**Summary:**

Epidemiological and clinical studies on *Trypanosoma avium* are lacking in the Middle East. The aims of this study were to determine the *T. avium* incidence in falcons from Kuwait, report clinical signs and find an effective therapy. Blood smears from 921 diseased and 56 healthy falcons were examined between May 2003 and April 2004. 12 birds (1.3 %) were found infected by *T. avium* and ten of these were treated with melarsomine (Cymelarsan®) at a dosage of 0.25 mg/kg intramuscularly for four days. All affected birds presented clinical signs, including incapacity of flying high, poor appetite, lethargy, losing weight, weakness, dyspnoea and death. Signs disappeared within 1-7 days after administration of melarsomine. Trypomastigotes were not detected in blood smears made 1-7 days after the end of therapy. This study suggests that *T. avium* induces disease in falcons and that melarsomine can be an effective therapy eliminating both clinical signs and circulating trypomastigotes.

**MOTS CLÉS :** *Trypanosoma avium*, faucon, oiseau de proie, symptomatologie, chimothérapie, mélarsomine, Koweit.

**Observations made during the initial part of this study indicate that clinical signs are present in captive falcons infected by *T. avium*. This paper reports the incidence of *T. avium* in a large population of sick falcons, the clinical signs observed, and the response to melarsomine (Cymelarsan®, Merial), a trypanocide drug that has never previously been used in birds infected by *T. avium*.

**MATERIALS AND METHODS**

From early May 2003 to the end of April 2004, examinations for the presence of *Trypanosoma avium* were made on Diff-Quick-stained blood smears from 921 diseased birds of prey and 56 healthy control falcons, belonging to three species (*Falco peregrinus*, *Falco cherrug* and *Falco rusticolus*).

Blood samples were collected from the brachial vein. All falcons were used for hunting, a category given
constant special care in the Middle East. Consequently, reports on their health status and clinical signs were precise. Information evaluated included signalment, origin, date of consultation, clinical signs, intensity of infection (+ = low, ++ = moderate, +++ = high, ++++ = very high) and melarsomine (Cymelarsan®, Merial) therapy outcomes (Table I). Melarsomine, a registered medication for the treatment of *Trypanosoma evansi* in camels (“surra”), was injected by IM route at the recommended daily dose of 0.25 mg/kg (Touratier, 1992) for four consecutive days. Clinical and haematological outcomes were checked within 1-7 days after the end of therapy.

**RESULTS**

Twelve falcons out of 921 (1.3 %) were infected (Table I). None of the 56 healthy controls was carrying *T. avium*. In total, 977 birds of prey were examined. Trypanosome infections were detected between August and January, but not between February and July. Clinical signs were present in all affected raptors, and no concurrent diseases could be detected in any of them. Sudden death occurred in one case. Signs more often reported were incapacity of flying high (7; 58.3 %), poor appetite (6; 50 %), lethargy (5; 41.7 %), loose of weight (5; 41.7 %), weakness (3; 25 %) dyspnoea (2; 16.7 %) and a cessation of molting (2; 16.7 %). Within seven days after the end of therapy, the ten treated birds presented complete clinical recovery with the disappearance of trypomastigotes (Table I).

**DISCUSSION**

Scattered past reports have pointed out that *T. avium* can cause fatal infections in birds (Baker, 1976; Mungomba *et al.*, 1989). One falcon in the study group died before starting treatment, apparently confirming those previous observations. According to the recent literature, there was no evidence to indicate that *T. avium* is pathogenic (Peirce, 2003; Votypka *et al.*, 2002).

This assumption does not seem to be based on verifiable facts but on the lack of direct research, since today only morphological (Yurchenko *et al.*, 1999) and epidemiological informations (Kirkpatrick & Lauer, 1985; Munoz *et al.*, 1999) are available. Most of previous studies were carried out on wild animals captured and released after sampling (Rintamaki *et al.*, 1999; Deviche *et al.*, 2001; Holstad *et al.*, 2003). Under such field conditions pathogenic effects are hard to assess. In domestic or semi-domesticated animals living in proximity to human beings, such as captive falcons.

