Trypanosomiasis in the camel
(Camelus dromedarius)

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Summary: Trypanosomiasis in dromedaries is caused by Trypanosoma evansi, which is transmitted by biting flies. Clinical and laboratory diagnosis is described, together with chemotherapy and chemoprophylaxis.

KEYWORDS: Antiprotozoal agents - Camels - Chemoprophylaxis - Diagnosis - Dromedary - Drug therapy - Surra - Trypanosomiasis.

Most people generally associate trypanosomiasis with tsetse flies (Glossina spp.) and tropical Africa. Camel trypanosomiasis proves an exception. Most camels live outside of Africa’s tsetse belt, and their trypanosomiasis is caused by Trypanosoma (Trypanozoon) brucei evansi (hereafter referred to as Trypanosoma evansi), which is transmitted mechanically and is not dependent on the presence of tsetse flies. This trypanosome species causes the most widespread and the most important disease in camels.

Camels are also affected to a lesser extent by tsetse-transmitted trypanosome species, T. (T.) brucei brucei (10, 22, 23, 36, 37) and T. (Nannomanas) congolense (4, 10, 23). The latter trypanosome species causes acute disease with very high mortality. Tsetse flies might have prevented the movement of camels into Central Africa and thus impeded an Arab advance, considerably influencing the history of the African continent. Reports on the pathogenicity of T. (Duttonella) vivax (4, 10, 31) and T. (N.) simiae in camels are contradictory.

The term camel in this paper refers to the dromedary (Camelus dromedarius).

TRYPANOSOMA EVANSI

Historical observations, distribution and origin

This trypanosoma was discovered in India more than a hundred years ago by Evans (1880), who detected it in horses, mules and camels with a disease locally called “surra”. Subsequently, numerous reports of trypanosomiasis in horses and camels were recorded in North Africa, the Americas and Eurasia. Many different scientific names for the parasite were used, until it was found that all these non-tsetse transmitted
organisms of the subgenus *Trypanozoon* (except *equiperdum*) were *evansi* (11). *Trypanosoma evansi* may have originated in Africa, evolving from *brucei* (12) when camels entered the tsetse belt south of the Sahara, and the disease spread through camel herds as a result of transmission by biting flies. Continuous mechanical transmission by blood-sucking flies in the absence of *Glossina* caused the loss of cyclic transmissibility and gave rise to a predominance of slender parasite forms.

The ability to be transmitted by blood-sucking insects other than *Glossina*, has enabled *evansi* to extend its range into African areas north of the Sahara desert, into Asia Minor, Pakistan, India, the USSR, China, Sumatra, Java, the Philippines, Mauritius, Madagascar, and South and Central America. It was introduced by camels into Australia, North America and South-West Africa. In these three localities, however, it was eradicated as a result of major veterinary control measures.

**Transmission and hosts**

*Trypanosoma evansi* is transmitted mechanically by haematophagous biting flies. No developmental stage in a vector has been demonstrated which differentiates the parasite from *brucei*.

Tabanids (horseflies) play the major role in transmission, while *Stomoxys* spp. and *Lyperosia* spp. may also transmit it. An interrupted feed upon an infected host leaves the fly hungry. Whenever it moves to another host, it can establish a new infection through its trypanosome-contaminated mouthparts. Trypanosomes remain infective on the proboscis for a short period only.

The parasite replicates in camels, horses, donkeys, dogs, cattle, water buffaloes and even elephants. Equines and dogs are very susceptible and usually die after an acute course of the disease. Dogs may also become infected by eating meat from a trypanosome-infected carcass (23). Cattle, sheep, goats and antelopes often carry the parasite subclinically, acting as asymptomatic reservoirs.

**Parasite morphology and biology**

*Trypanosoma evansi* is morphologically identical with, and indistinguishable from, slender forms of other members of the subgenus *Trypanozoon*. Akinetoplastic populations are relatively common, particularly after drug exposure (18, 32). It can be distinguished from *brucei* by isoenzyme electrophoresis (8). *T. evansi* is not restricted to the blood stream. Like other members of the subgenus *Trypanozoon*, it enters tissue compartments or other body fluids; it may cross the blood-brain barrier (23) or enter the joint fluids (Röttcher and Schillinger, unpublished results), thus being less accessible to chemotherapy. This situation is comparable to chronic *brucei* infections in mice (16) or to the late stage of human sleeping sickness.

**Symptoms and the course of the disease**

Surra can attack camels at any age, even foetuses. There is a particularly high incidence of infection in juvenile camels shortly after weaning. Numerous environmental and host factors influence the course of the disease, such as other infections, nutritional status, age, pregnancy, previous exposure or immunosuppression by other diseases, and stress.

