were 101.58 ± 19.79, 99.00 ± 16.30 and 99.21 ± 21.03 µg/dl, respectively.

Dongre (2000) recorded in copper, iron, manganese and zinc levels in 10 adult healthy male camels. The values were 103.10 ± 6.09, 119.40 ± 8.19, 15.50 ± 1.82 and 107.50 ± 6.97 µg/dl, respectively. These values ranged between 78-126, 65-160, 8-25 and 77-160 µg/dl respectively.

Dixit et al*. (2008) recorded mean ± SE values for copper, cobalt, iron, manganese and zinc for healthy camels as 93.25 ± 5.88; 14.87 ± 1.38; 149.75 ± 5.88; 19.0 ± 1.64 and 153.37 ± 12.96 µg/dl, respectively and for mangy camel as 98.1±6.23; 13.3 ± 1.19; 150.5 ± 12.24; 17.1± 1.18 and 85.4 ± 5.52 µg/dl respectively.*

Histopathology and skin scrapping examination

Lodha (1966) described the histopathology of the infected camel skin. Hyperkeratosis, perakeratosis, acanthosis in the epidermis and oedema and congestion in the dermis were the observations recorded. Capillary dilatation, infilteration of lymphocytes, plasma cells and a few histiocytes were noticed without presence of eosinophils or neutrophils and no change in malphagian layer were observed.Rathore (1971) also observed similar histopathological findings.

Rathore and Lodha (1973) collected skin scrapping from about 540 camels in 3 districts over a period of 3 years. Average number of mites per square centimeter of skin collected from dead mangy camel was 330, while that of eggs was 284.

Higgins (1984) reported that it is difficult to find mites in early stages. He further stressed the importance of taking proper and adequate numbers of skin scrapings from individual mangy camel, care should be taken. Scrape at least 1 cm^2 area of mangy skin.

In camels experimentally infected with Sarcoptes scabiei var. cameli *and* Sarcoptes Scabiei var ovis*, Nayel et al. (1986c) examined skin scrapping from the expirementaly induced lesions, witch contained many mites of all stages, proving that the two strains had become equally well established and had reproduced actively in these lesions.*

Mourad et al. *(1987) collected blood samples and skin scrapings from 25 dromadaries aged 7-9 years suffering with mange. They found that dromadaries with mange had severe itching over large parts of the body.*

Mumcuoglu (1990) described a technique for quantitative evaluation of ectoparasitic mites and insects of domestic animals. A modified KOH (Potassium hydroxide) dissolution technique using tween 80 was developed for the diagnosis and quantitative evaluation of ectoparasitic mites and insects of veterinary importance. Fifty intact skin segments from ten different domestic animals (dog, cat, sheep, goat, rabbit, cattle, horse, pig, hamster and guinea pig) were artificially inoculated in vitro with 10 specimens each of the mites Sarcoptes scabie, Choriptes bovis, psoroptes ovis *and* Demodex caprae *and the insect* Haematopinus eurysysternus *and* Bovocola bovis*. 67-68 percent of mites and 80-99 percent of insects were recovered by this technique. Ectoparasitic arthropods were found and identified in all skin scrapings from all 10 naturally infested animals. Using E.F cook's dissolution technique in experimental design, 63.4% of* C. bovis *and 65% of* S. Scabiei *mites were recovered.*

Egbe Nwiyi and Chaudhary (1996) examined sample of 469 adult camels for infestation of Sarcoptes scabiei var. cameli*. Histopathological examinations of skin tissues from slaughtered animals revealed hyperkeratosis, parakeratosis and mononuclear cellular infiltration especially by lymphocytes, plasma cell and histiocytes into the epidermal and dermal layers of skin.*

Bornstein et al*. (1997) examined skin scrapings of 10 camels suspected clinically of mange to determine the presence of mites. These samples were inadvertently pooled prior to consignment to the laboratory for analysis and were suspended in a 10% solution of a potassium hydroxide in a water bath at 37°c for few hours and centrifuged at 3000 rpm for 3 minutes. The supernatant was discarded and one drop of glycerin added to the sediment before it was microscopically examined for mites, large numbers of different stages of* S. scabiei *were found in pooled skin scrapings.*

Gorakhmal et al. *(2000) performed histological examination of skin of camel infected with sarcoptic mange, which revealed epidermal hyperplasia with perivascular infiltration of eosinophils, lymphocytes and plasma cells.*

Al Saad et al*. (2000) examined 30 Arabian dromedaries in laboratory showing signs of mange and identified sarcoptic mange, microscopic lesions revealed epidermal hyperplasia with marked hyperkeartosis. The epidermis was several times thicker than the normal adjacent epidermis. Finger like projections were seen extended from the epidermis and into the dermis. Sections of the mites were seen forming follicles within the stratum corneum. The whole epidermal lesion was covered with a scab consisting of erythrocytes, tissue derbis and keratin. In the dermis, there was a perivascular dermatitis (infiltration of mononuclear cells with few neutrophils and eosinophils), hyperemia and oedema. Kinney and Wernery (2003) also reported the same findings; in acute infections mites were rarely found however in severe and long standing chronic cases exhibiting severe hyperkeratosis in several host species,* S. scabiei *mites were found in thousand. In such cases, mites were observed in the hyperkeratosis and in the upper cell layers of the epithelium.*

Mathur (2004) studied cutaneous ectoparasitoses in camels in 5 districts of Rajasthan. Camels suffering from sarcopticosis microscopically revealed the presence of minute cavities in the epidermal layer, extended into dermis. These cavities were filled with tissue debris along with infiltration of mononuclear cells. The epidermis showed hyperkeratosis and acanthosis, as there was increase in stratum corneum and stratum germinativum of the epidermis. There was proliferation of fibrous connective tissue at some foci and marked eosinophillic infiltration with oedema was observed.

Parmar et al*. (2005) carried out skin scrapping examination of 138 camels for mange infection and out of which 80 camels were found positive for mange-mite infection.*

Dixit et al. (2008) performed histological examination of skin of camel infected with sarcoptic mange. The epidermis showed hyperkeratosis and acanthosis. The inflammatory cells were also present around the blood vessels and the affected follicles contained mites, keratinous debris along with cellular infiltration.

TREATMENT AND CONTROL

Higgin (1984) recommended spraying with HCH or other acaricides, or a single s/c injection of 1% IVOMEC (Ivermectin) at 1ml/50kg weight, which is effective in controlling the mite infestation in Egyptian camel.

Opferman (1985) examined dromedary with large patches of alopecia and hyperkeratinized skin and revealed S.scabiei. Camel estimated to weigh 400-450Kg were given 0.2 mg/kg Ivermectin s/c. A second dose was given 16 days after the first. By the fifth month after the second treatment the hair returned to normal thickness. No adverse effects were observed after treatment and there was no recurrence of the mange in 12 months period after treatment. Likewise, Hashim and Wasfi (1986) also tried the Ivermectin for the treatment of camel affected with mange @ 200 µg/kg body weight (1 ml/50kg body weight) given subcutaneously with second dose given two weeks later and found good results and reported that Ivermectin was a safer drug.

Hashim and Wasfi (1986) reported the effect of ivermectin on sarcoptic mange in camels at the dose of 200 µg/ kg body weights with a repeated dose after two weeks, and observed a significant effect. Aside from the swelling seen at the injection site, ivermectin was found to be a safe drug in camel.

Radwan et al. *(1987) injected Ivermectin in 4 groups each of 5 dromedaries affected with sarcoptic mange @ 100, 200, 300 or 400 fg/kg at weekly interval. Two higher doses eliminated mites after a single injection and kept the animals free from mites for the subsequent 8 weeks. No adverse effects were observed in pregnant she camels injected with Ivermectin, while 10 mg/kg caused severe depression, ataxia and death within 24 hours.*

Chellappa et al. *(1989) were able to eliminate the sarcoptic mange in two zoo camels with three injection of Ivermectin given at an interval of one week.*

Hassan et al. *(1989) evaluated the efficacy of ivermectin for treatment of sarcoptic mange in camel. They injected ivermectin @ 100 and 200 µg/kg body weight s/c in 60 dromadaries camels showing light, medium and extevsive infestation with* sarcoptes scabiei*. Both dose levels of the drug reduced live mites counts to zero in skin scrapping of camel with extensive lesions after 60 days of administration. In all treated camels rapid clinical improvement was apparent; untreated animals had live mites in all samplings.*

Hassan et al*. (1989) reported that ivermectin was a drug of choice for treatment of sarcoptic mange, which had a significant curative effect for mange in camels and generally used as a subcutaneous injection at the dose rate of 200-300 µg/ kg body weight.*

Similarly, Raisinghani et a*l. (1989) also studied efficacy of ivermectin in mangy camels which was evaluated on the basis of clinical, parasitological, haematological and biochemical responses at interval of 0, 15, 30, 45, and 145 days.The drug was injected s/c @ 200 µg/kg body weight and a second dose given after 15 days.*

Njanja (1991) studied therapeutic use of Ivermectin against sarcoptic mange in camels caused by Sarcoptes scabiei var. cameli *in Kenya. Thirty six naturally infected camels (Turkana breed) were observed for two months for presence of parasitic mites and helminthes. Alternative treatments with Ivermectin 1.0% w/v, injected s/c at 0.2 mg/kg were evaluated using two regimens; a single dose and 2 doses at weekly interval. The mites disappeared by the third week following treatment. Ivermectin (0.1- 0.2 mg/kg) has also been found effective against sarcoptic mange in dogs (Sarma* et al*., 1992), rabbits (Panday* et al*., 1992; Nfi, 1992) and guinea pigs (Schossier and Fehr, 1989).*

Kumar et al*. (1992) reported that sarcoptic mange in camels could be controlled by treatment with broad spectrum systematically administered acaricides and by improving the managerial condition, using a dietary supplement during the dry season to improve body condition and increase host resistance.*

Lumsden (1992) described that Ivermectin, administered to dromedaries by subcutaneous injection at a dose rate of 200µg/kg body weight, had been highly effective against a range of gastrointestinal nematodes and against the mange mite Sarcoptes scabiei var. cameli*. Goundie* et al. *(1993) reported parenterally administered doramectin has been shown to exhibit a wider spectrum and longer protective activity than ivermectin.*

Logan et al. *(1993) reported another long acting new endectocide , doramectin has been found to have wide spectrum of activity against ectoparasites. He observed that no mites were found on doramectin treated cattle at the dose rate of 200 µg /kg after 14 or 21 days after treatments.*

Mottelib (1993) studied efficacy of ivermectin, in camels naturally infected with sarcoptes scabiei *on the basis of clinical, parasitological and haematological responses. The drug was injected s/c @ 2000 mg/kg body weight and repeated after fifteen days. The treatment reduced the number of mites present on the fifteenth day of therapy. Anaemia, leucocytosis, eosinophilia and monocytosis were present before treatment; blood values returned to normal after the second injection. In addition ivermectin gave good results against nematode infections in some camels.*

Maqbool (1996) reported that ivermectin at the dose rates of 0.2 mg/kg body weight and 0.4 mg/kg body weight given by subcutaneous injections repeated 3 times at 15 days interval is 100% effective against light, medium and heavy infestations of sarcoptic mange in 30 camels.

Oukessou et al*. (1996) measured pharmacokinetics of ivermectin in the camel upto 90 days after s/c injection of ivermectin (0.2 mg/kg). Maximum plasma concentration (C_{max}) of 3.24 mg/ml was lower than those reported for cow and sheep (54.58 ng/ml) and 30.80 ng/ml, respectively) and the time to reach C_{max} was longer. The mean residence time was about three times longer in camel (21.50 days) than reported in cow and goat suugesting a slower transit time in camel. Despite the low plasma concentration seen in the study, ivermectin at this dose rate has been shown to be affective against the majority of endo and ecto parasites of camel. This could be explained by the longer duration of exposure of parasite to lower concentration of ivermectin.*

Alvenerie et al*. (1996) and McKellar and Benchaoui (1996) reported about the rate of extent of absorption of ivermectin which was found lower in camel, than in another animals, so it need longer and repeated treatment in camel.*

Smith (1996) stated ivermectin @ 0.2 mg/kg subcutaneously repeated in two weeks as an approved treatment for sarcoptic mange in cattle.

Clymer et al. *(1997) studied comparative efficacy of doramectin and ivermectin against* Psoroptic scabiei *in cattle. Doramectin given at a dose of 200 µg /kg showed 100% therapeutic efficiency after 28 days.*

Hayat et al. *(1997) evaluated acaricides against* sarcoptis scabiei var cameli *mites to dromedary camel in Pakistan. The acaricidal efficacy of diazinon (0.15% solution applied topically twice at 5 days interval) and ivermectin (a single dose s/c @ 0.2 mg/kg body weight) was comparedd in 15 camels with natural mite infestations. Skin scraping were examined 7, 15, 21, 30 and 45 days after treatment. Ivermectin was 98.7% and diazinon was 53.0% effective.*

Arends et al. *(1999) reported that doramectin is successful in single dose treatment of mange mites in pig and in experimental infestation of* Sarcoptes scabiei var suis *in pig.*

Singh and Gahlot (2000) reported decrease in total protein, albumin, neutrophil with increase in eosinophil count due to effect of mites which reverted back to normal after giving single injection of doramectin at the dose rate of one ml/50kg body weight subcutaneously.

Singari et al. *(2001) observed notoedric mange in six rabbits with serve skin lesions which confirmed on skin scrapings examination. With a Single injection of doramectin 400 µg /kg body weight along with supportive treatment. Rabbits showed improvement by 3rd day post treatment. By the 10th day post treatment mange infection was eliminated.*

Echeverria et al. *(2002) applied ivermectin s/c to 6 healthy animasl and 5 animals carrying natural mange infection. Absorption rate was faster in animals with mange. It is suggested that ivermectin is more bioavailable in healthy animals than in animals with mange.*

Kinne and Wernery (2003) reported that a combination of topical and injectible treatment as well as herd management is necessary for complete cure in infection with pathogenic strain of S. scabiei*.*

Anju Bala; Rath, S.S. (2006) reported comparative efficacy of doramectin, ivermectin and amitraz against sarcoptic mange in buffalo calves. One group was treated with doramectin (200µg/ kg body weight twice at 15 days apart). Another group was treated with ivermectin (200µg/kg body weight s/c twice at 15 days apart). Third group was given topical application of amitraz 12.5 per cent (2ml/litre water twice at 15 days apart). Examination of the skin lesions showed that the wounds and wrinkling of the skin disappeared within 7 days ater treatement with doramectin, whereas the thick crusts on the skin took 7 to 14 days to disappear and new hair growth started appearing between

14 to 21 days. In calves treated with ivermectin and amitraz, the thick crusts, wound and wrinkling of the skin disapeard between 14 to 21 days. Skin scrapings from doramectin and invermectin-treated calves was negative for mites seven days after injection of the drug while samples from amritaz-treated calves were negative after 14 days.

Minz et al. *(2006) evaluated therapeutic efficacy of different drugs against mange infection in 25 pigs which were infected with* Sarcoptes scabiei*. The pigs were divided into 5 equal groups and were subjected to the following treatment: (T1) – ivermectin premix given at 330 gm/ton given orally for 7 days. (T2) – pasture mixture of sulphur and karanj oil for 15 days. (T3) – teeburb powder at 2gm/100 kg body weight oral administration up to 15 days, (T4) – doramectin at 200µg/100 kg body weight intramuscularly, and (T5) – control. Fifteen days posttreatment, it could be confirmed that doramectin was 100 % effective in controlling mange infection and thus doramectin was recommended for total control of* S. scabiei.

Mouchira, M.M. and Khalid, A.K. (2009) reported that a single subcutaneous dose of ivermectin at the rate of 10mg/50 kg body weight demonstrated good efficacy in the treatment of scabies in camels.

Abdally, M.H. (2010) studied acaricidal activity of doramectin and ivermectin and found that the activity of doramectin at dose rate of 0.2 mg/kg body weight was stronger and last longer than ivermectin at the same dose rate of 0.2 mg/kg body weight.

MATERIALS AND METHODS

Animals

100 animals suspected for mange were examined by skin scrapping test. 20 animals were found positive for mange. Now a total 30 camels (20 diseased and 10 apparently healthy; irrespective of sex, age and breed) suffering from sarcopticosis (diagnosed on the basis of history, type of lesions and skin scraping examinations) were taken. These animals were brought to the Veterinary Medicine Clinic of Department of Clinical Veterinary Medicine, Ethics and Jurisprudence, College of Veterinary and Animal Science, Bikaner as well as camels belonging to individual holding in and around Bikaner. The 20 diseased camels were divided into 2 groups of 10 each; irrespective of sex,

breed and age for drug trials. Ten apparently healthy camels free from parasitic infestation were also selected for study from various places of Bikaner for haemato-biochemical examination

3.4 Clinical Examination

1. *Clinical examination of each animal was done as per the methods described by Radostits et al. (2007). It included history, duration of illness, changes in managemental and feeding practice, appetite of the animal, abnormalities in the behavior, gait, posture, rumination, defecation (quantity, consistency and frequency), urination, examination of visible mucous membranes, eyes, skin and anus, physical condition, clinical manifestations and general clinical examinations including rectal temperature, pulse, respiration and auscultation of heart and lungs.*

2. *Collection of blood sample for haemato-bochemical examination.*

3.5 Clinical diagnosis

Clinical diagnosis was carried out through history of the case, symptom, skin scrapping examination, hematological estimation and biochemical estimation.

