تكرير علاج الجمال المصرية المصابه طبيعي بالتربانوسما
بمكرك الكهدرسن وتأثيره على المقايس البيوكيميائية

بمهجة ابراهيم، أحمد رجب، زكية جابر، أحمد توفيق

الهدف من إجراء هذا البحث هو دراسة فاعلية الكهدرسن وتأثيره على التربانوسما
في الجمال المصرية المصابه طبيعي بهذا الطفيل ، وكذا تأثيره على مكونات الدم
وذلك بعد التأكد من اصابة الجمال عن طريق أخذ مسحة دموية رقيقة ، وكذا سمية
وصبغها ببصعة الجسما باستخدام اختبار كلوريد الرقيق.

وقد تم علاج 21 جمل مصابة وقد ظهرت النتائج مدى فاعلية هذا المركب في
خلال ثلاثة شهور متتالية أعقاب العلاج.

خلال هذه الدراسة أثبت أن الحيوانات المصابه بطفيل التربانوسما كانت ضعيفة
البروتين بالسبرم وكذا جلوكوز الدم والمقايس البيوكيميائية أقل من المجموعة الضابطة.
وبعد العلاج بالمكرك المدروس ارتفعت تلك المقايس في فترات زمنية تعتمد على
الحالة البحائية للعائلا.

كلا التربين امينين. اظهرت زيادة جلوكوز في الحيوانات المصابه بالطفيل و بعد
ثلاث أشهر من العلاج.
the blood. The second level disappear after treatment, the first one was 85.71, 35.71, 10.71 and zero % before the treatment, one month, two months and three months after treatment respectively.

After treatment of naturally infected camels with Quinrycide there were a significant increase in total serum protein with corresponding increase in both albumin and globulins (Fig. 1,2,3) with no changes in serum proteins of healthy camels received the same dose of the drug table (2).

Aminotransferases in infected camels were about 4 times higher than those of healthy ones, after treatment with Quinopyramin the figures dropped significantly (Fig. 4 a,b).

Haematological analysis showed reduction of Hb., PCV and RBCS count in infected camels (Fig. 5,6 and 7). Haematological parameters increased gradually after quinrycide injection. White cells counts in infected animals was significantly higher than those of healthy one. After treatment the count reached the level of healthy camels after one month of treatment (Fig. 9).

Blood glucose level was extremely low in infected camels (33-36 mg/100 ml) while in healthy camels was 68 mg/100 ml. After Quinopyramin treatment the blood glucose elevated to 65 mg/100 ml at the first month of treatment (Fig. 8).

**DISCUSSION**

In the present investigation the percentage of zero positive camels was much higher than those diagnosed by microscopic examination. The difference in percentage was due to the presence of Trypanosoma evansi in the peripheral circulation only during the paroxysm of fever. Our finding provides additional evidence to that obtained by ROBSON & ASHKAR (1972).

Results obtained indicate that Quinrycide as a single dose of 3 cc/100 kg body weight in the form of 10% aqueous solution W/V cured 36 infected camels within three months. The dose recorded in the present work 3 mg/kg body weight was lower than the maximum dose 5 mg/kg body weight which was recorded by ALEXANDER (1969) for that, the toxic effect not recorded after administration of quinopyramin in our study.

The decrease in the percentage of the positive results and the appearance of the degenerative forms of Trypanosoma evansi after administration of Quinrycide was due to its direct action on nucleic acid metabolism, as Quinpyramin binding to DNA, inhibits the incorporation of preformed purines into nucleic acid by virtue of its pyrimidyl group and interferes with growth by bringing about aggregation of cytoplasmic ribosomes (NEWTON, 1966). However ALEXANDER (1969) recorded that the significant property of Quinpyramin lies in its power to destroy infectivity and inhibition of growth and cell division of trypanosomes.

With regard to the serum protein, there was hypoproteinaemia in infected camels. The decrease in total serum protein have also been reported in acute camel trypanosomiasis (GOEL and SINGH, 1969). Moreover, BLOOD et al. (1979) discussed the vascular escape of serum proteins in trypanosoma infection leading to the subcutaneous oedema in chronic stage of the disease. The hypoalbuminaemia was reported in infected camels in the present investigation (table 2). These data emphasize previous observation recorded by RAZA et al. (1982), SINGH et al. (1982) and VERMA and GAUTAM (1982). The significant decrease in serum albumin may
be due to the destructive effect of Trypanosomes on hepatocytes leading to inadequate albumin and globulines production. After treatment with quinpyramin there were increase in protein, albumin and globulins. This may be due to the direct destructive power of the drug on the parasite leading to improving the metabolic situation of the host.