<table>
<thead>
<tr>
<th>No.</th>
<th>Species, sex, age, weight, origin and date of visit</th>
<th>Duration of the disease</th>
<th>Clinical signs and intensity of infection (from + to ++++)</th>
<th>Therapy outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Saker, F, 4 years, 975 gr, Kuwait, 25 August 2003</td>
<td>1 month</td>
<td>Poor appetite, lethargy, stopped moulting (++++)</td>
<td>Dead before starting the treatment</td>
</tr>
<tr>
<td>2</td>
<td>Peregrine, M, 1 year, 640 gr, Pakistan, 20 August 2003</td>
<td>1 week</td>
<td>Poor appetite, lethargy, loosing weight (++++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>3</td>
<td>Saker, F, 4 years, 1114 gr, Kuwait, 29 August 2003</td>
<td>3-5 days</td>
<td>Lethargy (++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>4</td>
<td>Saker, F, 3 years, 985 gr, Kuwait, 29 September 2003</td>
<td>2 months</td>
<td>Poor appetite, stopped moulting, loosing weight (+++)--</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>5</td>
<td>Peregrine, M, 4 years, 841 gr, Kuwait, 28 October 2003</td>
<td>10 days</td>
<td>Poor appetite, dyspnoea, weakness (+++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>6</td>
<td>Saker, M, 2 years, 850 gr, Kuwait, 15 November 2003</td>
<td>1 week</td>
<td>Weakness, not flying high, blood in the stool (+++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>7</td>
<td>Peregrine, M, 1 year, 608 gr, Iraq, 26 November 2003</td>
<td>1 week</td>
<td>Lethargy, loosing weight (+++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>8</td>
<td>Saker, M, 4 years, 900 gr, Kuwait, 26 November 2003</td>
<td>10 days</td>
<td>Poor appetite, not flying high, loosing weight (+++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>9</td>
<td>Saker, F, 1 year, 1068 gr, Kuwait, 6 December 2003</td>
<td>1 month</td>
<td>Weakness, not flying high, dyspnoea (+)</td>
<td>Treatment was denied</td>
</tr>
<tr>
<td>10</td>
<td>Peregrine, M, 1 year, 926 gr, Kuwait, 10 January 2004</td>
<td>1 week</td>
<td>Not flying high, haematoma on the back (++++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>11</td>
<td>Saker, M, 1 year, 790 gr, Iraq, 21 January 2004</td>
<td>1 month</td>
<td>Poor appetite, lethargy, not flying high (++++)</td>
<td>Clinical and haematological recovery</td>
</tr>
<tr>
<td>12</td>
<td>Saker, F, 3 years, 1,000 gr, Kuwait, 29 January 2004</td>
<td>1 month</td>
<td>Weakness, loosing weight, not flying high (++++)</td>
<td>Clinical and haematological recovery</td>
</tr>
</tbody>
</table>

Table 1. *Trypanosoma avium* and response to therapy in 12 falcons from Kuwait.
pathogenic effects are easier to detect and to describe. This seems to be the first attempt to study clinical signs and the response to therapy of *T. avium* infection in captive birds of prey. The results obtained indicate the constant presence of pathogenic effects and 9% mortality rate (Table I). Fifty-six healthy controls proved negative for *T. avium*, leading to the exclusion of healthy carriers in the study group. The disappearance of clinical signs and of trypanosomes shortly after trypanocide therapy with melarsomine indirectly confirmed that the pathogenic signs were linked to the presence of trypanosomes.

The identification of trypomastigotes in blood smears is the diagnostic test for trypanosomosis suggested by the OIE (OIE, 2000). Flagellate bodies observed in the blood from the 12 falcons showed shapes and sizes in accordance with the morphological features of *Trypanosoma avium* (Peirce, 2003). Although blood samples were always obtained from the brachial vein, it is acknowledged that there is no significant difference between *T. avium* prevalence in blood collected from the brachial vein or deep circulation (Holstad et al., 2003). Thus, the incidence rate of 1.3% obtained in this study should not be controversial with regard to the sampling site, although blood cultures may reveal a much higher prevalence. As an example, 1.2% of 259 birds of prey from southern New Jersey showed circulating *Trypanosoma* spp., while blood cultures from 142 of these raptors revealed a prevalence of 41.5% (Kirkpatrick & Lauer, 1985). It has been noted that the examination of stained blood smears will only show trypanosomes when present in the host at high levels (Peirce, 2003). This might explain the observation of clinical signs in infected birds, since it seems reasonable to assume that the higher the number of parasites the more pathogenic is their action. The 1.3% incidence reported here is low if compared with studies from areas at higher latitudes (Rintamaki et al., 1999; Deviche et al., 2001; Holstad et al., 2003), taking also into account that the population examined includes 921 ‘diseased’ falcons and not one healthy falcon.

However, results from studies carried out on free-ranging falcons admitted to wildlife rescue centres, accordingly to be considered ‘diseased’, are overlapping. In fact, 190 diseased falcons from Spain were found trypanosome-free (Munoz et al., 1999), and only 1.2% of 259 diseased birds of prey from USA were found to carry trypanosomes (Kirkpatrick & Lauer, 1985). Unfortunately, clinical signs were not recorded and no therapy was suggested.

The abundance of vectors enhances parasite transmission during favourable seasons (Rintamaki et al., 1999). This observation is in agreement with the results of this one-year study since all cases were recorded between August and January and none between February and July (Table I), apparently indicating a seasonal occurrence.

**References**


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