3.6 Skin scrapping examinations

Skin scrappings collected from different sites were suspended in a 10% solution of patassium hydroxide and kept in water bath at 37^0 C for few hours and then centrifuged at 3000 rpm for 3 minutes. The supernatant was discarded and one drop of glycerine was added to the sediment before it was microscopiclly examined for mites and different stages of S. scabiei *or their remnants (Sloss and Kemp, 1978).*

3.7. Collection of blood samples

After clinical examination of the animals, blood samples were collected from jugular vein with all aseptic precautions in sterilized test tubes. For hematological studies, blood was collected in sterile tubes having disodium salt of ethylene diamine tetra acetic acid (EDTA) as an anticoagulant added at the rate of 1 mg/ml of blood as recommended by Jain (1986).

For biochemical studies, blood was collected in other sterile tubes having no anticoagulant. The blood slants were made and incubated for 1 hour at 37^0 C . Blood clots were broken and tubes were centrifuged at 2,500 rpm for 30 min. The serum was pipette out in small Pyrex tubes and was kept immediately in the deep freeze at -20 ºC till analysis.

3.7.1. Haematological examination:

The blood samples were subjected for estimation of haemoglobin, packed cell volume, total erythrocyte count, total leukocyte count, differential leukocyte count and total eosinphil count. These parameters were analyzed as per the methods described by Jain (1986).

3.7.1.1 Haemoglobin (Hb)

Hemoglobin was determined by Sahli-Hellige haemoglo-binometer. Blood was drawn in Sahli's pipette up to 20 cubic millimeter mark. It was then transferred to haemoglobinometer tube containing 4-5 drops of 0.1N hydrochloric acid and mixed well. The tube was then kept for 5 minutes for the haemoglobin to change into acid haematin. The fluid was diluted with distilled water drop by drop and mixing after each drop until it matched to the colour of the standard comparison tubes. The haemoglobinometer tube was read to give the amount of haemoglobin in g/dl of the blood.

3.7.1.2. Packed cell volume (PCV)

For determination of packed cell volume, microhaematocrit method was adopted. Non-heparinized capillary tubes were filled with blood up to three- fourth of total length. The blood adhered over the end of capillary tubes was wiped off with the help of a moist filter paper. The opposite ends of tubes were sealed over the spirit lamp by rotating between the thumb and the index finger for 2-3 seconds over the flame near its base. After perfect sealing of the end, the tubes were centrifuged for 5 minutes at 12,000 rpm in microhaematocrit centrifuge machine.

After centrifugation, packed cell volume was determined with the help of a special microhaematocrit reader scale. The bottom of the red column of capillary tube was adjusted with the zero line and the plasma level was matched with the hundred lines and top of red column excluding buffy layer was read in per cent.

3.7.1.3. Total erythrocytes count (TEC)

The RBC pipette was filled up to 0.5 mark with the blood. The diluting fluid (Hayem's fluid) was drawn up to 101 mark. After shaking the pipette for the three minutes, the fluid in its stem was discarded. The counting chambers of the haemocytometer were carefully charged with the diluted blood after placing a cover slip. It was ensured that blood cells were evenly distributed over the counting chamber and overloading was avoided. The red blood corpuscles present in the four corner small squares and one small central square of the large central square were counted under high power of the microscope.

Calculations

Numbers of red blood cells per cubic millimeter were calculated after multiplying the number of cells counted by 10,000 according to be following formula:

$$\text{Total erythrocytes} = \text{Cells counted} \times 200 \times 10 \times 5 \text{ per cubic mm}$$

Where:

200 stands for dilution

10 stands for depth in mm

5 stands for the 1/5th of square millimeter counted

3.7.1.4. Total leucocytes count (TLC)

The WBC pipette was filled up to 0.5 mark with blood and the WBC diluting fluid was drawn up to 11 mark. After shaking the pipette for three minutes, the fluid in its stem was discarded. Counting chamber of the haemocytometer was carefully charged with diluted blood after placing cover slip. The cells were counted under low power objective of the microscope in the large four corner squares of the haemocytometer.

Calculations

The numbers of leucocytes in one cubic millimeter of blood were calculated by multiplying the total leucocytes counted by factor 50, according to the flowing formula:

$$\text{Total leucocytes per cubic mm} = 4 \quad \text{Cells counted} \times 20 \times 10$$

Where:

20 stand for dilution.

10 stand for depth in mm.

4 stands for the number of square millimeters counted.

3.7.1.5. Total eosinophil count

It was done by Pilot's method as presented by Darnody and Davenport (1958) as follows.

Principle

A diluting fluid for eosinophil count, propylene glycol was employed to lyse all the leucocytes except the eosinophils.

Reagent

Composition of diluting fluid (pilot's)

Propylene glycol	*50 ml*
Distilled water	*40 ml*
Phloxine (1% aqueous solution)	*10 ml*
Na_2CO_3 (10%aqueous solution)	*1 ml*

Mix and filter after which the solution is stable at room temperature for at least one month.

Procedure

The blood was drawn up to 1.0 mark in the white blood count pipette (WBC) it was diluted with Pilot's fluid up to 11 mark. The pipette was shaken for 5 minutes in a pipette shaker. Then it was kept for an hour for completing the lysis of all the cells except eosinophis. After charging the haemocytometer the eosiniphil count was carried out under low power of microscope in all nine chambers.

Calculation

Average number of cells×11.1=total No. of eosinophils per cmm.

3.7.1.6. Differential leucocyte count (DLC)

Thin smears of blood were prepared on dust, lint and grease free clean microscopic slides, immediately after the collection of blood and were air dried. These were then fixed for five minutes with methyl alcohol (methanol). The slides were dried and placed on a staining rack and flooded with Giemsa's stain (BDH) freshly diluted in the ratio of 1:10 and allowed to act for 30 minutes. The slides were washed with neutral distilled water, air dried and examined under oil immersion lens of the microscope for differential leucocyte count. One hundred cells were counted. Neutrophils, lymphocytes, monocytes, eosinophils and basophils were differentiated and expressed in per cent.

3.8 Biochemical estimations:

Biochemical analysis of serum samples were carried out to estimate serum glucose, serum total protein, albumin, globulin and A/G ratio. Determination principle, reagents required, procedure, calculation and precautions used for each of them are described below:

3.8.1 Determination of serum glucose

Serum glucose was estimated by enzymatic GOD-POD method as recommended by Tietz (1976), using kit (Span diagnostic Ltd., India).

Principle

Glucose is oxidized by the enzyme glucose oxidase (GOD) to give gluconic acid and hydrogen peroxide. In a subsequent peroxidase catalysed reaction, the oxygen liberated is accepted by the chomogen system to give a red coloured quinoneamine compound. The red colour so developed is measured at 505 nm and is directly proportional to glucose concentration.

Reagents:

Reagent 1 Glucose reagent : Glucose oxidase, peroxidase, 4-Amino antipyrine, Buffer, Stabilizer, Preservatives.

Reagent 2 Glucose diluent : Diluent preservatives.

Reagent 3 Phenol reagent : Phenol preservatives.

Reagent 4 Glucose standard : Dextrose 100mg/dl : Benzoic acid.

Procedure:

A. Preparation of Working Glucose Reagent

1. *Quantitatively transfer the contents of vial of reagent 1 (glucose reagent) to a clean amber coloured glass bottle, label it as "Working glucose reagent". Reconstitute the contents of each bottle to 500 ml with Reagent 2 (glucose diluent). Add 25 ml of Reagent 3 (phenol reagent) to the reconstituted glucose reagent. Mix well, swirl contents of the bottle gently to mix them thoroughly, avoiding vigorous shaking and store at 2-8^{o}C in dark.*

2. *Pipette out 10 µl of serum or plasma in a test tube to which 1000 µl of working glucose reagent were added. Incubate at 37^{o}C for 16 minutes or at room temperature for 30 minutes after proper mixing.*

B. Standard

Ten microlitre of Reagent 4 (glucose standard) was taken in a test tube to which 1000 µl of working glucose was added. Incubate at 37^{o} C for 15 minutes or at room temperature for 30 minutes after proper mixing.

C. Blank

One millilitre of working glucose reagent was as blank. The tubes were out of water bath, cooled, absorbance of test and standard were measured against blank at 505 nm.

Calculations

$$\text{Glucose in mg/dl} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times 100$$

Precautions:

1. *Haemolysed serum samples were discarded*

2. *Glassware was thoroughly washed, rinsed with distilled water and perfectly dried.*

3. *All the reagents were brought to room temperature before use.*

3.8.2. Determination of total protein

Total proteins in serum were estimated colorimetrically using kit (SPAN Diagnostic Ltd., India) as per modified Biuret and Doumas method (Doumas et al., 1981).

Principle

Cupric ions of biuret reagent form chelates with peptide bonds of proteins in an alkaline medium to form a blue-purple complex. Sodium-potassium tartarate keeps the cupric ions in solution. The intensity of the blue-purple color that is formed is proportional to the number of peptide bounds which, in turn, depends upon the amount of proteins in the specimen.

Reagents required

1. *Biuret reagent- 3 gram of copper sulphate was dissolved in 500 ml of distilled water. To it 9 gram of sodium potassium tartarate and 5 gram of potassium iodide were added and mixed. Then 24 gram of sodium hydroxide dissolved separately in 100 ml of water was added to it.*

2. *Protein standard- The total protein content of pooled serum was determined by kjeldhal method and calibrated against a control serum having a known protein concentration. Protein standard contained total protein concentration of 7 gram per 100 ml of serum.*

Procedure

Three tubes were marked as blank (B), standard (S) and test (T). 5 ml of modified biuret reagent was measured into each of the tube. 0.1 ml of serum into test, 0.1 ml of protein standard into standard and 0.1 ml of distilled water into blank were added, mixed well and allowed to stand at room temperature for 5 minutes. Optical density (OD) of standard (S) and test (T) were measured on a colorimeter against blank (B) with a yellow green filter (550nm).

Calculations:

$$\text{Total serum protein in (gm/100 ml)} = \frac{O.D\ (test)}{O.D\ (standard)} \times \text{Concentration of total protein in standard}$$

$$= \frac{O.D\ (test)}{O.D\ (standard)} \times 7.0$$

Precautions:

1. *Haemolysed serum samples were discarded*

2. *Glassware was thoroughly washed, rinsed with distilled water and perfectly dried.*

3. *All the reagents were brought to room temperature before use.*

3.8.3 Determination of albumin

Serum albumin was determined using kit as per bromocresol green (BCG) dye binding method (Doumas et al., 1971).

Principle

Binding of a protein to an indicator changes its colour. Among serum protein, only albumin in serum binds with the dye Bromocresol green (BCG) at pH 4.2, to form a green coloured complex, which is measured colorimetrically. The pH is maintained during the reaction by a buffer.

Reagents required:

1. *Succinate buffer- 11.8 gram of succinic acid are dissolved in about 800 ml of distilled water. The pH was adjusted to 4.0 with 0.1 N sodium hydroxide. The volume was made up to 1 liter with distilled water. The solution was stored in refrigerator.*
2. *Bromocresol green (BCG) solution- 419 mg of bromocresol green was dissolved in 10 ml of 0.1 N sodium hydroxide solutions. The volume was made up to 1 liter with distilled water. The solution was stored in refrigerator.*
3. *Buffered BCG dye reagent- 250 ml of BCG solution was mixed with 750 ml of succinate buffer. The pH was adjusted to 4.2 with 0.1 N sodium hydroxide solution and then 4 ml of Brij - solution (30%) was added.*
4. *Standared albumin solution- An aqueous solution of albumin with a concentration of 3.9 gram/ 100 ml of serum was prepared and used as a standard.*

Procedure

Three tubes were marked as blank (B), standard (S) and test (T). Four ml of buffered dye reagent was measured into each of the tube. 0.03 ml of serum in test, 0.03 ml of standard albumin in standard and 0.03 ml of distilled water in blank were added, mixed well and allowed to stand at room temperature for 1 minute. Optical density (OD) of standard (S) and test (T) were measured on a colorimeter against blank (B) with a yellow red filter (600nm).

Calculations:

$$\text{Total serum albumin in (gm/100 ml)} = \frac{\text{O.D (test)}}{\text{O.D (standard)}} \times \text{Concentration of total albumin in standard}$$

$$= \frac{\text{O.D (test)}}{\text{O.D (standard)}} \times 3.9$$

Precautions:

1. *Haemolysed serum samples were discarded*
2. *Glassware was thoroughly washed, rinsed with distilled water and perfectly dried.*
3. *All the reagents were brought to room temperature before use.*

3.8.4 Determination of Globulin

Serum globulin was estimated in gm / 100 ml as a difference between total protein and albumin, which were estimated as per the modified Biuret and Doumas method (Doumas et al., 1981) and Bromocresol green dye binding method (Doumas et al., 1971).

Serum globulin (gm / 100 ml) = Total serum protein in (gm / 100 ml) - Albumin in (gm / 100 ml).

3.8.5 Determination of albumin globulin ratio

Albumin and globulin ratio (A: G) was calculated by dividing concentration of albumin by concentration of globulin in gm/100 ml.

3.8.6 Determination of serum creatinine

Serum creatinine was determined using kit as per Alkaline Picrate Method.

Principle:

Creatinine in a protein free solution reacts with alkaline picrate and produces a red coloured complex, which is measured colorimetrically.

Reagents :

Reagent 1 : Picric acid

Reagent 2 : NaOH, 0.75 N

Reagent 3 : Stock creatinine standard, 150mg %

Procedure:-

For colorimeter-

Step A deprotinization of test sample:

Serum : 0.5ml

Purified water : 0.5ml

Reagent 1: picric acid : 3.0ml

Mix well keep in boiling water bath exactly for one minute cool immidietly under running tap water and centrifuge or filter.

Step B. colour development :

		Blank (B)	*Standard (S)*	*Test (T)*
Filtrate/supernatant (from step A)	*:*	*–*	*–*	*2.0 ml*
Working standard	*:*	*–*	*0.5 ml*	*–*
Purified water	*:*	*0.5 ml*	*–*	*–*
Reagent 1 picric acid	*:*	*1.5 ml*	*1.5 ml*	*–*
Reagent 2 NaOH, 0.75N	*:*	*0.5 ml*	*0.5 ml*	*0.5 ml*

Mix well and allow to stand at room temperature exactly for twenty minutes and measure immediately the optical density of blank (B), standard (S), and test (T) against purified water on a colorimeter with a green filter.

CALCULATIONS

$$\text{Total serum creatinine in (gm/100 ml)} = \frac{\text{O.D (test)} - \text{O.D (Blank)}}{\text{OD. Standard} - \text{O.D. Blank}} \times 3$$

PEACAUTIONS:

1. *Use clean and dry glassware.*
2. *Bring all the solutions to room temperature before use.*
3. *Mark the test tubes properly as Blank (B),Standard (S) and Test (T) before proceeding for the estimations, because marking may come off when the tubes are placed in the boiling water bath during deproteinization (Step A)*

3.8.7 Determination of serum urea:

Serum urea was determined using kit as per DAM Method

PRINCIPLE :

Urea reacts with hot acidic Diacetylmonoxime in presence of Thiosemicarbazide and produces a rose – purple colored complex, which is measured colorimetrically.

REAGENTS:

Reagent 1 Urea Reagent

Reagent 2 Diacetylmonoxime (DAM)

Reagent 3: Working Urea Standard, 30mg%

PROCEDURE

A. For Colorimeter

		Blank (B)	*Test (T)*	*Standard (S)*
Solution 1	*:*	*2.5 ml*	*2.5 ml*	*2.5 ml*
Sample	*:*	*–*	*0.01ml*	*–*
Reagent 3:working urea std, 30mg %	*:*	*–*	*–*	*0.01ml*
	Mix Well			
Reagent 2 : Diacetylmonoxime	*:*	*0.25ml*	*0.25 ml*	*0.25ml*

Mix well and keep the tubes in the boiling water exactly for 10 minutes. Cool immediately under running water for 5 minutes, mix by inversion and measure the color intensity within 10 minutes using a green filter against blank

CALCULATION:

$$\text{Total serum urea in (mg/100 ml)} = \frac{\text{O.D (test)}}{\text{O.D (standard)}} \times 30$$

PEACAUTIONS:

1. *Use chromic acid washed dry glassware.*
2. *Bring all the solutions to room temperature before use.*
3. *Prepare a standard for each series of determination.*
4. *Since the method is very sensitive, sample and standard shuold be measured accurately*

3.9 Estimation of serum zinc by Atomic Absorption Spectrophotometer:

Serum zinc levels were estimated by Atomic Absorption Spectrophotometer

Digestion

The digestion procedure was adopted as prescribed by Singh (1989). One ml of serum sample was taken in to duly labeled kjeldahls flask. One ml each of perchloric acid and concentrated sulphuric acid and two ml of concentrated nitric acid were added to the flask and mixed by rotary motion. The content of the flask were digested until a clear solution was obtained. Further, the flask was allowed to heat until 0.5ml to 1.0ml solution remained. The flask was then allowed to cool. The volume of digested sample was made to 10 ml by adding double glass distilled water and stored in clean double capped zinc free plastic bottles until analyzed.