The serum aminotransferases were higher in naturally infected animals although ALAT was not raised as the ASAT. This could have been due to the limited distribution of ALAT which is mainly in the liver only where ASAT is found in most of body tissues (KANEKO & CORNELIAS, 1971). The observed elevation of these enzymes in infected camels could be due to the damaged tissue cells as suggested by KAGGWA, et al. (1984) in dogs experimentally infected with Trypanosoma brucei. Our results agree with those of BUCCIIL, et al. (1979) to whatever extent, in which they found more elevation in ALAT. That disagreement may be due to the stage of the disease (acute or chronic) which is not clear in their work. The decrease in both serum aminotransferases after administration of the drug were clear. Although statistically still significant even after three months of treatment, this indicated the severity of tissue destruction of infected camels.

Haematological changes observed in camels naturally infected with trypanosoma did not differ from those reported by MWAMBU (1979) in other animals. Anaemia observed in our studies may be due to the excessive destruction of red blood cells. It had been shown that, the half life of RBCs becomes reduced in Trypanosomiasis in other animals (SAYER, et al. 1979). The destructive effect of Trypanosoma on the blood cells disappear at the 3rd month after administration of the drug. The occurrence of leucocytosis in camelian trypanosomiasis was similar to what has been reported in dogs (KAGGWA, et al. 1984). That increase was not recorded one month after administration of Quintrycide, that indicated the decrease of body reaction against the destroyed trypanosoma.

Blood analysis of camels suffered from trypanosoma infection have shown hypoglycaemia, was in agreement with the results obtained by KATHIRIA and AVSATHI (1985) and JOSHUA, et al. (1985) in trypanosoma infection in other animals. KNOWLES DAS-GUPTA (1927) reported that the pathogenic trypanosomes consume blood glucose that leads to exhaustion of carbohydrate reserves of the host. Much later, FIENNES (1970) cited that, the high motile blood stream forms of pathogenic african trypanosomes consumes glucose at a rate corresponding to 50-100% of the dry weight of the organisms in one hour under vitro condition. The significant increase of blood glucose after injection of Quinopyramine may be due to the direct action of the drug on the parasite and glucose returned to the normal level.

REFERENCES


Bennet, S.C.L. (1929); The mercuric chloride test for camel trypanosomiasis. J. Comp. path. and Therap., 13, 116-121.


QUNITRYCIDE IN CAMELS


### Table (1)
Parasitic situation before and after treatment of camels with Quintricide

<table>
<thead>
<tr>
<th>Diagnostic procedures</th>
<th>before treatment</th>
<th>1st observation one month after treatment</th>
<th>2nd observation two months after treatment</th>
<th>3rd observation three months after treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood film examination</td>
<td>-ve 22.23% (8)</td>
<td>-ve 58.33% (21)</td>
<td>-ve 91.66% (33)</td>
<td>-ve 100% (36)</td>
</tr>
<tr>
<td></td>
<td>+ve 77.77% (28)</td>
<td>+ve 38.88% (14)</td>
<td>+ve 8.33% (3)</td>
<td>+ve Zero% (0)</td>
</tr>
<tr>
<td>Mercuric chloride test</td>
<td>-ve Zero (0)</td>
<td>-ve 53% (19)</td>
<td>-ve 92% (33)</td>
<td>-ve 100% (36)</td>
</tr>
<tr>
<td></td>
<td>+ve 69% (25)</td>
<td>+ve 47% (17)</td>
<td>+ve 8% (3)</td>
<td>+ve Zero% (0)</td>
</tr>
<tr>
<td></td>
<td>++ve 19% (7)</td>
<td>++ve Zero% (0)</td>
<td>++ve Zero% (0)</td>
<td>++ve Zero% (0)</td>
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<tr>
<td></td>
<td>+++ve 11% (4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The number of trypansoma/one drop of blood</td>
<td>85.71% (24)</td>
<td>35.71% (10)</td>
<td>10.71% (3)</td>
<td>Zero% (0)</td>
</tr>
<tr>
<td>from 1 to 5</td>
<td>14.19% (4)</td>
<td>Zero% (0)</td>
<td>Zero% (0)</td>
<td>Zero% (0)</td>
</tr>
</tbody>
</table>

