Control of human African trypanosomiasis in the Quiçama focus, Angola

José Antonio Ruiz, ¹ Pere P. Simarro, ² & Teofilo Josenando ³

Objective To update the epidemiological status of human African trypanosomiasis (HAT), also known as sleeping sickness, in the Quiçama focus, province of Bengo, Angola, and to establish a HAT control programme.

Methods In 1997, 8796 people (the population of 31 villages) were serologically screened for *Trypanosoma brucei gambiense*, the causative agent of HAT. In 1998 and 1999, surveys were carried out in villages where HAT cases had been identified in 1997. Individuals were screened using the card agglutination trypanosomiasis test (CATT), and then examined for the presence of the parasite. CATT-positive individuals in whom the presence of the parasite could not be confirmed were further tested with the CATT using serum dilutions, and those with a positive antibody end titre of 1-in-4 or above were followed-up. Patients with \leq 10 white cells/ μ I and no trypanosomes in their cerebrospinal fluid (CSF) were classified as being in the first stage of the disease. Vector control was not considered necessary or feasible.

Findings The main transmission areas were on the Kwanza riverbanks, where 5042 inhabitants live. In 1997, the HAT prevalence was 1.97%, but this decreased to 0.55% in 1998 and to 0.33% in 1999. The relapse rate was 3% in patients treated with pentamidine and 3.5% in patients treated with melarsoprol. In patients treated with pentamidine, there was no difference in the relapse rate for patients with initial CSF white cell counts of 0–5 cells/ μ l or 6–10 cells/ μ l. The overall mortality rate was 0.6% and the rate of reactive arsenical encephalopathy among the melarsoprol-treated patients was 1.7%.

Conclusion The epidemiological status of the disease was updated and the transmission areas were defined. The control methods implemented allowed the disease prevalence to be reduced.

Keywords Trypanosomiasis, African/epidemiology/diagnosis/drug therapy; Trypanocidal agents; Treatment failure; Risk factors; Epidemiologic studies; Angola (*source: MeSH, NLM*).

Mots clés Trypanosomiase africaine/épidémiologie/diagnostic/chimiothérapie; Trypanocides; Echec thérapeutique; Facteur risque; Etude analytique (Epidémiologie); Angola (*source: MeSH, INSERM*).

Palabras clave Tripanosomiasis africana/epidemiología/diagnóstico/quimioterapia; Agentes tripanocidas; Insuficiencia del tratamiento; Factores de riesgo; Estudios epidemiológicos; Angola (*fuente: DeCS, BIREME*).

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Voir page 744 le résumé en français. En la página 744 figura un resumen en español.

Introduction

Angola is ranked second in the world in the number of new trypanosomiasis cases notified, despite its weak surveillance system for human African trypanosomiasis (HAT), also known as sleeping sickness (1, 2). However, the true epidemiological situation in the country is unknown due to the civil war, ongoing since 1975, which has limited the number of functional mobile teams for control activities. Although the Quiçama municipality had been little affected by the war, security deteriorated in July 1999 and HAT control activities stopped. This affected both follow-up and case-finding activities in some villages.

The first reported case of HAT in Angola was in the Quiçama municipality (3), and studies carried out in 1900 confirmed that HAT was an important health problem in the area of the Kwanza River (including the Quiçama municipality) (3). At that time, the disease prevalence was reported to be 12% (3). Between 1946 and 1963 some 2000 cases were

reported in the Luanda province, which included the Quiçama municipality at that time (4). Thereafter, between 1964 and 1975, more than 10 000 people were screened each year in Quiçama, but no cases were diagnosed (5).

From 1976 to 1996, no control activities were implemented in the Quiçama municipality. Consequently, at the beginning of 1997, information about HAT epidemiology was limited. The only information came from the Viana National Sleeping Sickness Centre (NSSC) in Luanda, where people from the Quiçama municipality presented themselves for diagnosis and treatment. The project in the present study focused on updating the epidemiological information on HAT, and on establishing a disease control programme, based on the updated epidemiological observations.

