1. Introduction

Trypanosomes are microscopic unicellular protozoan which belong to the genus *Trypanosoma* in the family Trypanosomatidae. Trypanosomes are blood parasites of vertebrates including common domestic animals and man[1]. Trypanosomosis is associated with the release of inflammatory response which causes degenerative changes in infected animals[2]. Such degenerative changes could be found on the islets of Langerhans in the pancreas and thus attribute to alterations in blood sugar levels and diabetes mellitus[3]. The condition is associated with depressed immunity and suppressed phagocytosis, and thus enhances the level of susceptibility to opportunistic infections[3-8]. In addition to severe anaemia, mortality in trypanosomosis could be related to alteration in blood sugar. Hence, the relevance of this study is to determine the effect of *Trypanosoma brucei* (*T. brucei*) on blood sugar level of infected rats.

2. Materials and methods

A total of 42 pathogen-free albino rats aged nine weeks old of both sexes weighing between 150–300 g were used in the study. The rats were bred in the laboratory animal house of Department of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria. The rats were fed and watered *ad libitum* prior to commencement of the study. Each rat was identified with picric acid stain.

The *T. brucei* parasite used in this study was a Federe strain obtained from the National Institute of Trypanosomosis and Onchocerciasis Research Vom, Plateau State, Nigeria. The
parasites were cryopreserved in liquid nitrogen from where the donor rats were initially infected. The parasites were maintained by serial passage in rats at the Department of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike.

An estimated \(2.5 \times 10^6\) trypanosome suspended in 1 mL of normal saline was used to infect each experimental rat through the intraperitoneal route using 1 mL tuberculin syringes. The quantity of parasite was estimated using the rapid matching method of Herbert and Lumsden[9].

Imidocarb dipropionate (12%) (Imizole®, Intervet/Merck Animal Health NADA 141-071, Approved by FDA Germany) was administered to the Group C at the dose of 24 mg/kg subcutaneously for 2 consecutive days.

Diminazene aceturate (Veribin® CEVA Sante Animale-La Ballasteière 33501 Libourne Cedex, France), a generic brand of trypanocide, was also administered to Group B at the dose of 3.5 mg/kg intramuscularly stat.

2.1. Ethical approval

All authors hereby declare that “Principles of Laboratory Animal Care” (NIH publication No. 85-23, revised 1985) were followed, as well as specific national laws where applicable. All experiments have been examined and approved by the appropriate ethics committee.

2.2. Experimental design

A total of 42 albino rats were randomly grouped into 3 groups of 14 members each. Group A was uninfected (control group); Group B was infected with \(T. brucei\) and treated with diminazene aceturate at the dose of 3.5 mg/kg once and Group C was infected with \(T. brucei\) and treated with imidocarb dipropionate at the dose of 24 mg/kg for 3 consecutive days. All treatment commenced on Day 5 post infection.

The blood sugar test was initially determined at Day 0 prior to the commencement of experiment. Subsequently, it was determined 2 days post infection till Day 12 of the experiment.

2.3. Determination of blood sugar

Blood samples were collected from the media canthus of the experimental rats on Days 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 for the assessment of blood sugar levels. The blood sugar levels were determined with a glucometer (Accu-check active serial No. GN: 10023338). The instrument was first charged with a chip before use according to the manufacturer’s instructions and the results of blood samples were read only when the reading on the glucometer remained steady for 3 s but not more than that.

2.4. Statistical analysis of data

Data obtained from this study were presented as mean ± SEM. Statistical significance was analyzed using One-way ANOVA and Duncan’s multiple range test with SPSS version 16 software package. The acceptance of significance level was at \(P < 0.05\).[10]

3. Results

From the experimental groups (Table 1), Group B and Group C were infected with \(T. brucei\) on Day 2 of the experiment and established parasitaemia by Day 4 post infection. By Days 5 and 6, there were significant increases (\(P < 0.05\)) in the blood sugar levels of both Group B and Group C. Treatment from Day 5 with diminazene aceturate at 3.5 mg/kg once in Group B and with imidocarb dipropionate at 2.4 mg/kg for 3 consecutive days in Group C respectively improved the blood sugar levels in the groups. And by Day 7, there was no significant difference (\(P > 0.05\)) between the infected groups (Group B and Group C) until by Day 10 of the experiment. There was no mortality recorded throughout the experimental period.

Table 1

<table>
<thead>
<tr>
<th>Experimental period (days)</th>
<th>Groups</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Group A (control)</td>
</tr>
<tr>
<td>0</td>
<td>113.30 ± 2.01a</td>
</tr>
<tr>
<td>1a</td>
<td>98.90 ± 2.00b</td>
</tr>
<tr>
<td>2</td>
<td>101.02 ± 0.01c</td>
</tr>
<tr>
<td>3</td>
<td>98.20 ± 1.01d</td>
</tr>
<tr>
<td>4</td>
<td>92.30 ± 2.01e</td>
</tr>
<tr>
<td>5</td>
<td>81.00 ± 1.10f</td>
</tr>
<tr>
<td>6</td>
<td>113.10 ± 2.01g</td>
</tr>
<tr>
<td>7</td>
<td>98.00 ± 1.10h</td>
</tr>
<tr>
<td>8</td>
<td>121.23 ± 1.11i</td>
</tr>
<tr>
<td>9</td>
<td>101.10 ± 0.12j</td>
</tr>
<tr>
<td>10</td>
<td>96.86 ± 8.06k</td>
</tr>
</tbody>
</table>

All values were expressed as mean ± SE. a: The homogeneity between the experimental groups at probability \(P < 0.05\); b: Day of \(T. brucei\) infection; c: Day of treatment with diminazene aceturate; d: Day of treatment with imidocarb dipropionate.

4. Discussion

Several factors could be responsible for hyperglycaemia recorded in Group B and Group C by the 4th and 5th days post infection. Previously, trypanosomes have been associated with alteration in blood sugar levels of infected animals and a significant decrease (\(P < 0.05\)) in blood sugar level was attributed to rapid consumption of sugar by trypanosomes.[11] Hypoglycaemia was also recorded in both \(T. brucei\) and Trypanosoma congolense infected sheep and Trypanosoma congolense infection in rabbits.[12,13] Conversely, a significant increase (\(P < 0.05\)) in blood sugar levels was associated
with some degrees of degenerative changes in the pancreas which may precipitate type 2 diabetes mellitus[14]. However, type 2 diabetes mellitus is a condition characterized by persistent hyperglycaemia which was not observed in this study[15]. Hence, the increases in blood sugar levels observed in Group B and Group C may be not due to degenerative changes in the pancreas or from classical diabetes mellitus. Though administration of certain drugs elevates blood glucose level in the body[16], it however was not drug induced due to null effect of diminazene aceturate on blood glucose level in rats[17]. In addition, imidazole derivates such as copper (II) imidazole complexes are likely to induce hypoglycaemia rather than hyperglycaemia in diabetic rats[18]. Nonetheless, transient elevations in blood glucose in this study may have resulted from conditions of some degrees of liver damage and acute stress reaction[19]. Severe liver damage may result from the sequestration of T. brucei parasites in the liver tissues during disease process[20]. Such liver damage may compromise the sugar regulatory function of the liver which normalized on treatment. Similarly, acute stress may induce transient increase in blood sugar level which could be induced by mere presence of trypanosomes in the blood[21]. It then seems that transient increase in T. brucei infected groups in this study was due to acute stress reaction or slight liver damage in the infected rats. The study therefore showed that T. brucei infection induced a transient elevation in blood sugar in the infected rats.

Conflict of interest statement

We declare that we have no conflict of interest.

References