Analysis by Atomic Absorption Spectrophotometer

The Shimadzu Atomic Absorption Spectrophotometer AA 646 (Shimadzu Corporation, Spectrophotometric instruments plant, Analytical instruments division, Kyoto, Japan) was used for the estimation of serum zinc levels.

For the estimation of zinc, the instrument was set in order as per the specification of the AA 646. Operating parameter for estimation of zinc was as follows:

Parameters	*Zn*
Instrument	*GSB 932 AAS*
Wavelength	*213.9 nm*
Slit setting	*0.5 nm*
Light source	*Hollow cathode lamp*
Flame type	*Air-acetylene flame oxidizing (lean, blue)*
Operating current	*5.0 mA*

Standardization procedure for zinc

Hundred milligram of pure zinc metal was dissolve in minimum amount of dilute hydrochloric acid and made to one liter by adding double glass distilled water. This stock solution contained 100μg zinc/ml. In six different 100 ml flasks, aliquots were taken and standard of 0, 0.5, 1.0,1.5, 2.0 and 2.5 ppm zinc solution were made and standard curve was prepared against the reading of the AAS after setting the operating parameter and calibration of the instrument (AAS).

3.10 Therapeutic trials

A total of 20 positive cases of Sarcopticosis were selected for treatment. These 20 camels were classified into 2 groups irrespective of sex, breed and age (Group I and II) having 10 number in each group for drug trials.

The treatment was administered after completion of the skin scrapping examination.

(i) First group was given Ivermectin at the rate of 0.2mg/kg body weight; s/c. Second inj. repeated 10 days after first dosing. (Ivectin, marketed by Indian Immunological Limited, 1% Ivermectin w/v)

(ii) Second group was given injection Doramectin at the rate of 0.2mg/kg body weight, i/m. Second inj. repeated 10 days after first dosing. (Pfizer Animal Health, Mumbai, each ml contain 10mg doramectin.)

After administration of the drug in each group, blood samples were again collected on 10th day, 20th day for haematobiochemical estimations to note the efficacy of drug.

3.12 Statistical analysis

The data obtained in research work undertaken were statistically analyzed and comparedd as per the standard statistical procedures suggested by Sendecor and Cochran (1994) and significance of mean difference were tested by Duncan's new multiple range test (DNMRT).

RESULT AND DISCUSSION

An effort has been made to draw and evaluate a picture of clinical and haemato-biochemical parameters of camels suffering from sarcopticosis. Efficacy of Doramectin as compared to Ivermectin has been seen on the basis of examination of mites in skin scrapping, clinical signs and haemato-biochemical parameters to access superiority of one drug over the other. For this a total of thirty (30) camels (irrespective of sex and age) were examined. Out of thirty, ten camels were apparently healthy which were kept as control (HC). The rest twenty camels were suffering from sarcopticosis and divided into two groups of ten each. Out of these, ten animals were treated with ivermectin while other ten were treated with doramectin.

Treatment trial

All the twenty camels were subjected to the therapeutic efficacy of various drug trials. The general condition of all the diseased camels was poor with variable degree of emaciation. These animals were randomly classified irrespective of age and sex into two groups consisting of ten camels in each. The Group- I was treated with injection Ivermectin and Group- II with injection Doramectin.

Group- I

All the ten positive camels subjected to this group were administered injection Ivermectin 1% (Ivectin, Indian Immunologicals) @ 1ml/50 kg body weight subcutaneously. The second injection was administered after 10 days in all the cases because clinical symptoms persisted after the first treatment.

In four less severe cases clinical symptoms were itching and alopecia with lesions over the face and neck The clinical symptoms did not disappear after first injection. The lesions disappeared after second dose but still there were presence of scar over the site. In remaining six more severe cases two doses were used. After 20 days, the scar and roughness of skin remained and alopecia was not cured but other clinical manifestations subsided.

Similar to present investigation, Higgins (1984), Raisinghani et al*. (1989), Njanja (1991), Lumsden (1992), Mottelib (1993) and Mouchira* et al. *(2009) used injection Ivermectin for the treatment of sarcopticosis in camels.*

Group- II

In Group- II sarcopticosis suffering camels were given treatment with injection Doramectin 1% (Dectomax, Pfizer) @ 1ml/50 kg body weight intramuscularly. The second injection was administered after 10 days on those cases in which the clinical symptoms persisted after first treatment.

In four less severe cases the initial symptoms were itching and alopecia with lesions over the face and neck. The clinical symptoms disappeared after first injection and camels clinico-parasitologically were cured completely with no lesions on the body. In remaining six more severe cases, two doses were used, the recoveries in these camels were manifested by subsided clinical symptoms like itching, alopecia, corrugation and thickening of skin, hyperemia and oozing of blood and even keratinization of the skin also disappeared.

Similar to present, Veer *et al.* (2001), Parmar, A.J. (2005) and Abdally (2010), used injection Doramectin for the treatment of sarcopticosis in camels.

The results of all parameters studied during the course of study are presented here under:

Clinico-physiological parameters

The basic parameters of health i.e. temperature, pulse and respiration were observed in all the thirty animals taken for study.

Note:- The values of temperature, pulse and respiration for healthy control camels were taken once only. Same values have been presented on 10th and 20th day for the purpose of comparision with G-I and G-II group values.

(1) Temperature (°F)

The mean ± SE values of temperature (°F) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 1. The data of temperature (°F) is presented in Table i – iii of appendix.

Table: 1 Mean ± SE values of temperature (^{0}F) of camels of different groups (n=10)

Treatment	*HC (Healthy control)*	*G-I (Ivermectin)*	*G-I (Doramectin)*
Pre-treatment	*97.08 ± 0.14*	*97.12 ± 0.21*	*95.34 ± 1.73*
10th day	*97.08 ± 0.14*	*97.18 ± 0.20*	*97.14 ± 0.16*
20th day	*97.08 ± 0.14*	*97.18 ± 0.13*	*97.30 ± 0.14*

The mean ± SE value of healthy control (HC) for temperature was 97.08 ± 0.14^{0}F The pre-treatment and post-treatment value for group-I were 97.12 ± 0.21 and 97.18 ± 0.13 ^{0}F respectively. Such values for Group-II were 95.34 ± 1.73 & 97.30 ± 0.14^{0}F respectively.

The statistical analysis did not reveal any significant (P ≤ 0.01) difference in any of the group mean value of temperature recorded at pre-treatment (1st) and post-treatment (10th , 20th) day.

(2) Pulse (rate/minute):-

The mean ± SE values of pulse (rate/minute) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 2. The data of pulse (rate/minute) is presented in Table i – iii of appendix.

Table: 2 Mean ± SE values of pulse (rate/minute) of camels of different groups (n=10)

Treatment	*HC (Healthy control)*	*G-I (Ivermectin)*	*G-II (Doramectin)*
Pre-treatment	*43.5 ± 0.58*	*48.0 ± 1.27*	*48.0 ± 0.94*
10th day	*43.5 ± 0.58*	*48.2 ± 1.17*	*48.4 ± 0.71*
20th day	*43.5 ± 0.58*	*49.5 ± 1.40*	*47.6 ± 1.02*

The mean ± SE value of healthy control (HC) for pulse was 43.50 ± 0.58 per minute. The pre-treatment and post-treatment values for Group-I were 48.00 ± 1.27 and 49.5 ± 1.40 per minute. respectively. Such values for Group-II were 48.00 ± 0.94 and 47.60 ± 1.02 per minute respectively.

During the whole period of study, no significant (P ≤ 0.01) variation was recorded in the pulse of healthy control group as well as in the camels affected with sarcopticosis .

(3) Respiration (rate/minute):-

The mean ± SE values of respiration (rate/minute) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 3. The data of respiration (rate/minute) is presented in Table i – iii of appendix.

Table: 3 Mean ± SE values of respiration (rate/minute) of camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Dormectin)***
Pre-treatment	*13.6 ±0.42*	*14.2 ±0.95*	*15.1 ±0.95*
10th day	*13.6 ±0.42*	*14.6 ±0.96*	*14.6 ±0.90*
20th day	*13.6 ±0.42*	*14.6 ±0.94*	*16.1 ±0.91*

The mean ± SE value of healthy control (HC) for respiration was 13.60 ± 0.42 per minute. The pre-treatment and post-treatment values for Group–I were 14.20 ± 0.95 and 14.60 ± 0.94 per minute respectively. Such values for Group–II were 15.10 ± 0.95 and 16.10 ± 0.91 per minute respectively.

The statistical analysis revealed, no significant (P ≤ 0.01) variation in values of respiration in healthy control as well as treatment groups.

The present finding of temperature, pulse and respiration are in agreement with the findings of Sardari lal (1988), Sena et al. (1997-98), Dongre (2000) , Mali (2002) and Rathore (2006).

Clinical manifestations

The clinical manifestations varied from camel to camel in all the two groups except healthy control animals. Sarcopticosis was confirmed by skin scraping which were taken from various affected sites of the animals. Presence of mites, as such, their remnants or the presence of eggs was considered positive. The general condition of all the camels sufferings from sarcopticosis was poor with variable degree of emaciation. The gray coloured gross skin lesion having intense irritation due to itching, alopecia, thickening and corrugation were present on the face, neck, limbs, ventral and lateral part of abdomen. The lesions also showed hyper pigmentation, hyperemia and severe scratching with oozing of blood which could possibly be due to itching or intense irritation caused by ectoparasite penetrating the skin in turn provoking the animal to rub its body against rough objects like trees walls, electric poles etc. Self mutilation by mouth was also noticed. Deterioration of quality of skin, Keratinization and leathery appearance of skin was also recorded.

Above findings of skin lesions are in agreement with the findings of Rathore (1971), Higgins (1983), Raisinghani and Kumar (1990), Singh and Gahlot (2000), Parmar and Singh (2005) and Mouchira and Khalid (2009).

Haematological Parameters:-

Note:- The value of haematological Parameters of healthy control camels were taken once only. Same values have been presented on 10^{th} and 20th day day for the purpose of comparision with G-I and G-II group values.

(1) Haemoglobin (gm %)

The mean ± SE values of haemoglobin (gm%) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 4. The data of haemoglobin (gm %) is presented in Table iv –vi of appendix.

Table: 4 Mean ±SE values of Haemoglobin (g %) of camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Doramectin)***
Pre-treatment	*12.04 ±0.36 $^{a}{}_{b}$*	*8.75 ±0.26 $^{a}{}_{a}$*	*9.18 ±0.24 $^{a}{}_{a}$*
10th day	*12.04 ±0.36 $^{a}{}_{b}$*	*9.48 ±0.28 $^{a}{}_{a}$*	*10.1 3 ±0.30 $^{b}{}_{a}$*
20th day	*12.04 ±0.36 $^{a}{}_{a}$*	*10.42 ±0.28 $^{b}{}_{a}$*	*11.4 ±0.24 $^{c}{}_{a}$*

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camel suffering with sarcopticosis had significant lower mean haemoglobin value as compared to healthy control animals.

The mean ± SE value of healthy control (HC) for haemoglobin was 12.04 ± 0.36 gm per cent. The pre-treatment and post-treatment mean ± SE value for group-I were 8.75 ± 0.26 and 10.42 ± 0.28 gm per cent. respectively. Such value for group-II were 9.18 ± 0.24 and 11.44 ± 0.24 gm per cent, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value had no significant variation. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly higher than pre-treatment value. As there was no significant (P ≤ 0.01) difference of G-II, 20th day compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

The 20th day value of G-I and G-II had no significant difference but the value attained in G-II group was more nearer to the healthy control group as compared to G-I group, indicating better efficacy of drug given to G-II group animals.

The possible reason for increasing trend of Haemoglobin content of blood might be due to influence on restoration of feeding behavior of camels which appear to be a natural process after withdrawl of irritation.

The above findings are in agreement with the findings of Ibrahim et al. (1981), Radwan et al. (1987), Raisinghani et al. (1989), Singh and Gahlot (2000), Parmar, A.J. (2005), Rathor (2006) and Kamal, A.M. (2008).

(2) Pack Cell Volume (%)

The mean ± SE values of PCV (%) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 5. The data of PCV (%) is presented in Table iv – vi of appendix.

Table: 5 Mean ± SE values of PCV (%) of camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Doramectin)***
Pre-treatment	$35.5 \pm 0.76^{a}{}_{b}$	$26.3 \pm 0.47^{a}{}_{a}$	$27.4 \pm 0.77^{a}{}_{a}$
10th day	$35.5 \pm 0.76^{a}{}_{b}$	$29.2 \pm 0.94^{b}{}_{a}$	$30.4 \pm 0.74^{b}{}_{a}$
20th day	$35.5 \pm 0.76^{a}{}_{b}$	$32.2 \pm 1.01^{c}{}_{a}$	$34.6 \pm 1.04^{c}{}_{b}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

Similar to haemoglobin, the mean PCV values were also found significantly (P≤0.01) lower in camels suffering from sarcopticosis in comparision to healthy control.

The mean ± SE value of healthy control (HC) for PCV was 35.5 ± 0.76 %. The pre-treatment and post-treatment mean ± SE value for group-I were 26.3 ± 0.47 and 32.2 ± 1.01 %, respectively. Such values for group-II were 27.4 ± 0.77 and 34.6 ± 1.04 %, respectively. G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly higher. The value on 20th day was significantly higher than pre-treatment value. As there was no significant (P ≤ 0.01) difference of G-II, 20th day compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

The 20th day value of G-II was significantly higher than G-I which showed more efficacy of drug given to G-II group animals.

There was a decreased in PCV value in infested animals which may be contributed by the decreased cellular contents in blood after infestation of mange mites (Tung et al. 1975).

The above findings are in agreement with the findings of Raisinghani et al*. (1989), Alsaad* et al. *(2000), mathur (2004), Parmar A.J.(2005), Rathore (2006) and Kamal, A.M. (2008).*

(3) Total leucocyte count (thousand/cmm)

The mean ± SE values of total leucocyte count of healthy control camels and in diseased camels subjected to different treatment are presented in Table 6. The data of total leucocyte count is presented in Table iv – vi of appendix.

Table: 6 Mean ± SE values of TLC (thousand/ cumm) of camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Doramectin)***
Pre-treatment	8.84 ± 0.09	7.90 ± 0.22	7.89 ± 0.22
10th day	8.84 ± 0.09	8.31 ± 0.20	8.86 ± 0.20
20th day	8.84 ± 0.09	8.67 ± 0.18	9.56 ± 0.11

The mean ± SE value healthy control (HC) for TLC was 8.84 ± 0.09 thousand/ cumm. The pre-treatment and post-treatment mean ± SE value for group-I were 7.90 ± 0.22 and 8.67 ± 0.18 thousand/ cumm respectively. Such values for group-II were 7.89 ± 0.22 and 9.56 ± 0.11 thousand/ cmm respectively.

The group mean values of Total leukocyte count of all groups under study indicated that there was no variation among the prê-treatment and post-treatment of TLC. The mean values on 10th and 20th day of Group- I and Group- II showed no significant (P ≤ 0.01) variation as compared to healthy control animals.

The findings of present investigation are in agreement with the findings of Musa and Mukhtar (1982), Raisinghani et al.(1989), Dongre (2000) and Mali (2002).

(4) Total erythrocyte count (million/cumm)

The mean ± SE values of total erythrocyte count of healthy control camels and in diseased camels subjected to different treatment are presented in Table 7. The data of total erythrocyte count is presented in Table iv – vi of appendix.

Table: 7 Mean ± SE values of TEC (Million/cumm) of camels of different groups (n=10).

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$8.96 \pm 0.24^{a}{}_{b}$	$7.18 \pm 0.26^{a}{}_{a}$	$7.22 \pm 0.27^{a}{}_{a}$
10th day	$8.96 \pm 0.24^{a}{}_{b}$	$7.65 \pm 0.26^{ab}{}_{a}$	$8.16 \pm 0.30^{b}{}_{a}$
20th day	$8.96 \pm 0.24^{a}{}_{a}$	$8.40 \pm 0.20^{b}{}_{a}$	$9.05 \pm 0.23^{c}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The mean ± SE value for healthy control was 8.96 ± 0.24. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 7.18 ± 0.26 and 8.40 ± 0.20 respectively. Such values for Group- II were 7.22 ± 0.27 and 9.05 ± 0.23 millions/cumm respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value had no significant variation. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-II, 20th day value as compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

Erythrocyte count decreased because there were oozing of blood from wound which is originated from self mutilation and also there were reduce nutrient intake which cause decrease value of total erythrocyte count.

The above findings are in agreement with Radwan et al. (1987), Raisinghani et al. (1989), Gorakhmal et al.(2000), Al saad et al. (2000), Parmar, A.J. (2005), Rathore (2006) and Kamal, A.M. (2008).