Quiçama municipality

The Quiçama municipality covers 13 500 km² (Fig. 1). It is situated to the south of Luanda (9°51′S; 13°44′E) in the fertile

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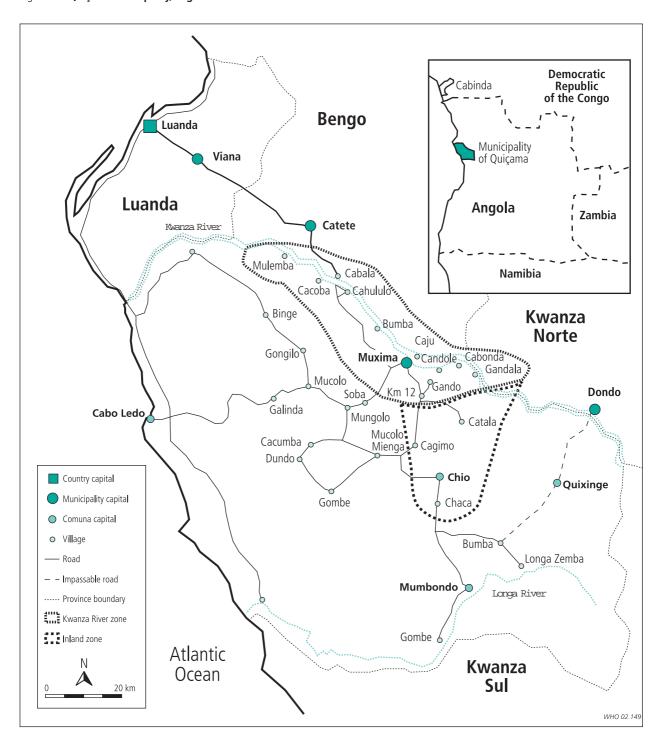
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Kwanza River valley, and has a flat landscape with no hills over 50 m. It has a tropical climate, with a dry season from May to August and a rainy season from September to April. Temperatures range from 21 °C during the dry season to 39 °C during the rainy season. Riverbank vegetation includes forests with palm, mango, and banana trees. The surrounding area is humid savannah.

The 1997 population figures, obtained from village chiefs, indicated that 16 474 people lived in the area and almost all (99%) were of the Mbundu ethnic group. The municipality is territorially divided in four "comunas": Cabo Ledo, Chio,

Quixinge, and Mumbondo. Muxima, the capital of the municipality, has a church that has been an important centre for religious pilgrimage since the middle of the 17th century. The main activities in the area are agriculture (cultivation of cassava, sweet potato, beans, and oranges; and palm oil production), fishing (in the Kwanza River) and hunting (in the inland areas). Men fish, hunt and occasionally help in some of the agricultural tasks, such as land clearing. Women traditionally care for the fields, while young children stay at home and spend most of their time collecting and carrying water when they are outside the home. Teenagers help adults in their tasks.

Fig. 1. The Quiçama municipality, Angola



Methods

Study description

From January to March 1997, a general census of the Quiçama municipality was carried out to monitor trypanosomiasis control activities. The knowledge and attitude of people and local authorities towards HAT was studied: all were extremely concerned about the disease situation in the area. A mobile team of one supervisor and three technicians was trained to perform field surveys and seroparasitological tests for HAT. Health workers from the Muxima Health Centre were also trained to treat HAT.

Seroparasitological survey *Quicama focus*

In July 1997, a seroparasitological survey was carried out in 31 of the 35 villages in the municipality (Fig. 1). Villages on the Atlantic coast were excluded, because they were considered unaffected: there were no historical accounts of the disease, no recent reports of cases, nor could the presence of the vector be demonstrated. Villages close to the Kwanza River in the comuna of Quixinge were screened by members of the mobile team in Kwanza Norte province, who were closer to the area than the Muxima team. One village in the comuna of Mumbondo was not visited due to the presence of landmines.

