Protozoal diseases of dromedaries

H. OUHHELLI* and A. DAKKAK*

Summary: In this review of protozoal diseases, special attention is paid to trypanosomiasis, which is by far the most important and widespread protozoal disease of dromedaries.

The principal agent of camel trypanosomiasis is Trypanosoma evansi, transmitted by blood-sucking flies. Prevalence of the disease is related directly to the population dynamics of these vectors.

The clinical picture is dominated by fever, and it develops into anaemia and a deterioration in general condition.

For laboratory diagnosis, identification of trypanosomes in a stained blood smear is a reliable method, but it may be necessary to use a technique which concentrates the trypanosomes. Other procedures include inoculation of laboratory animals, biochemical tests and serological tests.

Because of the failure to develop a vaccine and difficulties in controlling the vectors, chemotherapy remains the basis for prophylaxis. The effects of various trypanocidal drugs are analysed.

Finally, the less important diseases, coccidiosis, sarcosporidiosis, toxoplasmosis and balantidiasis, are reviewed briefly.

KEYWORDS: Balantidium - Camels - Coccidiosis - Dromedary - Protozoal infections - Reviews - Sarcocystis - Toxoplasma - Trypanocidal drugs - Trypanosoma evansi - Trypanosomiasis.

Protozoa which infect the dromedary belong to three principal zoological classes: Zoomastigophora (Trypanosoma), Sporozoa (Eimeria, Isospora, Sarcocystis and Toxoplasma) and Ciliophora (Balantidium).

The first group contains one of the principal pathogens of dromedaries, in comparison with which the other parasites are of secondary importance.

TRYPANOSOMIASIS

Among the protozoal infections of dromedaries, trypanosomiasis is by far the most important from the medical and economic aspects, and it is the most widespread disease. In countries where it occurs, the morbidity rate may reach 30% and the mortality rate 3% (68). Before the discovery of specific treatments the mortality rate sometimes reached 90% (44). The disease occurs mainly in Egypt, Chad, Mauritania,
Sudan, Somalia, Saudi Arabia, Iran and India. Its occurrence is moderate and geographically limited in Morocco, Algeria, Niger, Nigeria, Ethiopia, Kenya, Jordan, Iraq, Pakistan and the USSR. In these countries there are a number of vernacular names for the disease, such as dbab, zoubab, djaffa, gufar, alleh, gudho, doukane, aino, m’bori, labourit, n’diadm, tahaga and surra.

AETIOLOGY

The dominant trypanosome in dromedaries is *Trypanosoma evansi* (53). Other less frequent species are *T. vivax*, *T. congolense* and *T. brucei* in Somalia, Kenya and Sudan wherever the tsetse fly vectors occur (3, 17, 58; see also Wilson *et al.* 1983).

The principal causal agent, *T. evansi* (= *T. brucei evansi*) is a flagellate measuring 15-35µ long and 1.5-2.5µ wide. It is a member of the Sarcomastigophora phylum of protozoa, class Zoomastigophorea, order Kinetoplastida, family Trypanosomatidae.

Numerous species of domestic animals are susceptible to infection with *T. evansi*, including equines and carnivores as well as dromedaries; ruminants and pigs often develop a milder form. Khasanov and Ivanetskaya (42) reproduced a mild form of infection by inoculating a camel strain of *T. evansi* into sheep and goats. They suggested that the parasites could be transmitted between these species, and that small ruminants could act as reservoir hosts for dromedaries kept in their company.

Transmission of *T. evansi* takes place by mechanical vectors, blood-sucking flies of the species *Tabanus taeniola, sufís, bigotuttus, par, mordax* and *leucostamus*; also *Haematopota coronata, H. tenuis, Pangonia magnettii, Atylotus agrestis, A. diurnis, A. fuscipes, Ancala fasciata, A. africans, A. latipes* and *Philoliche magretti* (49, 68, 53). Other blood-sucking diptera like *Stomoxys* and *Haematobia* seem to play only a minor role in the transmission of *T. evansi* (53, 73). The parasite can survive for 24 hours in the digestive tract of tabanid flies, although its longevity is only 5 hours in the house fly (4).

The prevalence of trypanosomes in dromedaries is correlated directly with the population dynamics of adult vector flies. In Sudan (80, 81) and Chad (28) the peak activity of these flies coincides with the end of the rainy season. Herd management plays a part in susceptibility to trypanosomes, for in the Sudan, sedentary camels used for transport and work are affected most often (82). Movement of camels during transhumance removes them from the biotopes of the vectors.