(5) Total eosinophil count (/µl)

The mean ± SE values of Total eosinophil count per microlitre of healthy control camels and in diseased camels subjected to different treatment are presented in Table 8. The data of Total eosinophil count is presented in Table – vii of appendix

Table: 8 Mean ± SE values of total eosinophilic Count (/μl) of camels of different groups (n=10)

Treatment	HC (Healthy control)	G – I (Ivermectin)	G–II (Doramectin)
Pre-treatment	$381.1 \pm 3.88^{a}{}_{a}$	$1075.6 \pm 43.02^{c}{}_{c}$	$974.77 \pm 46.01^{c}{}_{b}$
10th day	$381.1 \pm 3.88^{a}{}_{a}$	$777.8 \pm 34.39^{b}{}_{c}$	$655.07 \pm 35.39^{b}{}_{b}$
20th day	$381.1 \pm 3.88^{a}{}_{a}$	$540.7 \pm 35.36^{a}{}_{b}$	$384.04 \pm 16.59^{a}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The mean ± SE value for healthy control was 381.1 ± 3.88 per μl. The pre treatment and post treatment mean ± SE values for G-I were 1075.6 ± 43.02 and 540.7 ± 35.36 per μl respectively . Such value for G-II were 974.77 ± 46.01 and 384.04 ± 16.59 per μl respectively.

G -I pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals could not attain normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was no significant difference of G-II, 20th day value compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

The G-I group value on 20th day was significantly (P ≤ 0.01) higher as compared to healthy control while this value in G-II group had no significant difference and almost attained normalacy as compared to healthy control indicating a better recovery in G-II group animals.

Increase in total eosinophlic count may be due to hyper sensitivity developed due to allergic reactions which in turn lead to increased number of eosinophil (Radostits et al. 2007). The post treatment decrease in total eosinophilic count may be due the effect of drug (Dixit et al. 2004).

Increase eosinophilic counts in sarcoptic camel have been recorded by Mourad et al. (1987), Sena et al (1999), Singh and Gahlot (2000), Kataria and Kataria (2004), Parmar, A.J. (2005) and Rathore (2006).

(6) Differential leucocyte Count

The results of differential leucocyte count studied during the course of study are presented and discussed under following subheading:

(i) Neutrophil count (%)

The mean ± SE values of neutrophil count (%) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 9. The data of neutrophil count (%) is presented in Table viii – x of appendix

Table: 9 Mean ± SE values of neutrophil count (%) of camels of different groups (n=10)

Treatment	HC (Healthy control	G -I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$50.1 \pm 1.29^{a}{}_{b}$	$42.6 \pm 0.70^{a}{}_{a}$	$42.5 \pm 0.61^{a}{}_{a}$
10th day	$50.1 \pm 1.29^{a}{}_{b}$	$46.0 \pm 0.57^{b}{}_{a}$	$46.3 \pm 0.65^{b}{}_{a}$
20th day	$50.1 \pm 1.29^{a}{}_{a}$	$48.8 \pm 0.59^{c}{}_{a}$	$50.1 \pm 0.56^{c}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The mean ± SE value for helthy control was 50.1 ± 1.29 per cent. The pre-treatment and post-treatment (20th day) mean ± SE values for Group- I were 42.60 ± 0.70 and 48.8 ± 0.59 per cent respectively. Such values for Group-II were 42.4 ± 0.61 and 50.1 ± 0.56 per cent respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-I, 20th day value as compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-II, 20th day value as compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

The 20th day value of G-I and G-II as compared to the healthy control group had no significant (P ≤ 0.01) difference but the value attained in G-II group were more nearer to the healthy control group as compared to G-I group.

In any infection neutrophil are the first cells which reaches at the site of infection and destroy the foreign bodies, during this process some neutrophils get destoyed and finally engulfed by phagocytic cells which causes decrease in number of neutrophils.

Statistical analysis revealed significant (P≤0.01) difference within the period of study and between treatments as compared to control group but their mean value was within physiological limits of neutrophil value.It can be attributed to reduced parasitic burden as a result of treatment.

The above findings are in agreement with the findings of Mathur (2004) and Rathor (2006).

(ii) Lymphocyte count (%)

The mean ± SE values of Lymphocyte count (per cent)of healthy control camels and in diseased camels subjected to different treatment are presented in Table 10. The data of Lymphocyte count (per cent) is presented in Table viii – x of appendix.

Table: 10 Mean ± SE values of lymphocyte count (%) of camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	39.1 ± 0.9	39.0 ± 1.44	41.5 ± 0.85
10th day	39.1 ± 1.9	41.9 ± 1.07	43.7 ± 0.81
20th day	39.1 ± 0.9	42.5 ± 1.24	44.3 ± 0.63

The mean ± SE value for healthy control was 39.1± 0.9 %. The pre-treatment and post-treatment mean ± SE values for G-I were 39.0 ± 1.44 and 42.5 ± 1.24%, respectively. Such values for G-II were 41.5 ± 0.85 and 44.3 ± 0.63 %, respectively.Statistical analysis revealed that there were no significant (P ≤ 0.01) variations

(iii) Monocyte (per cent)

The mean ± SE values of monocyte count (per cent)of healthy control camels and in diseased camels subjected to different treatment are presented in Table 11. The data of monocyte (per cent) is presented in Table viii – x of appendix.

Table: 11 Mean ±SE values of monocyte count (%) of camels of different groups (n=10).

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$3.6 \pm 0.22^{a}{}_{a}$	$4.8 \pm 0.55^{b}{}_{b}$	$4.4 \pm 0.22^{b}{}_{b}$
10th day	$3.6 \pm 0.22^{a}{}_{a}$	$3.3 \pm 0.33^{a}{}_{b}$	$3.5 \pm 0.16^{b}{}_{b}$
20th day	$3.6 \pm 0.22^{a}{}_{a}$	$3.6 \pm 0.40^{a}{}_{b}$	$2.4 \pm 0.16^{a}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The mean ±SE values for healthy control was 3.6 ± 0.22 %. The prtreatment and post-treatment (20th day) mean ± SE value for Group- I were 4.8 ± 0.55 and 3.6 ± 0.40 %, respectively. Such values for Group- II were 4.4 ± 0.22 and 2.4 ± 0.16 %, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was no significant difference of G-I, 20th day value as compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value had no significant (P ≤ 0.01) difference. The value on 20th day was significantly lower than pre-treatment value. As there was no significant difference of G-II, 20th day value compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

There were increase in number of monocyte because at the site of infection monocyte reach in more number and convert into macrophages. Macrophages are responsible for protecting tissues from foreign substances (Ziegler-Heitbrock, L. 2007).

The above findings are in agreement with the findings of Sena et al. *(1999) and Rathor (2006).*

(iv) Eosinophil count (%)

The mean ± SE values of eosinophil count (%) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 12. The data of eosinophil count. (%) is presented in Table viii – x of appendix.

Table: 12 Mean ±SE values of eosinsphil count (%) of camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$3.7 \pm 0.3^{a}{}_{a}$	$13.2 \pm 1.16^{c}{}_{c}$	$11.2 \pm 0.61^{c}{}_{b}$
10th day	$3.7 \pm 0.3^{a}{}_{a}$	$8.6 \pm 1.34^{b}{}_{c}$	$5.8 \pm 0.46^{b}{}_{b}$
20th day	$3.7 \pm 0.3^{a}{}_{a}$	$5.0 \pm 0.57^{a}{}_{b}$	$2.9 \pm 0.23^{a}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row diffe significantly (P≤0.01).

The mean ± SE value for healthy control was 3.7 ± 0.31 %. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 13.2 ± 1.16 and 5.0 ± 0.57 %, respectively. Such values for Group- II were 11.2 ± 0.61 and 2.9 ± 0.23 %, respectively.

G -I pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was no significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was no significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

Increase in total eosinophlic count may be due to hyper sensitivity developed due to allergic reactions which in turn lead to increased number of eosinophil (Radostits et al. 2007). The post treatment decrease in total eosinophilic count may be due the effect of drug (Dixit et al. 2004).

The above findings are in agreement with the findings of Mourad et al. (1987), Sena et al (1999), Singh and Gahlot (2000), Kataria and Kataria (2004), Parmar, A.J. (2005) and Rathore (2006).

(v) Basophil count (%)

The mean ± SE values of basophill count (%) of healthy control camels and in diseased camels subjected to different treatment presented in Table 13. The data of basophill count (%) is presented in Table viii – x of appendix.

Table: 13 Mean ± SE value of basophil count (%) camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	0.6 ± 0.16	0.4 ± 0.16	0.5 ± 0.16
10th day	0.6 ± 0.16	0.4 ± 0.16	0.6 ± 0.16
20th day	0.6 ± 0.16	0.4 ± 16	0.3 ± 0.15

There was no significant difference of G-I and G-II values with the healthy control and also value within the groups.

Biochemical parameters

Note:- The value of biochemical Parameters of healthy control camels were taken once only. Same values have been presented on 10th and 20th day day for the purpose of comparision with G-I and G-II group values.

(1) Serum glucose (mg/dl)

The mean ± SE values of serum glucose (mg/dl) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 14. The data of serum glucose (mg/dl) is presented in Table xi – xiii of appendix.

Table:14 Mean ± SE values of Serum glucose (mg/dl) of camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$81.0 \pm 1.28^{a}{}_{c}$	$58.1 \pm 2.26^{a}{}_{a}$	$62.3 \pm 0.84^{a}{}_{b}$
10th day	$81.0 \pm 1.28^{a}{}_{c}$	$66.9 \pm 2.34^{b}{}_{a}$	$76.5 \pm 1.24^{b}{}_{b}$
20th day	$81.0 \pm 1.28^{a}{}_{b}$	$74.4 \pm 1.65^{c}{}_{a}$	$82.0 \pm 0.69^{c}{}_{b}$

Means with different superscript in a column differ significantly (P≤ 0.01).
Means with different subscript in a row differ significantly (P≤ 0.01).

The camels suffering from sarcopticosis had significant lower value of glucose compared to healthy control camels.

The mean ± SE value for healthy control (HC) was 81.0 ± 1.28 mg/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group–I were 58.1 ± 2.26 and 74.4 ± 1.65 mg/dl, respectively. Such values for Group–II were 62.3 ± 0.84 and 82.0 ± 0.69 mg/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals could not attain normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day

was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significant difference of G-II, 20th day compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

The 20th day value of G-I was significantly (P ≤ 0.01) lower as compared to healthy control while this value in G-II group had no significant difference and almost attained normalacy as compared to healthy control indicating a better recovery in G-II group animals.

The reduced blood glucose levels were seen in all mange affected camels which might be the result of poor consumption of feed and other nutrients. After treatment, the blood glucose levels were elevated and almost attained the normal values.

These findings are in agreement with the the findings of Ibrahim et al. (1981), Radwan et al. (1987), Singh and Gahlot (2000), Mathur (2004), Parmar, A.J. (2005) and Abdally, M.H. (2010).

(2) Total serum protein (g/dl)

The mean ± SE values of Total serum protein (g/dl) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 15. The data of total serum protein (g/dl) is presented in Table xi – xiii of appendix.

Table: 15 Mean ±SE values of total protein (gldl) of camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$7.88 \pm 0.12^{a}{}_{c}$	$6.81 \pm 0.17^{a}{}_{b}$	$6.18 \pm 0.11^{a}{}_{a}$
10^{th} day	$7.88 \pm 0.12^{a}{}_{a}$	$7.44 \pm 0.13^{b}{}_{a}$	$7.56 \pm 0.12^{b}{}_{a}$
20^{th} day	$7.88 \pm 0.12^{a}{}_{a}$	$8.11 \pm 0.08^{c}{}_{a}$	$8.17 \pm 0.05^{c}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camels suffering from sarcopticosis had significant lower value of total protein compared to healthy control camels.

The mean ± SE value for healthy control (HC) was 7.88 ± 0.12 g/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 6.81 ± 0.17 and 8.11 ± 0.08 g/dl, respectively. Such values for Group- II were 6.18 ± 0.11 and 8.17 ± 0.05 g/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. The G-I 20th day value attained normalacy with no significant (P ≤ 0.01) difference as compared to healthy control.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was no significantly (P ≤ 0.01) difference of G-II, 20th day value compared to healthy control and become even better than the protein value of healthy control animals.

Low level of total protein in affected animals may be the result of poor consumption of feed and other nutrients and might be due to the habbit of the mites of chewing epidermal layer of the skin (Arlian et al. *1988).*

These findings are in agreement with Ibrahim et al. (1981), Radwan et al. (1987), Singh and Gahlot (2000), Mathur (2004), Parmar, A.J. (2005), Rathore (2006) and Kamal, A.M. (2008).

(3) Albumin (g/dl)

The mean ± SE values of Albumin (g/dl) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 16. The data of Albumin (g/dl) is presented in Table xi – xiii of appendix.

Table: 16 Mean ±SE values of Albumin of camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Doramectin)***
Pre-treatment	$4.31 \pm 0.10^{a}{}_{b}$	$2.97 \pm 0.11^{a}{}_{a}$	$2.98 \pm 0.11^{a}{}_{a}$
10^{th} day	$4.31 \pm 0.10^{a}{}_{a}$	$3.17 \pm 0.13^{a}{}_{b}$	$3.66 \pm 0.08^{b}{}_{b}$
20^{th} day	$4.31 \pm 0.10^{a}{}_{a}$	$3.64 \pm 0.10^{b}{}_{b}$	$4.09 \pm 0.10^{c}{}_{c}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camels suffering from sarcopticosis had significant lower value of Albumin compared to healthy control camels.

The mean ±SE value for healthy control (HC) was 4.31 ± 0.10 g/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 2.97 ± 0.11 and 3.64 ± 0.10 g/dl, respectively. Such values for Group- II were 2.98 ± 0.11 and 4.09 ± 0.10 g/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10^{th} day as compared to pre-treatment value had no significant variation. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value.The G-I, 20^{th} day value was significantly (P ≤ 0.01) lower than healthy control meant that the G-I animals could not attain normalacy on 20^{th} day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was significantly (P ≤ 0.01) lower value of G-II, 20th day as compared to healthy control, meant that the G-II animals could not attained normalacy on 20th day of treatment.

The 20th day value of G-I and G-II had significant (P ≤ 0.01) variation and the value attained in G-II were more nearer to the healthy control group indicating better recovery in G-II animals.

There was decrease value of albumin because albumin is the fraction of total protein and there were decreased total protein value so albumin value decreased and stress conditions also caused decrease in albumin value.

Above findings as in agreement with Singh and Gahlot (2000), Dongre (2000), Mali (2002), Parmar, A.J. (2005), Rathore (2006) and Kamal, A.M. (2008).

(4) Globulin (g/dl)

The mean ± SE values of Globulin (g/dl) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 17. The data of Globulin (g/dl) is presented in Table xi – xiii of appendix.

Table: 17 Mean ± SE values of Globulin of camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre–treatment	$3.65 \pm 0.13^{a}{}_{c}$	$3.43 \pm 0.21^{a}{}_{a}$	$3.20 \pm 0.08^{a}{}_{b}$
10th day	$3.65 \pm 0.13^{a}{}_{a}$	$4.26 \pm 0.16^{b}{}_{c}$	$3.89 \pm 0.09^{b}{}_{b}$
20th day	$3.65 \pm 0.13^{a}{}_{a}$	$4.49 \pm 0.14^{b}{}_{b}$	$4.14 \pm 0.07^{b}{}_{b}$

Means with different superscript in a column differ significantly (P≤ 0.01).
Means with different subscript in a row differ significantly (P≤ 0.01).

The mean ± SE value for healthy control (HC) was 3.65 ± 0.13 g/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 3.43 ± 0.21 and 4.49 ± 0.14 g/dl, respectively. Such values for Group- II were 3.20 ± 0.08 and 4.14 ± 0.07 g/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher.The value on 20th day was significantly higher than pre-treatment value. As there was significantly (P ≤ 0.01) higher value of G-I, 20th day as compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was significantly (P ≤ 0.01) higher value of globulin of G-II, 20th day as compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment. Decreased level of globulin was because of stress condition and thereafter because of stimulation of immune system and induction of immunoglobulins there was increased value of globulin.

The above findings are in agreement with the findings of Mahran, O.M. (2004), Parmar, A.J. (2005) and Rathore (2006).

(5) A: G ratio

The mean ± SE values of A: G ratio of healthy control camels and in diseased camels subjected to different treatment are presented in Table 18. The data of A: G ratio is presented in Table xi – xiii of appendix.

Table: 18 Mean ± SE values of A:G ratio of camels of different groups (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)

Pre-treatment	*1.18 ± 0.04$^{a}{}_{b}$*	*0.829 ± 0.06$^{a}{}_{a}$*	*0.94 ± 0.5$^{a}{}_{a}$*
10th day	*1.18 ± 0.04$^{a}{}_{b}$*	*0.76 ± 094$^{a}{}_{a}$*	*0.93 ± 0.03$^{a}{}_{a}$*
20th day	*1.18 ± 0.04$^{a}{}_{b}$*	*0.815 ± 0.04$^{a}{}_{a}$*	*0.97 ± 0.03$^{b}{}_{a}$*

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camels suffering from sarcopticosis had significant lower value of A:G ratio compared to healthy control camels.