All villages with identified cases of HAT were considered to be endemic for the disease. Villages located close to endemic villages but without identified cases were also considered to be endemic, since the villages had a similar ecosystem and the populations carried out comparable activities to those in the endemic villages. Villages with no reported cases, but where the vector was present, were considered to be at risk for the disease. Villages in which the vector could not be found were considered to be free of the disease. Lancien traps (6) were used to detect the vector, and all trapped tsetse flies were identified. The area encompassing endemic and at-risk villages was defined as the Quiçama focus. In 1998 and 1999, additional surveys were carried out in all endemic villages identified during the 1997 survey.

Survey method

People participating in the survey were registered and screened using a card agglutination trypanosomiasis test (CATT), on fresh undiluted blood (7). Parasitological examinations were then done sequentially on all CATT-positive individuals until the parasite was detected (Fig. 2). If individuals had enlarged glands, lymph node fluid was examined first. If enlarged glands were not present, or if examination of the lymph node fluid did not demonstrate the presence of the parasite, the capillary tube centrifugation technique was used to detect the parasite in the bloodstream. If the blood sample still tested negative for the parasite after the centrifugation test, an additional blood examination was carried out, using the mini-anion exchange centrifugation technique. All cases positive for the parasite were treated.

Treatment

Prior to treatment, a lumbar puncture was performed on all cases to determine the stage of the disease. Individuals whose cerebrospinal fluid (CSF) had ≤ 10 white cells/ μ l and no trypanosomes were classified as being in the first stage of the disease and were treated with pentamidine: one complete

treatment consisted of 10 injections of 4 mg/kg body weight/ day on alternate days (8). Patients with a CSF white cell count of >10 cells/ μ l or \leq 10 cells/ μ l but trypanosomes were detected in their CSF during the cell count, or after centrifugation or double centrifugation of the CSF were considered to be in the second stage of the disease and were treated with melarsoprol. Nine injections in total were given in three courses of three injections, with an interval of 6 days between series. The melarsoprol treatment was preceded by a single dose of pentamidine (4 mg/kg body weight) on day 1, and anthelminthic and antimalarial drugs (on days 2-4). Melarsoprol treatment began on day 5 and the dose increased progressively with each injection, from 1.8 mg/kg body weight to 3.6 mg/kg body weight, to give a total dose of 26.18 mg/kg body weight (Table 1). Prednisolone was administered (1 mg/kg body weight, to a maximum of 40 mg) at the same time as melarsoprol. Administration started the day before the first melarsoprol injection and was also given during the rest periods of melarsoprol treatment (days 8-13 and 17-22). Administration of prednisolone continued for three days after the melarsoprol treatment had finished, but it was gradually reduced (0.75 mg/kg body weight on day 26, 0.50 mg/kg body weight on day 27, and 0.25 mg/kg body weight on day 28). Follow-up studies were carried out 6, 12, and 24 months after treatment.

A twofold serum serial dilution in CATT buffer was systematically performed on CATT-positive, but parasite-negative individuals. Individuals with a positive antibody end titre below 1-in-4 were considered free of infection and were not followed up. Infection was suspected in those with positive antibody end titre of 1-in-4 or 1-in-8, and strongly suspected in those with positive antibody end titre above 1-in-8. Those with suspected or highly suspected infection were followed up quarterly, using seroparasitological methods, and these results have been published (9).