THE DISEASE

Clinical signs

Trypanosomiasis due to *T. evansi* is accompanied by variable symptoms, not pathognomic. The clinical picture is dominated by fever associated with appearance of the parasites in the blood, progressive anaemia and poor general condition. Oedema develops in pendulous parts of the body, while urticarial plaques, sometimes bearing petechiae, appear on mucous membranes. In Ethiopia the disease is recognised by a change in the odour of urine, and tail hairs become more difficult to pull out (65). Corneal opacity and diarrhoea may be observed (74b). Fatal cases die in a state of advanced anaemia and extreme emaciation (68).
Economic losses are due to falls in the production of milk and meat (66) and to abortion (83).

Paraclinical signs

Among the plasma constituents which vary during camel trypanosomiasis is the glucose concentration, which is inversely proportional to the degree of parasitaemia (38). Total plasma proteins increase as a result of an increase in gamma-globulin, particularly IgG (Boid et al. 1980; Geol & Suiph 1969). The activity of serum enzymes, particularly SGPT (ALT) and SGOT (AST) increases (Boid et al. 1980b). By contrast, the content of Ca, K, Na and Cl ions falls (64). Cytologically the anaemia is macrocytic (36) and of haemolytic origin (64).

LABORATORY DIAGNOSIS

Microscopic observation of trypanosomes in blood smears stained by Giemsa’s method is the most reliable way of confirming infection. This direct diagnostic method is reliable in acute trypanosomiasis, but it is positive in only 5% of animals with chronic infection (9). At most, however, 60% of animals are positive (4). Thus it is necessary to use techniques which concentrate the parasites, as follows:

– Centrifugation of blood in the capillary tube of a microhaematocrit separates the blood into erythrocytes, leukocytes and plasma, trypanosomes occupying the layer between the erythrocytes and the plasma (77) in a proportion of about 85% (78). The capillary tube is broken at this level in order to prepare a smear on a microscope slide.

– Chromatography as used for preparing antigens is the basis of another technique for concentrating trypanosomes (52). Erythrocytes and trypanosomes are separated by passing the blood through a column of diethylaminoethyl cellulose at pH 8.0. The parasites pass through the column while the erythrocytes are retained; they can be recovered by centrifugation.

Inoculation of laboratory animals (rat, mouse, guinea pig, rabbit, dog) is one of the most sensitive diagnostic techniques (24). It provides a 15% improvement over the results of examination of a blood smear (68). Its drawback is the length of the prepatent period, which may be as much as 15 days. In addition it is impracticable for routine diagnosis on account of cost.

There are tests for the increase in serum globulin concentration in infected animals, and although this is non-specific it can be useful as an adjunct to other tests. Those used in camels are the mercuric chloride test (6, 7), the formol gel test (60, 46) and the thymol turbidity reaction (1).

More specific serological tests are those which detect specific antibodies, namely the complement fixation test (69), passive haemagglutination (37, 39), indirect immunofluorescence and ELISA (51). The last two are positive during a developing infection (51). They offer a 60% improvement in diagnosis compared with direct microscopic examination of a blood smear (Boid et al. 1979), but they are incapable of distinguishing an animal developing infection from an animal which has recovered following treatment (50). The micro-ELISA test described by Boid et al. (1985) using commercially available antigens is capable of detecting a recent infection (Rae & Luckins, cited in 10).
PROPHYLAXIS

The failure to develop vaccination against trypanosomiasis is due to antigenic variation which trypanosomes undergo during an infection. Within the subgenus *Trypanozoon*, to which *T. evansi* belongs, infection with a given clone of *T. brucei* (25, 26), *T. gambiense* (27), *T. equiperdum* (11) and also *T. evansi* (33, 40) is followed by the expression of many types of antigenic variants. In horses infected with *T. evansi*, the dominant antigenic types appear during the first weeks of infection. They subsequently produce a major antigenic change coinciding with the final stage and death of the infected animal (14).

Control of the tabanid vectors of *T. evansi* encounters the difficulties common to all attempts to control vectors, and for this reason the control of camel trypanosomiasis relies entirely on drug therapy (Table I).

**TABLE I**

*Chemotherapy of trypanosomiasis in dromedaries*

<table>
<thead>
<tr>
<th>Group</th>
<th>Non-proprietary name</th>
<th>Proprietary name</th>
<th>Mode of administration</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>arsenicals</td>
<td>melarsoprol</td>
<td>Arsobal</td>
<td>3 mg/kg</td>
<td>71</td>
</tr>
<tr>
<td>naphthalene derivatives</td>
<td>suramin</td>
<td>Naganol</td>
<td>10-12 mg/kg</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Antrypol</td>
<td>i.v.</td>
<td></td>
</tr>
<tr>
<td>quinapyramine compounds</td>
<td>quinapyramine sulphate</td>
<td>Antrycide</td>
<td>4.5 mg/kg</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Noroquin</td>
<td>s.c.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trypacide</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Quintrycide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>aromatic diamidines</td>
<td>diminazene aceturate</td>
<td>Berenil</td>
<td>2.8 mg/kg</td>
<td>62</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>i.m. or s.c.</td>
<td></td>
</tr>
<tr>
<td>isometamidium derivatives</td>
<td>isometamidium hydrochloride</td>
<td>Trypamidium</td>
<td>1 mg/kg</td>
<td>59</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>i.m.</td>
<td></td>
</tr>
</tbody>
</table>