In a typical case, the dromedary loses weight, develops a drooping hump, is unable to walk long distances, and may or may not develop oedema of the feet, brisket,
underbelly and eyelids; the coat becomes rough. In the initial attack of fever there may be lacrimation, shivering, reduced appetite and mild diarrhoea. The animal always shows progressive anaemia and fluctuating body temperature with initial peaks of fever up to 41°C. Later, the appetite is relatively unimpaired and the temperature may become normal or slightly elevated.

The mucous membranes are pale and the packed cell volume (PCV) drops to below 25% (v/v), sometimes as low as 10% (v/v). The herders may notice a characteristic odour of the camel's urine and identify infected animals by this sign alone (23). The odour of the urine may be due to ketone bodies, which were found to be elevated in trypanosome-infected camels (Schillinger, unpublished results). Abortion in all stages of pregnancy is common (20, 23). If the foetus happens to be full term it may be born alive but weak with parasitaemia (34). Death of the newborn calf ensues within two weeks. Lactating females produce less milk, and cases of blindness and central nervous lesions have been reported to be sequelae of trypanosomiasis.

The herd eventually reaches an endemic disease situation. Some animals may carry trypanosomes for years whereas others never do. Within such a group there are all forms and stages of surra from new infections to subclinical and chronic conditions. The course of the disease varies widely. In Kenya, a small percentage of animals die within 2 to 5 months of contracting the disease. Some live for up to four years with subclinical infections and some eventually self-cure.

The overall productivity of a camel herd regarding calves, milk and weight gains is greatly impaired. A lethal outcome is relatively rare, but mortality may reach 20%.

**Diagnosis**

Trypanosomiasis is diagnosed by demonstrating the parasite. However, dromedaries are usually far away from laboratory facilities. A tentative diagnosis can be reached without microscopy, by taking into account the owner's observations and clinical examination of camels in the field.

The herder may report weight loss, weakness, blindness, abortions or changes in the odour of the urine. For the veterinarian, the leading sign is anaemia, the mucous membranes being pale or white. Tachycardia results. There may or may not be pyrexia, lacrimation, cachexia, enlarged lymph nodes, reduced appetite and oedemas, the latter appearing first on the underbelly.

Post-mortem examination reveals no absolutely typical signs, but some degree of anaemia is often visible. Skeleton and heart muscles are pale, and there are signs of dehydration, pericardial effusion, enlarged lymph nodes and splenomegaly.

The direct methods of trypanosome detection, which usually confirm the presence of the parasite, utilise a wet blood film, a stained thick drop of blood, and a thin blood smear. Concentration techniques can also be employed, such as the haematocrit centrifuge technique (HCT) of Woo (38), the buffy coat technique under darkground illumination of Murray et al. (DGI) (28) and the miniature anion-exchange centrifugation technique (mAECT) of Lumsden et al. (25). Centrifugation of blood, to determine the PCV and to use the buffy coat within a micro-haematocrit capillary tube for concentration techniques (HCT or DGI), is essential to assess the degree of anaemia and, at the same time, to establish a confirmed diagnosis. A battery-operated minicentrifuge has proved to be extremely valuable, because both these tests can be performed in the field without resorting to a laboratory (17).
Inoculation of camel blood into laboratory rodents is valuable for detecting sub-patent *evansi* infections in camels (9). This has been confirmed by Pegram and Scott (29), who considered the inoculation of camel blood into laboratory rodents to be the best direct diagnostic method.

For mass screening of dromedary herds there are numerous indirect tests. The formol-gel test (19) and the mercuric chloride test (3) detect only high serum globulin levels, which are a common feature in camel trypanosomiasis, but both are non-specific. Specific serological tests have been widely applied: the capillary agglutination test of Jatkar *et al.* (15), the passive haemagglutination test of Jatkar and Singh (14), the immunofluorescent antibody test and the enzyme-linked immunosorbent assay (ELISA) of Luckins *et al.* (24). Recently, Zweygarth *et al.* (41) described a simplified ELISA for camel trypanosomiasis using a commercially available protein A-peroxidase conjugate. The serodiagnosis of camel trypanosomiasis with these tests requires a somewhat sophisticated laboratory. Recently, however, a card agglutination test set has been introduced for the diagnosis of Gambian sleeping sickness (“Testryp CATT”, Smith & Kline). This card test has been successfully adapted for the serodiagnosis of *evansi* infection in camels (40).