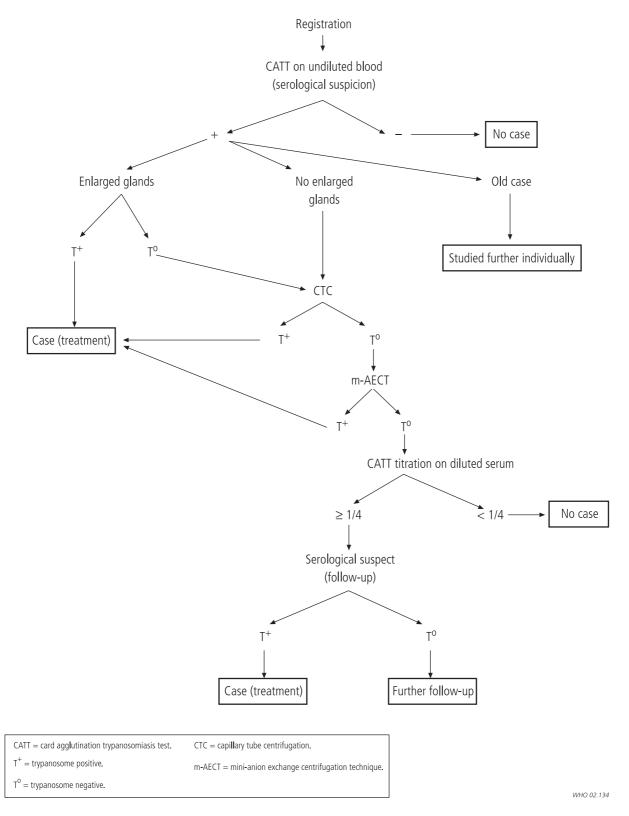
Data analysis was performed using Epi Info (10) and all screening and prevalence rates were compared by the χ^2 method.

Table 1. Treatment schedule for the second stage of human African trypanosomiasis

Day of treatment	Drug	Dose (mg/kg body weight)
1	pentamidine	4.00
5	melarsoprol	1.80
6	melarsoprol	2.20
7	melarsoprol	2.56
14	melarsoprol	2.56
15	melarsoprol	3.00
16	melarsoprol	3.26
23	melarsoprol	3.60
24	melarsoprol	3.60
25	melarsoprol	3.60

See text for details of the administration of prednisolone, and anthelminthic and antimalarial drugs.

Fig. 2. Flow chart for diagnostic decision-making



Results

Geographical distribution of HAT and epidemiological data

In 1997, we screened 8796 of 10 285 people in 31 villages in the Quiçama municipality (a screening rate of 85.5%). HAT was endemic in 18 of the villages, and 14 villages had confirmed

cases (Cahululo, Bumba, Catondo, Muxima, Pita, Caju, Km 12, Gando, Candole, Cabonda, Gandala, Catala, Cagimo, and Chio). Four villages without confirmed cases (Mulemba, Cacoba, Cabala, and Chaca) were also considered endemic because they had a similar ecosystem and the populations carried out comparable activities to those in the endemic villages. In the 18 endemic villages, 4753 of the 5042 in-

habitants were screened (94.3% of those living in endemic areas) and 94 cases of HAT were detected (prevalence of 1.97%) (Table 2).

Thirteen villages in the Quiçama municipality were considered to be at risk because the vector was present (Soba, Mungolo, Cacumba, Gombe, Mucolo-Mienga, Mucolo, Galinda, Gongilo, Binge, Bumba, Mumbondo, Gombe, and Longa Zemba). However, even though 4043 of the 5243 inhabitants were screened (screening rate of 77.1%), no parasitologically positive cases were detected.

The epidemiological status of HAT in the Quiçama municipality was derived from these results, and the focus of the HAT geographically circumscribed. Considering human activities and the ecosystem in the area, two zones of endemicity were identified (Table 2, Fig. 1). The Kwanza River zone included all villages on the riverbanks and lakeshores. The population of this zone was 3738 inhabitants, and the prevalence of HAT was 1.91% (66/3452). The second was the inland zone: it included all villages away from the riverbanks and lakeshores. It had a population of 1304 inhabitants and the prevalence of HAT was 2.15% (28/1301). There was no significant difference between HAT prevalence in the two zones (P=0.6).

Active detection rate

The mobile team detected 80.7% (126/156) of the HAT cases treated in Muxima Health Centre between July 1997 and July 1999. All other cases presented themselves for treatment.

Vector identification

During the vector assessment phase of the project, both *Glossina palpalis palpalis* and *Glossina tahaniformis* were identified (C. Laveissière, personal communication, 1997).