The mean ± SE value for healthy control (HC) was 1.18 ± 0.04 g/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 0.829 ± 0.06 and 0.815 ± 0.04 g/dl, respectively. Such values for Group- II were 0.94 ± 0.5 and 0.97 ± 0.03 g/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value had no significant (P ≤ 0.01) variation. The value on 20th day as compared to pre-treatment value had no significant variation. As there was significant (P ≤ 0.01) lower value of G-I, 20th day value compared to healthy control, meant that the G-I animals could not attain normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value had no significant variation. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was significant (P ≤ 0.01) lower value of G-I, 20th day value compared to healthy control, meant that the G-I animals could not attain normalacy on 20th day of treatment.

The 20th day value of G-II was more nearer to healthy control as compared to G-I group, indicating better recovery in G-II group animals.

There was decreased value of A:G ratio because in mange infestation albumin value decreased more as compared to globulin so A:G ratio will be decreased.

Above findings are in agreement with findings of Singh and Gahlot (2000), Dongre (2000), Mali (2002) and Rathore (2006).

(6) Serum urea (mg/dl)

The mean ± SE values of serum urea (mg/dl) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 19. The data of serum urea (mg/dl) is presented in Table xi – xiii of appendix.

Table: 19 Mean ± SE value of serum urea (mg/dl) in camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Doramectin)***
Pre-treatment	*20.78 ± 1.03$^{a}{}_{b}$*	*11.75 ± 0.53$^{a}{}_{a}$*	*12.77 ± 0.40$^{a}{}_{a}$*
10th day	*20.78 ± 1.03$^{a}{}_{b}$*	*15.08 ± 0.45$^{b}{}_{a}$*	*16.61 ± 0.47$^{b}{}_{a}$*
20th day	*20.78 ± 1.03$^{a}{}_{b}$*	*17.18 ± 0.40$^{b}{}_{a}$*	*19.31 ± 0.51$^{c}{}_{b}$*

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camels suffering from sarcopticosis had significant lower value of serum urea compared to healthy control camels.

The mean ± SE value for healthy control (HC) was 20.78 ± 1.03 mg/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 11.75 ± 0.53, and 17.18 ± 0.40 mg/dl, respectively. Such values for Group- II were 12.77 ± 0.40, and 19.31 ± 0.51 mg/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. There was significant difference of G-I, 20th day value as compared to healthy control, meant that the G-I animals could not attain normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. There was no significant difference of G-II, 20th day as compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

The 20th day post-treatment value of G-II group attained normalacy, indicating a better response of drug in G-II animals as compared to G-I animals.

Decreased serum urea values may be attributed to the decreased feed intake in the affected animals as the blood urea concentration varies according to the levels of protein in ration (Mohapatra et al., 1994,1995).

These findings are in agreement with the findings of Mohapatra et al.(1994,1995). Singh et al. (2003) and Abdally, M.H. (2010).

(7) Serum creatinine (mg/dl)

The mean ± SE values of serum creatinine (mg/dl) of healthy control camels and in diseased camels subjected to different treatment are presented in Table 20. The data of serum creatinine (mg/dl) is presented in Table xi – xiii of appendix.

Table: 20 Mean ±SE value of serum creatinine (mg/dl) in camels of different group (n=10)

Treatment	HC (Healthy control)	G-I (Ivermectin)	G-II (Doramectin)
Pre-treatment	$2.02 \pm 0.18^{a}{}_{a}$	$3.92 \pm 0.14^{c}{}_{b}$	$3.88 \pm 0.18^{c}{}_{b}$
10^{th} day	$2.02 \pm 0.18^{a}{}_{a}$	$3.18 \pm 0.11^{b}{}_{b}$	$3.02 \pm 0.11^{b}{}_{b}$
20^{th} day	$2.02 \pm 0.18^{a}{}_{ab}$	$2.46 \pm 0.13^{a}{}_{b}$	$1.92 \pm 0.10^{a}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camels suffering from sarcopticosis had significant (P ≤ 0.01) higher value of serum creatinine as compared to healthy control camels.

The mean ±SE value for healthy control (HC) was 2.02 ± 0.18 mg/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 3.92 ± 0.14 and 2.46 ± 0.13 mg/dl, respectively. Such values for Group-II were 3.88 ± 0.18 and 1.92 ± 0.10 mg/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was no significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals attained normalacy on 20th day of treatment.

G-II pre-tratment value was significantly (P ≤ 0.01) higher than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) lower. The value on 20th day was significantly (P ≤ 0.01) lower than pre-treatment value. As there was no significant difference of G-II, 20th day compared to healthy control, meant that the G-II animals attained normalacy on 20th day of treatment.

Creatinine level increase in muscle disorder in which there is increased breakdown of muscle cell or reduced muscle mass.The mite burrow and make tunnels in the skin causing severe irritation to the animals. The animals bite, scratch and rub the affected parts with inanimate objects injury to muscles, which probably causes rise in serum creatinine levels in the affected animals (Mohapatra et al., 1995).

These findings are in agreement with the findings of Mohapatra et al.(1995), Singh et al. (2003), Kamal, A.M. (2008) and Abdally, M.H. (2010).

(8) Serum zinc (µg/dl)

The mean ± SE values of serum zinc (µg/dl) of healthy control camels and in mangy camels subjected to different treatment have been presented in Table 21. The data of serum zinc (µg/dl) is presented in Table xi – xiii of appendix.

Table: 21 Mean ±SE value of zinc (µg/dl) of camels of different groups (n=10)

Treatment	***HC (Healthy control)***	***G-I (Ivermectin)***	***G-II (Doramectin)***
Pre-treatment	$105.32 \pm 3.02^{a}{}_{b}$	$81.21 \pm 1.61^{a}{}_{a}$	$79.84 \pm 1.26^{a}{}_{b}$
10th day	$105.32 \pm 3.02^{a}{}_{b}$	$85.98 \pm 1.43^{b}{}_{a}$	$85.84 \pm 1.14^{b}{}_{a}$
20th day	$105.32 \pm 3.02^{a}{}_{b}$	$89.18 \pm 1.36^{b}{}_{a}$	$91.50 \pm 1.06^{b}{}_{a}$

Means with different superscript in a column differ significantly (P≤0.01).
Means with different subscript in a row differ significantly (P≤0.01).

The camels suffering from sarcopticosis had significant (P ≤ 0.01) lower value of serum urea compared to healthy control camels.

The mean ± SE value for healthy control (HC) was 105.32 ± 3.02 µg/dl. The pre-treatment and post-treatment (20th day) mean ± SE value for Group- I were 81.21 ± 1.61and 89.18 ± 1.36 µg/dl, respectively. Such values for Group- II were 79.84 ± 1.26 and 91.90 ± 1.06 µg/dl, respectively.

G-I pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly (P ≤ 0.01) higher than pre-treatment value. As there was significant difference of G-I, 20th day value compared to healthy control, meant that the G-I animals could not attain normalacy on 20th day of treatment.

G-II pre-treatment value was significantly (P ≤ 0.01) lower than healthy control animals. Within the group, the value on 10th day as compared to pre-treatment value was significantly (P ≤ 0.01) higher. The value on 20th day was significantly higher than pre-treatment value. As there was significant difference of G-I, 20th day value compared to healthy control, meant that the G-II animals could not attain normalacy on 20th day of treatment.

The 20th day value of both group were significantly (P ≤ 0.01) lower as compared to healthy control but G-II value was more nearer to healthy control group, indicating better recovery in G-II animals.

Decreased serum zinc values may be attributed to the zinc being utilized by leucocytes to fight various infections and infestations in the body leading to lower level of zinc. Thus, lower level of serum zinc in mangy camel might be due to utilization of zinc by blood cells to fight the mites and secondary bacterial infections (Mal et al. 2002).

These findings are in agreement with the findings of Mal et al. (2002), Singh et al. (2003) and Dixit et al. (2000).

SUMMARY

A total of 30 camels (of either sex and different age groups) were taken for study, out of which ten camels were taken as control and rest 20 mange affected camels were divided into two groups of ten each. The first diseased group (G-I) of ten camels was treated with injection ivermectin given at the dose rate of 1ml/50 kg. body weight subcutaneously and repeated at an interval of 10 days. Similarly the second group (G-II) of mange affected camels was treated with injection doramectin given at the dose rate of 1ml/50 kg. body weight intramuscularly and repeated at an interval of 10 days. Comparative efficacy of ivermectin with doramectin was seen with regard to clinical, haematological and biochemical parameters before and after treatment.

The clinical manifestations varied from camel to camel in all the two groups except healthy control animals. The gray coloured gross skin lesion having intense irritation due to itching, alopecia, thickening and corrugation were present on the face, neck, limbs, ventral and lateral part of abdomen. The lesions also showed hyper pigmentation, hyperemia and severe scratching with oozing of blood which could possibly be due to itching or intense irritation caused by ectoparasite penetrating the skin in turn provoking the animal to rub its body against rough objects like trees walls, electric poles etc. Self mutilation by mouth was also noticed. Deterioration of quality of skin, Keratinization and leathery appearance of skin was also recorded.

Among haematological parameters Haemoglobin, PCV, TEC, neutrophil count and monocyte count had decreased significantly ($P \leq 0.01$) in mange affected camels as compared to healthy control camels, whereas the total eosinophilic count and eosinophil count (DLC) increased significantly ($P \leq 0.01$) in mange affected camels as compared to healthy control camels. The 20^{th} day post-treatment haematological study revealed that the recovery was better in doramectin treated group because they almost attained the normal haematological value.

Among biochemical parameters serum glucose, total protein, albumin, globulin A:G ratio, serum urea and serum zinc decreased significantly ($P \leq 0.01$) in mange affected camels as compared to healthy control camels, whereas serum creatinine increased significantly ($P \leq 0.01$) in mange affected camels as compared to healthy control camels. The 20^{th} day post-treatment study of diseased camels revealed that the clinical recovery was faster in doramectin treated group as compared to ivermectin treated group of animals. The disturbed biochemical parameters of mange affected camels almost attained normalacy in doramectin treated group as compared to ivermectin treated group of animals.

CONCLUSION

Comparative efficacy of ivermectin with doramectin was carried out with regard to clinical, haematological and biochemical recovery of mange affected camels. It was seen that the cases which were treated with ivermectin recovered satisfactorily with two doses of ivermectin repeated at an interval of 10 days but there were remenants of scar and rough hair coat in some of the cases while the doramectin treated animals either recovered in single dose or cured completely after the second dose which was also given after an interval of 10 days with subsided scar and roughness of skin. The haemato-biochemical study revealed that the recovery was fast in maximum parameters like haemoglobin, PCV, TEC, total eosinophil count, serum glucose, total protein and serum urea in doramectin treated group animals as compared to ivermectin treated group animals. It was also noted in this study that animals treated with doramectin felt more comfortable and docile. Bearing above results in mind, it can be concluded that Doremectin is superior to Ivermectin.

LITERATURE CITED

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Figure 5: Effect of treatment on haemoglobin content of blood