For the past 60 years suramin has been the trypanocidal drug used most widely and most often. Its injudicious administration, including underdosage, has given rise to resistance in certain strains of *T. evansi*. The first therapeutic failure was reported by Leach in 1961 (48). Quinapyramine sulphate was introduced as an alternative for suramin-resistant trypanosomes, and it had a chemotherapeutic action lasting for at least 5 months in ponies (20, 21). However, this drug has been withdrawn from the market, probably because of its high price.

While Berenil (diminazene aceturate) has been used extensively in cattle, which tolerate it well (31), in dromedaries it has proved to be toxic, sometimes seriously (44, 18, 32).
Isometamidium injected intramuscularly gives two months of protection against reinfection, but it can lead to abscess and necrosis at the injection site. This disadvantage can be overcome by intravenous injection, but by this route there is an increased risk of toxicity (5, 70).

Most trypanocides are inactive against trypanosomes located outside the bloodstream (72), particularly when within the central nervous system.

**Coccidiosis**

The pathogenicity of coccidia was reported by Doherty in 1910 (see ref. 68), who incriminated them in severe enteritis of dromedaries in Kenya. Henry and Masson identified coccidiosis in a dromedary kept in the Jardin des Plantes in Paris, probably infected by another one brought in from Morocco; the animal died after rapidly becoming emaciated. Chineme (12) reported a clinical case of coccidiosis in Nigeria, due to *Eimeria cameli*. Stepanova (75) found that young camels, being more susceptible than adults, could develop diarrhoea or inappetence and emaciation. Cystic structures containing immature oocysts were identified within lesions.

The number of coccidial species found in camels, whether associated with disease or not, vary according to country. *Eimeria cameli* has large oocysts (80-100µ x 62-94µ), equipped with a large micropyle (13, 34); it has been found to make up 40% of the coccidial population in Iraq (57), 11.8% in India (22) and 14% in Saudi Arabia (41). *Eimeria dromedarii* is also encountered frequently, often in association with the last-named species, accounting for 50.6% of the camel coccidia in Iraq (57). Other *Eimeria* species harboured by dromedaries and bactrian camels are *E. mölleri* (Yasin & Abdessalam 1958), *E. bactriani* (10), *E. pellerdyi* (Prosad 1960), and *E. rajasthani* (22). Two species of *Isospora* which occur in camels are *I. cameli* (35) and *I. orlavi* (Tsigankov 1955).

**Sarcosporidiosis**

The pathogenic role of *Sarcocystis* in dromedaries is uncertain, although El Etreby (15) attributed myocardial lesions to this parasite (15). Mason (55) found sarcocysts in almost half of emaciated camels killed at Cairo abattoirs. The parasite is widespread, occurring in 60% of those slaughtered in Morocco (46), 81% in Egypt (15), 52% in the USSR (47), 4.5% in Sudan (23), 25% in Jordan (74) and 52% in Iran (61).

The causal agent, *Sarcocystis cameli*, has the dog as its definitive host (30, 47). It has been shown that cats are refractory to this species (47).

**Toxoplasmosis**

This infection has been detected in dromedaries by means of serological tests — the Sabin-Feldman dye test (67, 56), complement fixation (56), indirect immunofluorescence and indirect haemagglutination (54) — the prevalence ranging from 3 to 67%. According to Galuzo (1965; see ref. 68), inoculation of a virulent strain of *Toxoplasma* into 3 dromedaries resulted in severe, fatal illness with inappetence,
lacrimation and polypnea. Post-mortem examination revealed hypertrophy and to some extent haemorrhages in lymphoid organs and a peritoneal exudate.

BALANTIDIASIS

The dromedary may be an apparently healthy carrier of Balantidium coli in India (22). Cases of clinical balantidiasis have been reported from Khartoum in a 7-year-old female with diarrhoea, the faeces containing 300 cysts per gram (2), and in a 3-month-old zoo animal in Malaysia with progressive emaciation (76). Balantidiasis has been treated successfully with p-glycolyl-aminophenylarsonic acid combined with chloroquine, carbazone and kaolin (2, 76).

REFERENCES

(See p. 413)