-ve = negative        +ve = positive        D = Degenerated
## Table (2)

<table>
<thead>
<tr>
<th>Group</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<th>13</th>
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<tr>
<td>Pre-treatment</td>
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<td>Post-treatment</td>
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</tr>
</tbody>
</table>

**Effect of quinpyridine on blood and blood chemistry of *E. coli* isolated with *T. pyrogenes evans***

<table>
<thead>
<tr>
<th>Group</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<th>11</th>
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<th>13</th>
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<th>16</th>
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</thead>
<tbody>
<tr>
<td>Pre-treatment</td>
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<tr>
<td>Post-treatment</td>
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</tbody>
</table>

**Statistical analysis**

- Table 1: Clotted
- Table 2: No clotted

**Conclusion:**

- Group A had a significant increase in quinpyridine levels compared to Group B.
- Group C showed a decrease in quinpyridine levels.

**Significance Levels:**

- U/L: 1.5
- A: 0.01
- B: 0.05
- C: 0.1

**Further Analysis:**

- Serum globulin levels increased in Group A by 9/100ml.
- Serum albumin levels increased in Group B by 9/100ml.
- Total protein levels increased in Group C by 2.5/100ml.
Continued.

Table (2)

Effect of Quinrycide on blood and blood chemistry of Egyptian camels infected with Trypanosoma evansi

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-treatment</th>
<th>Post-treatment values</th>
<th>Statistical analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 observation</td>
<td>1st observation</td>
<td>2nd observation</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>RBC count x10^9/(mm)^3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>3.72±0.11</td>
<td>4.11±0.10</td>
<td>4.93±0.08</td>
</tr>
<tr>
<td>B</td>
<td>5.24±0.02</td>
<td>5.23±0.13</td>
<td>5.37±0.06</td>
</tr>
<tr>
<td>C</td>
<td>3.61±0.04</td>
<td>3.62±0.03</td>
<td>3.56±0.06</td>
</tr>
<tr>
<td>Hb conc. g/100 ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>7.50±0.19</td>
<td>9.53±0.38</td>
<td>10.33±0.44</td>
</tr>
<tr>
<td>B</td>
<td>11.33±0.14</td>
<td>11.40±0.21</td>
<td>11.44±0.26</td>
</tr>
<tr>
<td>C</td>
<td>7.91±0.13</td>
<td>7.95±0.14</td>
<td>7.95±0.11</td>
</tr>
<tr>
<td>PCV %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>18.33±0.39</td>
<td>20.03±0.63</td>
<td>21.28±0.83</td>
</tr>
<tr>
<td>B</td>
<td>26.96±0.57</td>
<td>26.76±0.47</td>
<td>26.73±0.49</td>
</tr>
<tr>
<td>C</td>
<td>17.15±0.89</td>
<td>17.17±0.36</td>
<td>17.10±0.20</td>
</tr>
<tr>
<td>Glucose mg/100 ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>33.74±1.01</td>
<td>65.16±5.61</td>
<td>65.00±5.43</td>
</tr>
<tr>
<td>B</td>
<td>68.44±1.51</td>
<td>67.59±0.91</td>
<td>67.73±0.82</td>
</tr>
<tr>
<td>C</td>
<td>36.20±0.94</td>
<td>36.40±0.71</td>
<td>35.75±0.44</td>
</tr>
<tr>
<td>WBC count x10^9/(mm)^3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>20.44±0.94</td>
<td>17.85±0.05</td>
<td>17.93±0.81</td>
</tr>
<tr>
<td>B</td>
<td>16.25±0.63</td>
<td>16.90±0.61</td>
<td>17.23±0.62</td>
</tr>
<tr>
<td>C</td>
<td>23.47±0.89</td>
<td>22.43±0.89</td>
<td>21.97±0.70</td>
</tr>
</tbody>
</table>

A = infected camels treated  
B = non infected treated  
C = infected not treated  
Values having a,b letters are significantly differ from their respective values at P/0.001 and P/0.05 respectively.
Fig. (3) Globulins in camels

Fig. (7) Albumin in camels

Fig. (1) Total serum protein (TP) in camels

Key:
- Uninfected camel (treated)
- Infected camel (treated)
- Infected camel (not treated)

Fig. (4a) ALAT

Fig. (4b) ASAT

Key:
- Uninfected camel (treated)
- Infected camel (treated)
- Infected camel (not treated)