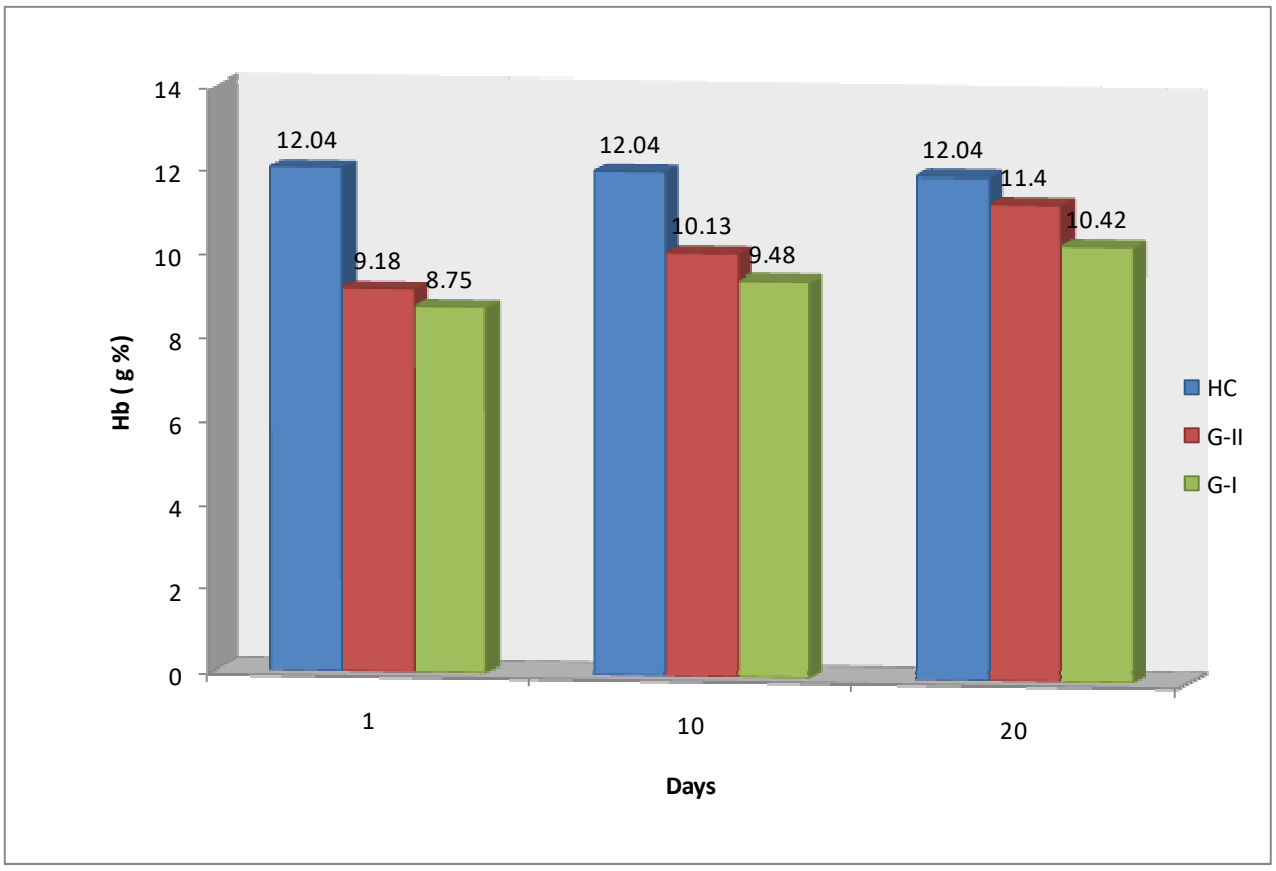

Figure 6: Effect of treatment on TEC count of blood

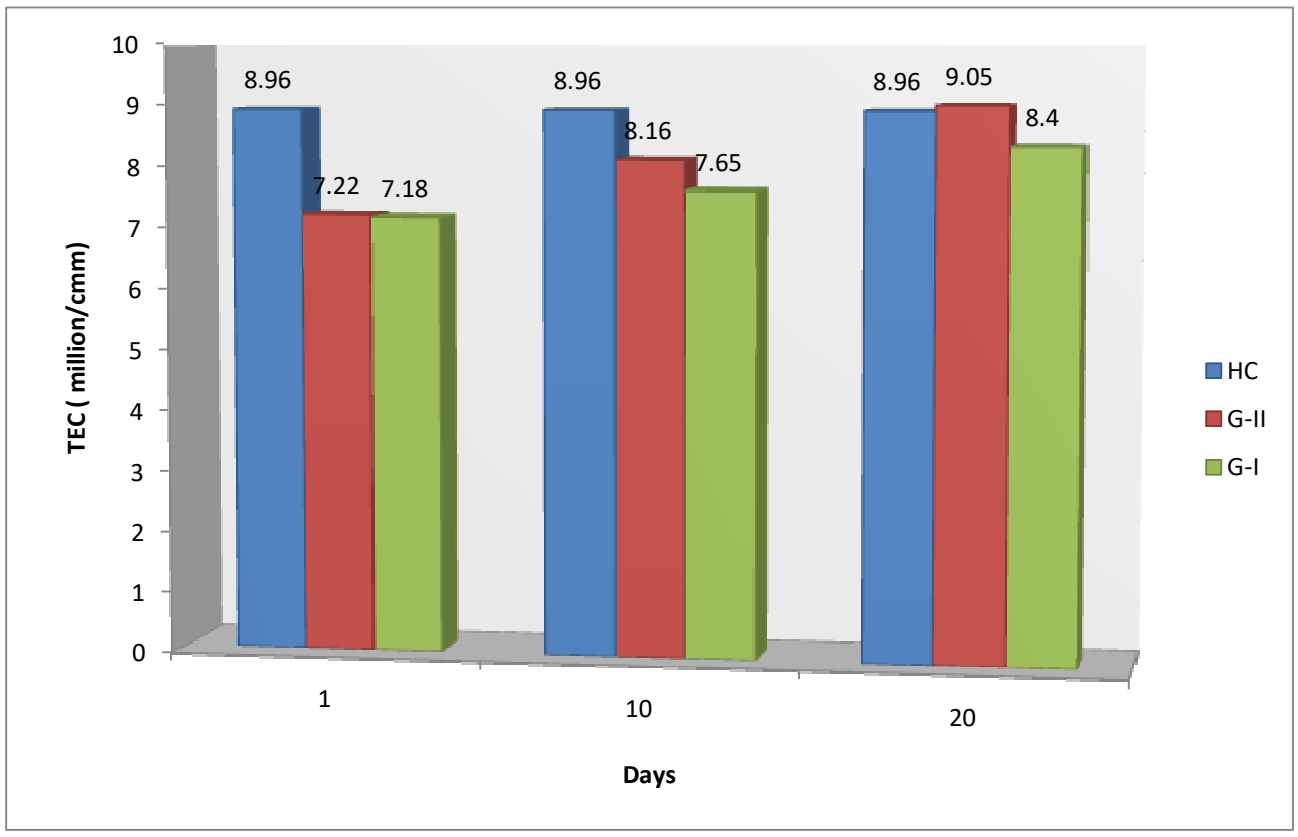

Figure: 7 Effect of treatment on total eosinophilic count of blood

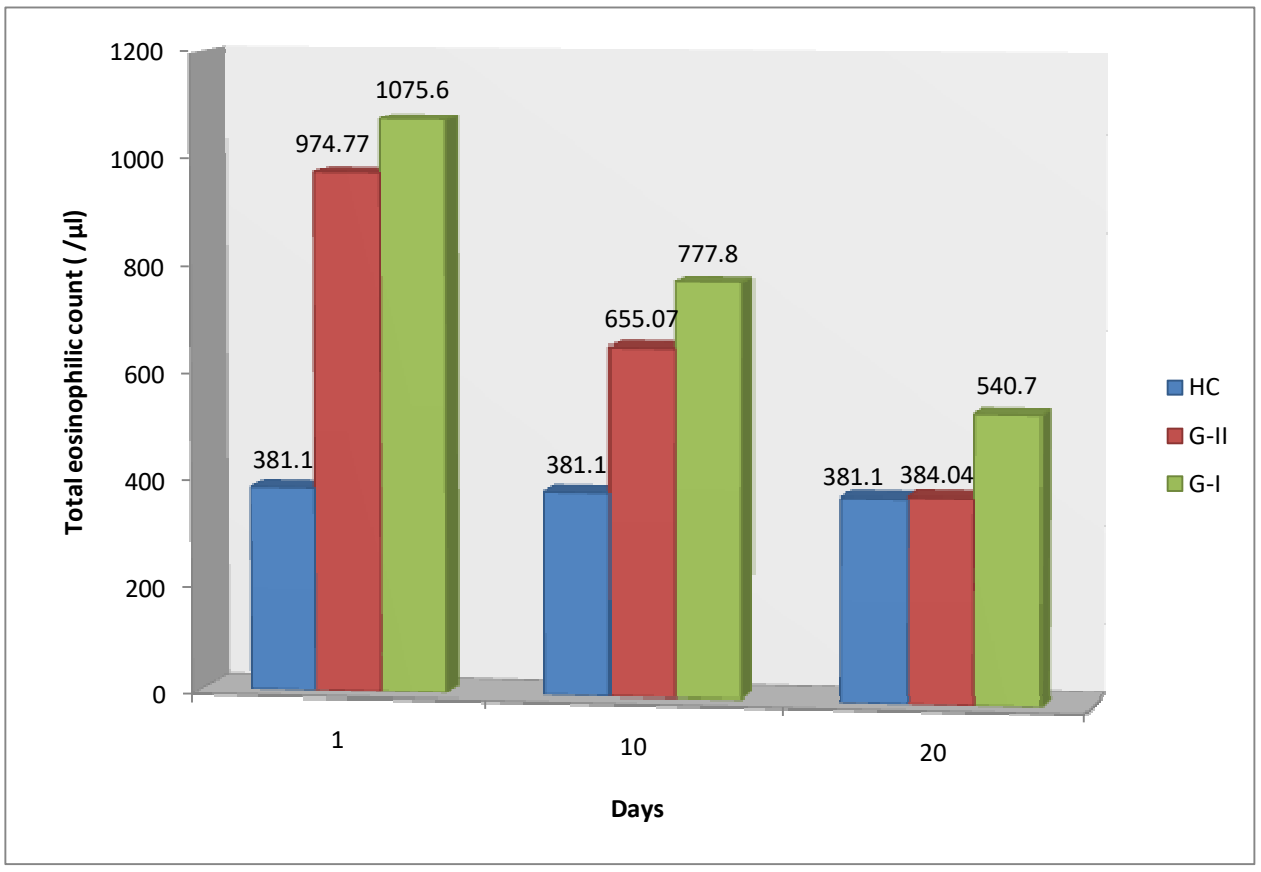

Figure: 8 Effect of treatment on eosinophilic count of blood

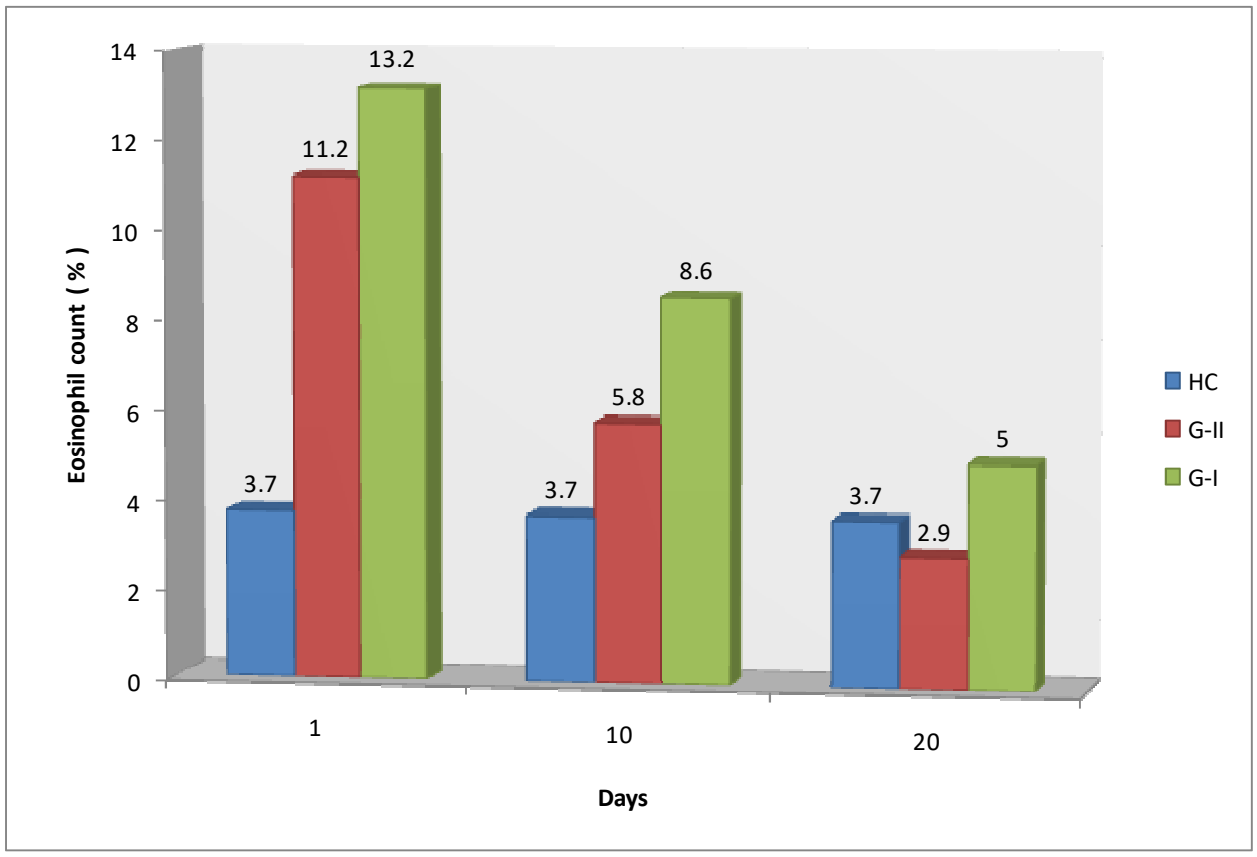

Figure: 9 Effect of treatment on serum glucose

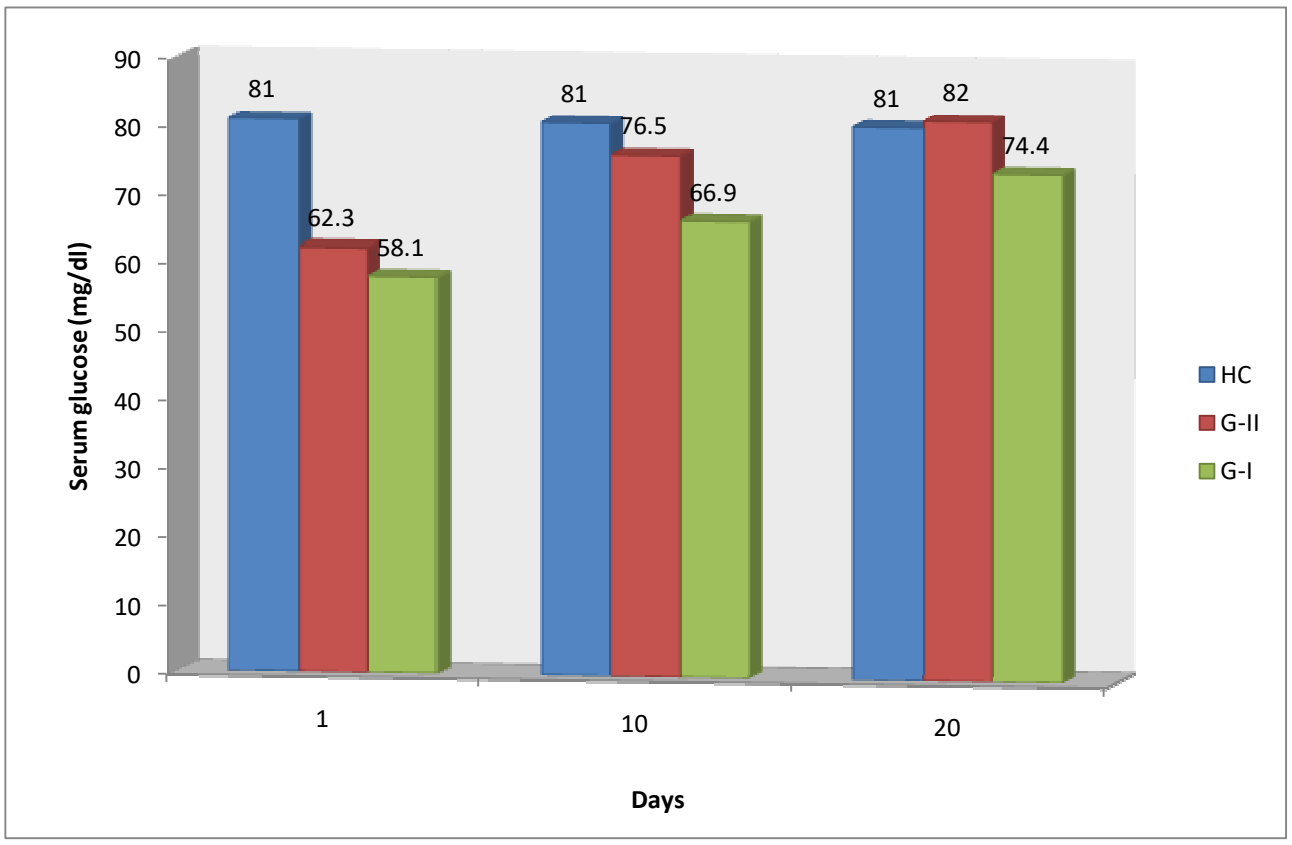

Figure: 10 Effect of treatment on serum total protein

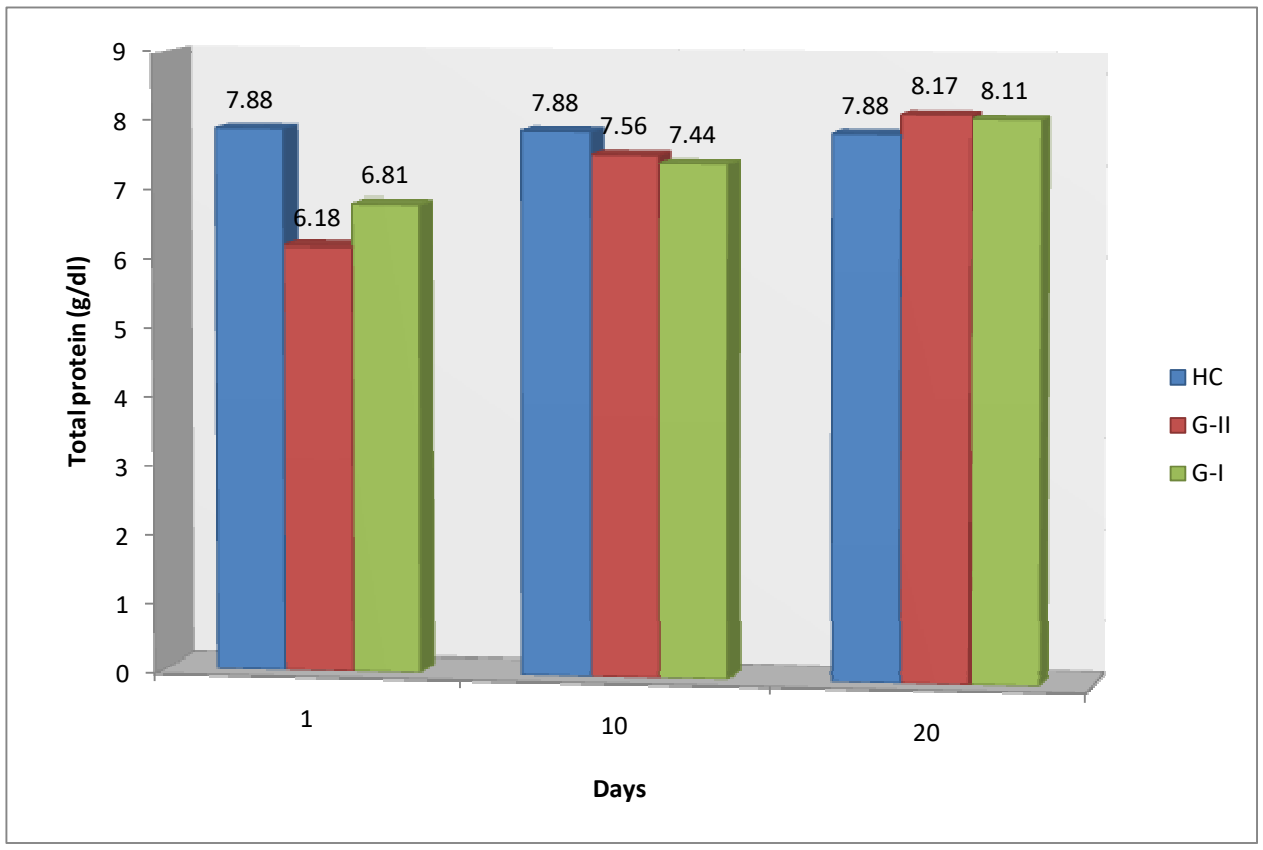

Figure: 11 Effect of treatment on serum Albumin

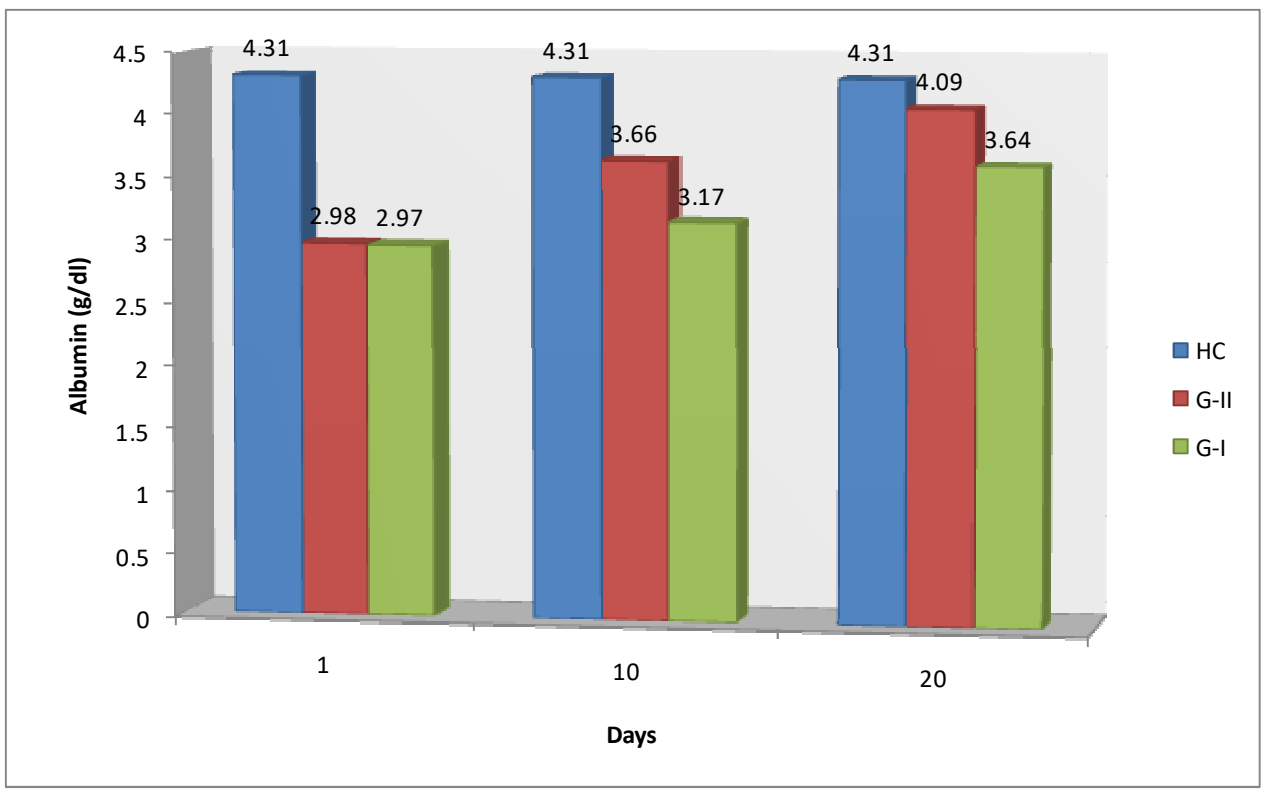

Figure: 12 Effect of treatment on serum urea

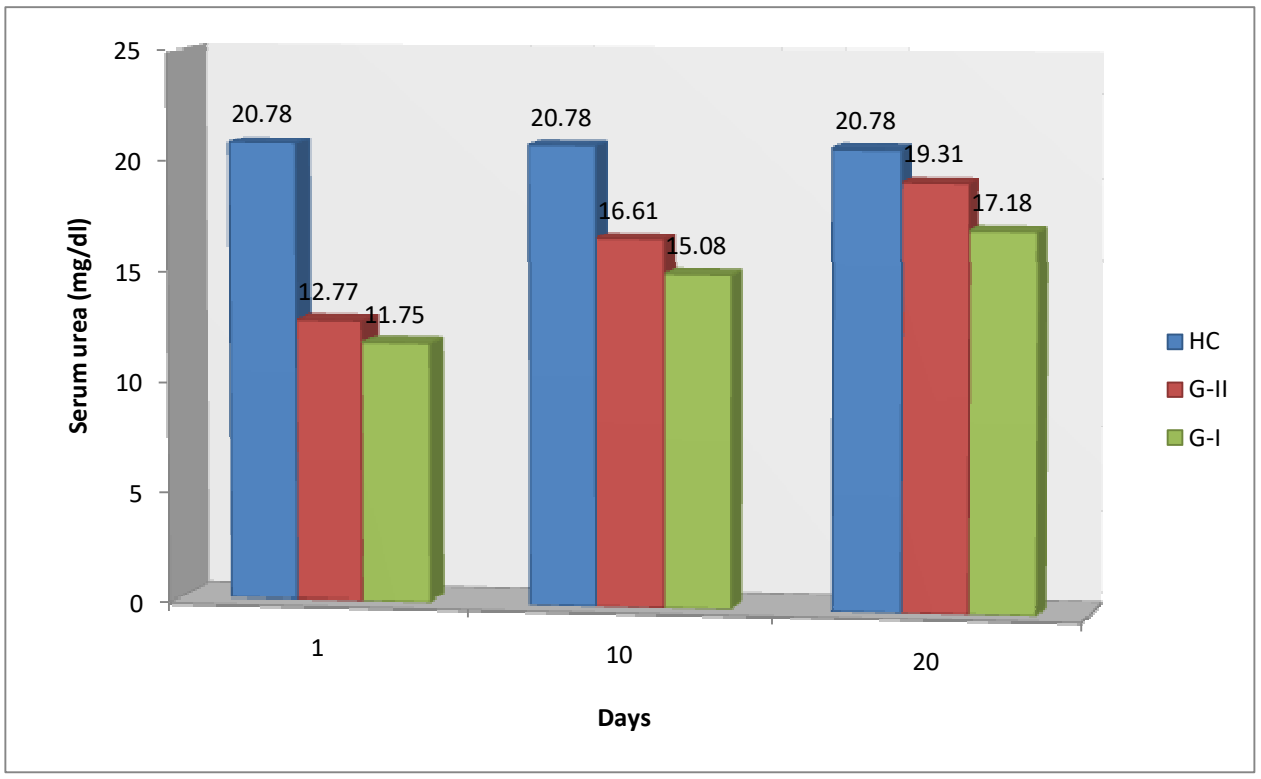

Figure: 13 Effect of treatment on serum creatinine

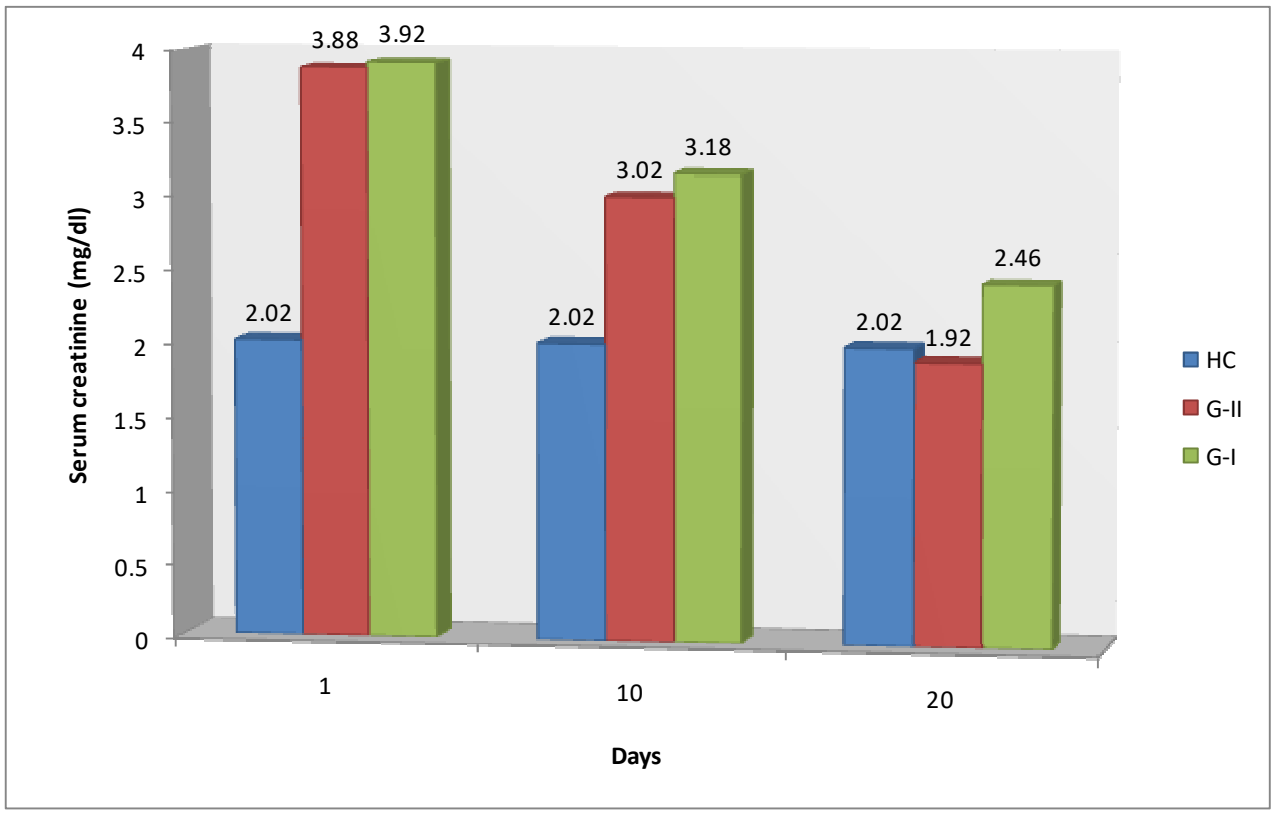

Figure: 14 Effect of treatment on serum zinc

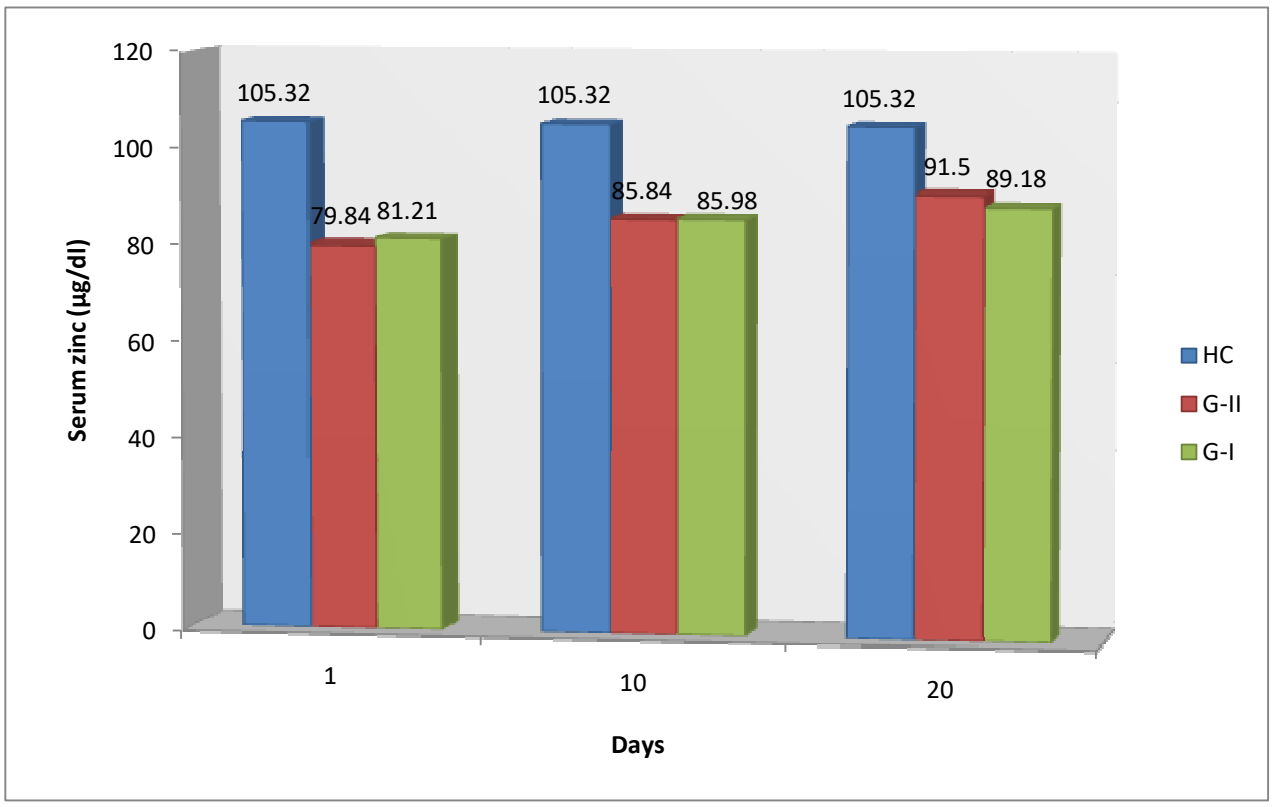

Fig. 1 Camel suffering from sarcopticosis showing lesions at the neck with alopecia

Fig. 2 Camel showing lesions of mange on the shoulder and chest region with self mutilated wound

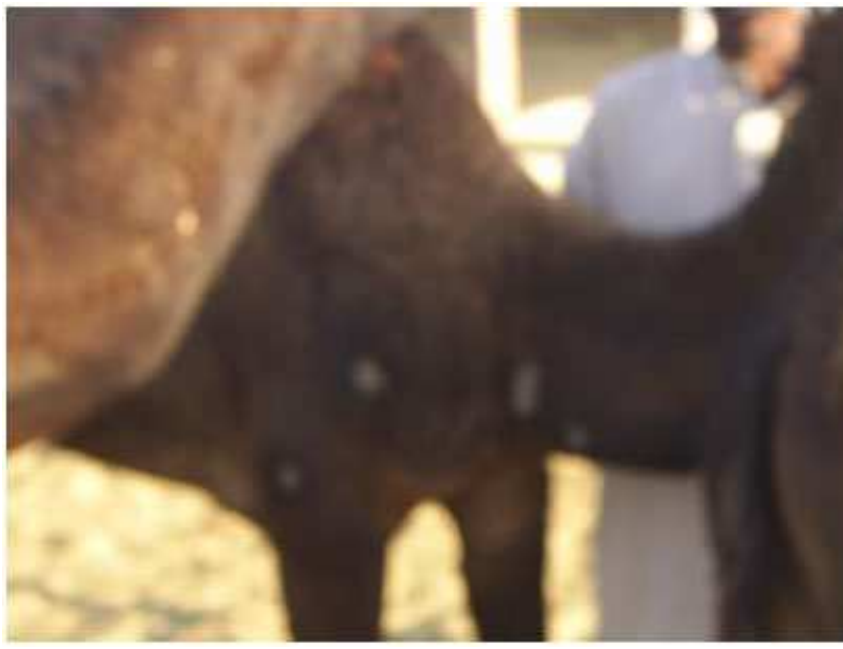

Fig. 3 Camel suffering from sarcopticosis showing lesions at the shoulder and neck

Fig. 4 Camel suffering from sarcopticosis showing lesions at the back region

Fig. 17 Camel showing lesions of mange at shoulder and abdomen region.

Fig. 18 Camel of G-II showing improvement in lesions of mange on shoulder and abdomen region after treatment with doramectin