**Treatment**

Two drugs are recommended for the treatment of *evansi* infections in dromedaries: suramin and quinapyramine, as sulphate or prosalt (Table I). Most of the drugs for cattle trypanosomiasis are either not curative (homidium bromide = Ethidium; pyrithidium bromide = Prothidium) (35) or are too toxic for camels (diminazene aceturate = Berenil) (7, 13, 21).

**TABLE I**

**Recommended drugs for the treatment of T. (T.) b. evansi infections in camels**

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Commercial name and supplier</th>
<th>Recommended dosage</th>
<th>Type of activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suramin</td>
<td>Naganol Bayer</td>
<td>12 mg/kg BW i.v.</td>
<td>curative and prophylactic</td>
</tr>
<tr>
<td>Quinapyramine</td>
<td>Antrypol ICI</td>
<td>3-5 mg/kg BW s.c.</td>
<td>curative</td>
</tr>
<tr>
<td>– methyl-sulphate</td>
<td>Antrycide Sulphate Gharda Chemicals</td>
<td>5-8.3 mg/kg BW s.c.</td>
<td>curative and prophylactic</td>
</tr>
<tr>
<td>– chloride / methylsulphate</td>
<td>Antrycide Pro-Salt Norbrook Laboratories</td>
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</table>
Suramin has been in use for almost 60 years. The recommended dosage is 12 mg/kg or about 5 g per adult camel, given by slow intravenous injection. Paravenous application can result in phlebitis. The drug is excreted slowly, which results in some prophylactic drug cover for 6-12 weeks depending on the dosage and the infection challenge. Suramin is well tolerated at up to three times the recommended dose. There are now numerous suramin-resistant strains of *T. evansi*.

Most of the resistant strains were sensitive to quinapyramine when this drug was introduced. Quinapyramine is simpler to dissolve than suramin and its subcutaneous application is easier. Quinapyramine methylsulphate is used as a curative drug, whereas a mixture of two salts, quinapyramine methylsulphate and quinapyramine chloride at a ratio of 3:2, is applied for prophylactic purposes (Pro-Salt RF). Prophylactic cover lasts for about 4 to 6 months. Severe overdose causes salivation, muscle tremors, stiffness and collapse or death following “curare-like” symptoms whereas a moderate overdose has a mainly nephrotoxic effect (6). Numerous resistant strains have also developed against quinapyramine, and many *evansi* strains with dual resistance to suramin and quinapyramine exist nowadays (26, 33).

Another drug used for the treatment of trypanosomiasis in cattle, isometamidium (Samorin, May & Baker; Trypamidium, SPECIA) has only a moderate effect against *T. evansi* and has been used in camels as an emergency measure where dual resistance against suramin and quinapyramine existed. When given intramuscularly, isometamidium produces severe local reactions. In camels, 0.5 mg/kg of isometamidium given intravenously as a 2% solution is well tolerated (1, 2). This route of application is curative in acute cases but fails when parasites have already entered extravascular sites.

*Trypanosoma evansi* may enter body compartments other than the vascular system. Available trypanocides, with the exception of the arsenicals, do not cross the blood-brain barrier in adequate concentration. Melarsoprol (Arsobal, SPECIA) has been tested using mice infected with *evansi* strains possessing single or dual resistance. All were fully sensitive to the drug, which is able not only to overcome resistance but also offers a cure in the later stages when trypanosomes have entered the central nervous system, where they are inaccessible to other drugs. Melarsoprol is curative, but has considerable disadvantages because it causes severe local reactions, unless given strictly intravenously. It has a narrow therapeutic index and treatment depends on accurate body weight and dosage determination. A dosage of 3.6 mg/kg is tolerated but should not be exceeded (Schillinger and Röttcher, 1986).

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**La trypanosomose du dromadaire.**

*Résumé* : L’agent de la trypanosomose du dromadaire est *Trypanosoma evansi*, dont la transmission est assurée par des mouches hématophages. Les auteurs présentent successivement le diagnostic clinique, le diagnostic expérimental, la chimiothérapie et la chimio prophylaxie de la maladie.

*MOTS-CLÉS* : Antiprotozoaires - Camélidés - Chimio prophylaxie - Diagnostic - Dromadaire - Surra - Thérapeutique médicamenteuse - Trypanosomose.
Resumen: El agente de la tripanosomiasis del dromedario es *Trypanosoma evansi*, transmitido por las moscas hematofagas. Los autores presentan sucesivamente el diagnóstico clínico, el diagnóstico experimental, la quimioterapia y la quimioprofilaxis de la enfermedad.


REFERENCES