Fig. 15 Lesions of mange on the shoulder and chest region with thickening of skin

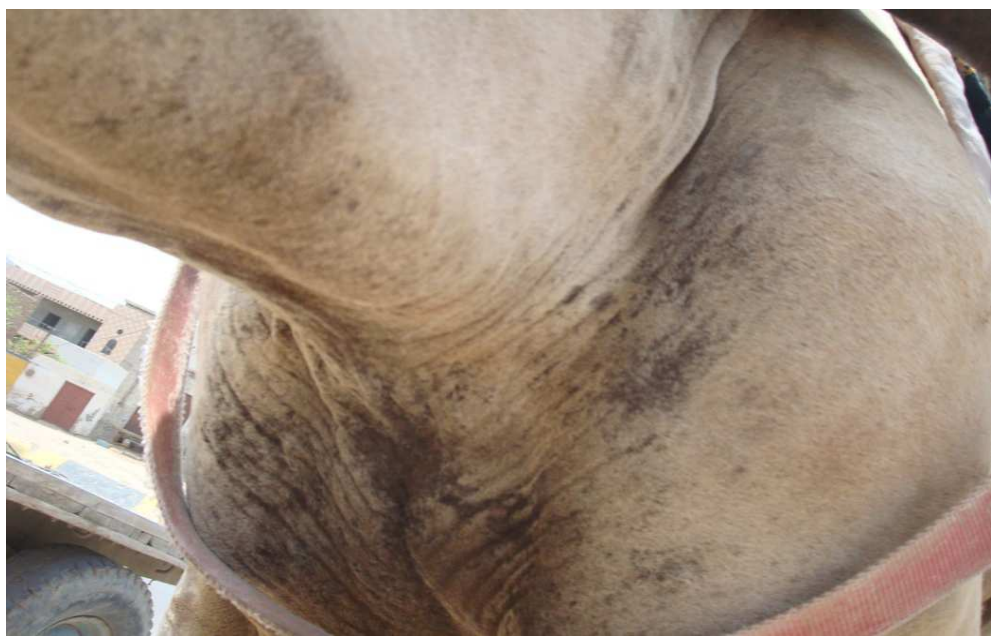

Fig. 16 Camel of G-I showing improvement in lesions of mange on the shoulder and chest region with thickening of skin after treatment with ivermectin

Table (i) values of temperature, pulse and respiration rate of apparently healthy control camels (HC)

A.NO.	Temperature (^{0}F)	Pulse (rate/minute)	Respiration (rate/minute)
1	97.2	42	13

2	96.4	44	13
3	96.8	42	16
4	97.0	44	14
5	97.0	42	13
6	97.2	46	16
7	97.4	44	13
8	96.6	42	12
9	98.0	47	13
10	97.2	42	13
Mean	97.08	43.50	13.60
Mean ±SE	97.08 ± 0.14	43.50 ± 0.58	13.60 ± 0.42

Table (ii) Values of temperature, pulse and respiration rate of camels suffering from sarcopticosis treated with 1% Ivermectin (G-1)

A no./day	**Temperature (^{0}F)**			**Pulse (rate/min.)**			**Respiration (rate/min.)**		
	1st	**10th**	**20th**	**1st**	**10th**	**20th**	**1st**	**10th**	**20th**
1	97.4	97.2	97.0	48	46	50	14	16	15
2	97.2	98.0	97.8	44	50	54	18	20	17
3	98.0	97.6	97.2	44	44	46	12	14	10
4	96.4	96.8	97.0	52	48	54	13	15	14
5	96.2	96.2	96.2	46	44	42	14	12	13
6	97.8	98.0	97.4	56	54	54	13	14	12
7	96.8	96.4	97.0	43	44	43	10	11	13
8	96.4	96.6	97.2	50	52	52	12	10	14
9	98.0	97.6	97.4	48	52	50	16	17	20
10	97.0	97.4	97.6	49	48	50	20	17	18
Mean	97.12	97.18	97.18	48.00	48.20	49.50	14.20	14.60	14.60
MEAN ± SE	97.12±0.21	97.18±0.20	97.18±0.13	48.00±1.27	48.20±1.17	49.50±1.40	14.20±0.95	14.60±0.96	14.60±0.94

Table (iii) Values of temperature, pulse and respiration rate of camels suffering from sarcopticosis treated with 1% Doramectin (G-II)

A no./day	Hb (gm %)			PCV (%)			TEC (Milllion/cmm)			TLC (Thousand/cmm)		
	Hb (gm%)			PCV (%)			TEC (Milllion/cmm)			TLC (Thousand/cmm)		
A no./day	1st	10th	20th	1st	10th	20th	1st	10th	20th	1	10	20
NO./Day	Temperature (F^0)			Pulse (rate/minute)						Respiration(rate/minute)		
1	9.6	9.8	10.4	28	29	32	7.60	7.70	8.40	8.42	8.68	8.84
2	9.4	10.0	10.8	26	31	32	7.58	7.88	8.80	8.32	8.96	9.70
3	8.2	8.9	9.8	26	28	30	6.76	7.48	8.10	7.86	8.64	9.32
4	8.8	10.2	11.8	24	31	34	7.30	8.30	8.90	9.40	9.94	10.0
5	7.8	8.4	11.4	22	26	33	6.50	7.40	8.30	7.81	8.20	9.02
6	9.6	10.8	12.0	26	34	38	7.72	8.48	9.80	8.53	9.20	9.91
7	9.4	10.6	11.6	26	32	34	7.32	7.98	9.42	7.24	8.46	9.10
8	9.8	10.8	11.8	28	35	36	8.52	8.80	10.0	8.02	8.91	10.01
9	8.8	9.6	10.8	27	28	32	5.90	6.82	8.34	7.80	8.80	9.63
10	10.4	11.6	12.2	32	34	39	6.33	7.82	8.68	7.36	8.23	9.36
Mean	9.6	10.4	11.6	28.30	32.20	34	8.36	9.84	10.30	8.10	8.30	9.60
Mean±SE	9.18±0.24	10.13±0.30	11.44±0.24	27.40±0.77	30.40±0.74	34.60±1.04	7.22±0.27	8.16±0.30	9.05±0.23	7.89±0.22	8.86±0.20	9.56±0.11

Table (iv) values of haematological parameters of apparently healthy control camels (HC)

A.NO.	Hb (gm %)	PCV (%)	TEC (million/cmm)	TLC (thousand/cmm)
1	12.6	38	9.42	8.97
2	13.8	37	9.90	8.33
3	10.4	32	8.30	9.30
4	11.4	34	8.20	9.20
5	12.6	38	9.60	8.69
6	13.2	38	9.88	8.67
7	10.4	32	8.00	9.00
8	11.4	34	8.88	8.60
9	11.8	35	9.44	8.91
10	12.8	37	8.01	8.77
Mean	12.04	35.50	8.96	8.84
Mean±SE	12.04±0.36	35.50±0.76	8.96±0.24	8.84±0.09

Table (v) Values of haematological parameters of camels suffering from sarcopticosis treated with 1% Ivermectin (G-1)

Table (vi) Values of haematological parameters of camels suffering from sarcopticosis treated with 1% Doramectin (G-2)

Table (vii) Values of total eosinophil count (/µl) of various groups

A.NO./Day	Healthy control (HC)	Ivermectin (G-1)			Doramectin (G-2)		
		1st	10th	20th	1st	10th	20th
1	370	992	885	650	1023	732	460
2	376	1120	865	550	968	584	365
3	358	1310	792	665	1227	844	462
4	384	980	685	395	892	536	372
5	392	1170	830	398	848	475	325
6	402	1020	796	570	1004.7	706.70	402.49
7	382	1264	810	640	904	710	336
8	385	898	510	380	935	636	423
9	388	1042	775	535	1190	745	380
10	374	960	830	624	756	582	315
Mean	381.1	1075.6	777.8	540.7	974.77	655.07	384.04
Mean ±SE	381.1±3.88	1075.6±43.02	777.8±34.39	540.7±35.36	974.77±46.01	655.07±35.39	384.04±16.59

Table (viii) values of differential leucocyte count (%) of apparently healthy control camels (HC)

A.NO.	Neutrophil	Lymphocyte	Monocyte	Eosinophil	Basophil
1	51	41	5	3	1
2	40	38	3	4	0
3	51	41	4	2	1
4	52	36	3	5	1
5	51	35	4	4	0
6	53	44	3	3	0
7	53	42	3	5	1
8	51	37	4	4	1
9	46	38	3	3	0
10	53	39	4	4	1
Mean	50.1	39.1	3.6	3.7	0.6
Mean ±SE	50.1±1.29	39.1±0.9	3.6±0.22	3.7±0.3	0.6±0. 16

Table (ix) Values of differential leucocyte count (%) of camels suffering from sarcopticosis treated with 1% Ivermectin (G-1)

A no./day	Neutrophil			Lymphocyte			Monocyte			Eosinophil			Basophil		
	1st	10th	20th	1st	10th	20th	1st	10th	20	1st	10th	20th	1st	10th	2
1	43	45	48	36	38	40	6	4	3	15	13	8	0	0	
2	44	46	50	38	42	38	4	4	6	14	8	6	0	0	
3	42	47	51	36	40	37	5	2	5	17	10	6	0	1	
4	40	45	47	40	42	43	7	4	4	12	9	5	1	0	
5	46	49	50	40	41	41	3	2	4	10	7	5	1	1	
6	39	43	48	46	45	47	4	5	2	11	7	3	0	0	
7	44	46	49	32	37	42	3	4	4	20	15	7	1	0	
8	41	44	45	47	49	50	5	3	3	7	4	2	0	0	
9	45	48	51	37	42	43	3	2	2	14	7	4	1	1	
10	42	47	49	38	43	44	8	3	3	12	6	4	0	1	
Mean	42.6	46.0	48.8	39.0	41.9	42.5	4.8	3.3	3.6	13.2	8.6	5.0	0.4	0.4	
Mean ±SE	42.6± 0.70	46.0± 0.57	48.8± 0.59	39.0± 1.44	41.9± 1.07	42.5± 1.24	4.8± 0.55	3.3± 0.33	3.6± 0.40	13.2± 1.16	8.6± 1.34	5.0± 0.57	0.4± 0.16	0.4± 0.16	

Table (x) Values of differential leucocyte count (%) of camels suffering from sarcopticosis treated with 1% Doramectin

A no./day	Neutrophil			Lymphocyte			Monocyte			Eosinophil			Basophil		
	1st	10th	20th	1st	10th	20th	1st	10th	20	1	10	20	1	10	2
1	42	46	50	43	41	44	3	4	2	12	8	4	0	1	0
2	44	47	51	43	45	45	5	4	2	8	4	2	0	0	0
3	44	49	52	38	41	42	4	3	3	14	6	3	0	1	0
4	41	43	47	44	46	48	4	4	2	10	5	3	1	1	0
5	40	45	48	43	45	45	5	4	3	11	5	3	1	1	1
6	45	49	53	38	40	41	4	3	2	13	8	4	0	0	0
7	42	45	49	42	46	45	5	3	3	10	5	2	1	1	1
8	39	44	50	44	46	46	5	4	2	12	6	2	0	0	0
9	43	47	51	43	46	44	4	3	2	9	4	3	1	0	0
10	44	48	50	37	41	43	5	3	3	13	7	3	1	1	1
Mean	42.5	46.3	50.1	41.5	43.7	44.3	4.4	3.5	2.4	11.2	5.8	2.9	0.5	0.6	0
Mean ±SE	42.5± 0.61	46.3± 0.65	50.1± 0.56	41.5± 0.85	43.7± 0.81	44.3± 0.63	4.4± 0.22	3.5± 0.16	2.4± 0.16	11.2± 0.61	5.8± 0.46	2.9± 0.23	0.5± 0.16	0.6± 0.16	0

Table (xi) values of biochemical parameters of apparently healthy control camels (HC)

Table (xii) Values of biochemical parameters of camels suffering from sarcopticosis treated with 1%

A.NO.	Serum glucose(mg/dl)	Total protein(g/dl)	Albumin (g/dl)	Globulin (g/dl)	A:G ratio	Serum urea(mg/dl)	Creatinine(mg/dl)
1	79.0	7.60	4.20	3.40	1.23	20.2	1.6
2	80.0	8.60	4.46	4.14	1.07	18.0	2.2
3	79.0	7.36	4.02	3.34	1.20	24.8	1.4
4	88.0	8.40	4.88	4.42	1.10	26.0	2.5
5	77.0	7.70	4.64	3.06	1.51	23.0	2.9
6	84.0	7.84	4.20	3.64	1.15	17.6	1.3
7	76.0	7.57	3.98	3.59	1.11	21.2	2.0
8	79.0	7.82	3.98	3.84	1.04	16.0	1.4
9	81.0	7.64	4.66	3.98	1.17	18.6	2.1
10	87.0	8.30	4.14	3.16	1.31	22.4	2.8
Mean	81.0	7.88	4.31	3.65	1.18	20.78	2.02
Mean ±SE	81.0±1.28	7.88±0.12	4.31±0.10	3.65±0.13	1.18±0.04	2 0.78±1.03	2.02±0.18

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A.NO./DAY	Serum glucose(mg/dl)			Total protein(g/dl)			Albumin(g/dl)			Globulin(g/dl)			A:G ratio			Serum urea(mg/dl)			Creatinine(mg/dl)		
	1	10	20	1	10	20	1	10	20	1	10	20	1	10	20	1	10	20	1	10	20
1	60.0	64.0	75.0	6.70	7.32	8.52	3.20	3.30	3.65	3.50	4.02	4.87	0.9	0.82	0.75	10.5	15.4	17.9	4.6	3.4	2.3
2	58.0	70.0	76.0	6.48	7.12	7.83	2.91	3.10	3.59	3.57	4.02	4.24	0.82	0.77	0.85	12.6	14.8	18.3	4.1	3.6	2.5
3	65.0	74.0	80.0	5.87	6.72	7.64	2.98	3.31	3.66	2.89	3.41	3.98	1.03	0.97	0.92	9.6	12.6	14.9	5.3	3.8	2.3
4	50.0	56.0	68.0	6.59	7.68	7.96	3.56	3.86	4.01	3.03	3.82	3.95	1.17	1.01	1.01	11.4	15.4	17.6	4.0	3.1	2.2
5	52.0	62.0	69.0	6.81	7.69	8.10	2.72	2.94	3.20	4.09	4.75	4.90	0.66	0.61	0.65	13.4	16.8	18.3	4.4	2.9	2.0
6	66.0	73.0	79.0	6.77	7.74	8.21	2.94	2.99	3.40	3.83	4.75	4.81	0.76	0.62	0.70	10.2	13.4	16.4	3.9	3.3	2.1
7	68.0	78.0	82.0	6.62	6.86	8.18	2.24	2.42	3.66	4.38	4.44	4.52	0.51	0.54	0.80	11.2	14.7	15.8	5.0	4.1	3.5
8	51.0	59.0	67.0	6.93	7.54	7.92	3.46	3.84	4.32	3.47	3.70	3.80	0.99	1.03	1.08	15.3	17.6	18.8	4.6	3.2	2.7
9	62.0	72.0	76.0	7.72	7.89	8.34	2.90	3.02	3.40	4.82	4.87	4.94	0.85	0.62	0.68	12.3	15.4	17.5	4.0	3.2	2.6
10	49.0	61.0	72.0	7.68	7.84	8.46	2.87	3.00	3.52	4.81	4.84	4.94	0.60	0.61	0.71	11.0	14.7	16.3	4.3	3.2	2.4
Mean	58.1	66.9	74.4	6.81	7.44	8.11	2.97	3.17	3.64	3.43	4.26	4.49	0.829	0.76	0.82	11.75	15.08	17.18	3.92	3.18	2.46
Mean ±SE	58.1± 2.26	66.9± 2.34	74.4± 1.65	6.81± 0.17	7.44± 0.13	8.11± 0.08	2.97± 0.11	3.17± 0.13	3.64± 0.10	3.43± 0.21	4.26± 0.16	4.49± 0.14	0.829 ±0.09	0.76 ±0.9	0.82 ±0.0	11.75 ±0.53	15.08 ±0.45	17.18 ±0.40	3.92 ±0.14	3.18 ±0.11	2.46 ±0.13

Table (xiii) Values of biochemical parameters of camels suffering from sarcopticosis treated with 1%DoramectinTable Table

A.NO./DAY	Serum glucose(mg/dl)			Total protein(g/dl)			Albumin(g/dl)			Globulin(g/dl)			A:G ratio			Serum urea(mg/dl)			Creatinine(mg/dl)		
	1	10	20	1	10	20	1	10	20	1	10	20	1	10	20	1	10	20	1	10	20
1	59.0	66.0	78.0	6.53	7.56	8.20	3.0	3.43	3.74	3.53	4.13	4.46	0.85	0.83	0.84	11.4	15.6	17.3	4.3	3.2	2.1
2	65.0	76.0	81.0	6.10	7.74	8.23	2.98	3.46	3.83	3.12	4.28	4.40	0.95	0.80	0.87	12.3	16.3	18.7	5.1	3.3	2.3
3	63.0	77.0	82.0	5.88	6.83	7.92	2.74	3.56	3.91	3.14	3.27	4.01	0.87	1.08	0.98	10.3	13.7	16.9	3.8	3.1	1.98
4	60.0	75.0	80.0	5.54	6.92	7.88	2.27	3.10	3.54	3.27	3.82	4.34	0.69	0.81	0.82	13.30	17.4	19.3	3.4	2.92	1.6
5	64.0	78.0	83.0	6.30	7.64	8.34	3.41	3.93	4.59	2.89	3.71	3.75	1.17	1.05	1.22	14.33	17.8	20.5	4.6	3.2	1.9
6	61.0	79.0	82.0	6.10	7.53	8.10	3.33	3.87	4.10	2.77	3.66	4.00	1.20	1.06	1.03	13.41	17.2	20.6	4.1	2.8	1.4
7	58.0	78.0	81.0	6.77	7.89	8.41	3.46	3.93	4.29	3.31	3.96	4.12	1.04	0.94	1.04	11.8	14.7	17.6	3.8	2.3	1.3
8	66.0	79.0	83.0	6.30	7.67	8.13	3.14	3.87	4.23	3.16	3.80	3.90	0.99	1.02	1.08	13.6	17.3	19.7	4.3	3.4	2.2
9	64.0	79.0	84.0	5.93	7.89	8.33	2.74	3.68	4.34	3.19	4.21	4.23	0.86	0.87	0.96	14.0	18.3	21.1	5.3	3.6	2.2
10	63.0	78.0	86.0	6.43	7.93	8.23	2.81	3.86	4.41	3.62	4.07	4.23	0.78	0.95	0.95	13.3	17.8	21.4	4.2	3.3	1.86
Mean	62.3	76.5	82.0	6.18	7.56	8.17	2.98	3.66	4.09	3.20	3.89	4.14	0.94	0.93	0.97	12.77	16.61	19.31	3.88	3.02	1.92
Mean ±SE	62.3 ±0.84	76.5 ±1.24	82.0 ±0.69	6.18 ±0.11	7.56 ±0.12	8.17 ±0.05	2.98 ±0.11	3.66 ±0.08	4.09 ±0.1	3.20 ±0.08	3.89 ±0.09	4.14 ±0.07	0.94 ±0.5	0.93 ±0.03	0.97 ±0.03	12.77 ±0.40	16.61 ±0.47	19.31 ±0.51	3.88 ±0.18	3.02 ±0.11	1.92 ±0.10

Table (ivx) Values of serum zinc in various groups (µg/dl)

A.NO./Day	Healthy control (HC)	Ivermectin (G-1)			Doramectin (G-2)		
		1	10	20	1	10	20
1	108.4	75.4	80.2	83.6	77.4	81.4	88.6
2	95.6	78.6	84.4	86.70	74.0	79.4	84.6
3	99.8	84.2	88.6	93.4	84.6	90.2	95.4
4	94.0	76.2	81.4	86.4	83.6	86.6	90.4
5	93.8	86.7	90.6	94.2	85.0	90.2	93.8
6	120.8	74.6	79.2	82.3	82.6	88.6	96.2
7	118.4	78.6	87.4	90.2	76.8	84.6	90.4
8	108.0	85.4	89.2	91.4	75.0	83.4	91.2
9	106.2	83.8	86.4	89.2	80.4	86.6	92.4
10	108.2	88.6	92.4	94.4	79.0	87.4	92.0
Mean	105.32	81.21	85.98	89.18	79.84	85.84	91.5
Mean±SE	105.32±3.02	81.21±1.61	85.98± 1.43	89.18± 1.36	79.84± 1.26	85.84± 1.14	91.5± 1.06