Disease distribution among the population

In the study population, 48.1% (2287/4753) of the people were ≥ 15 years years of age, and 51.9% (2466/4753) were ≤ 14 years old. Despite this age distribution, adults were significantly more affected by HAT than children (P < 0.001). In the endemic villages, for example, 72.2% (91/126) of the cases detected were in individuals over 14 years old. However there was no significant difference in the sex distribution of the cases: 52.3% (66/126) of them were men and 47.7% (60/126) were women (P = 0.2).

First-stage cases of HAT

Of the cases detected actively, 72% (90/125) were in the first stage of the disease (one patient did not present for treatment).

Serological status

All patients (126/126) had a positive CATT result on whole blood; 96.6% (114/118) had a serum positive antibody end titre above 1-in-8; and 3.4% (4/118) a positive antibody end titre of 1-in-8. For eight patients, no serum was obtained and therefore they were not tested.

Impact of control activities observed in the field

Where possible, all villages considered endemic in 1997 were subsequently screened in 1998 and in 1999. In July 1999, three villages could not be accessed for security reasons (Table 2). Between 1997 and 1999, the overall HAT prevalence dropped by 83.3%, from an initial rate of 1.97% in 1997, to 0.55% in

1998, and to 0.33% in 1999. During this same period, the screening rate dropped from 94.3% in 1997, to 75.4% in 1998, and 70% in 1999, due to lack of interest by the population.

Impact of control activities on the Viana National Sleeping Sickness Centre

The impact of the control activities in the Quiçama municipality could also be observed in the Viana NSSC, where people from Quiçama used to seek treatment. In 1996, one year before starting control activities in Quiçama, 21.4% (554/2584) of the CATTs and 20% (391/1958) of the lumbar punctures carried out in Viana were on individuals from Quiçama. Between 1997 and 1999, these proportions decreased progressively, dropping to 9.4% and 7.3% respectively in 1997 (after the survey) and to 0.9% and 0.7% respectively in 1999, a total decrease of 95.7% and 96.5% respectively.

Treatment and follow-up

Between July 1997 and July 1999, 156 patients were treated. Of these, 79 had a CSF white cell count of \leqslant 5 cells/µl, and were treated with pentamidine; 21 patients had a CSF white cell count of 6–10 cells/µl and were also treated with pentamidine; and 56 patients had a CSF white cell count of >10 cells/µl, or \leqslant 10 cells/µl but trypanosomes were detected in their CSF: they were subsequently treated with melarsoprol (Table 1). Of the 156 patients, 123 (78.8%) came for follow-up at least once.

In the group with CSF white cell counts of $\leqslant 5$ cells/µl and who were treated with pentamidine, 2.5% (2/79) relapsed after treatment (mean follow-up time: 10 months; range: 4–22 months), while 4.7% (1/21) of those with a CSF white cell count of 6–10 cells/µl and who were also treated with pentamidine relapsed (mean follow-up time: 11 months; range: 5–18 months). In the group treated with melarsoprol, 3.5% (2/56) relapsed (mean follow-up time: 13 months; range: 6–22 months).

Reactive arsenical encephalopathy

One of the 56 cases (1.7%) treated with melarsoprol developed a reactive arsenical encephalopathy and subsequently died.

Mortality

Only 1 patient of the 156 (0.6%) diagnosed during screening and subsequently treated died of the disease.

Discussion

The distribution of HAT extends beyond the Quiçama municipality, and affects the neighbouring municipalities on the north bank of the Kwanza River. This emphasizes the importance of organizing field activities according to epidemiological trends and the distribution of the disease, and not solely according to administrative determinants, which are often inherited from colonial times.

Most (80.7%) of the trypanosomiasis cases detected in the Quiçama focus from July 1997 to July 1999 were diagnosed by the mobile team, and the majority of the cases detected actively (72% (90/125)) were in the first stage of the disease. In this study, we used the following criteria for classifying the first stage: a CSF white cell count of ≤ 10 cells/ μ l and no trypanosomes in the CSF. If the classical criteria of a CSF white

Table 2. Number of residents in villages studies (census), screening rate, and HAT^a prevalence in endemic villages of Quiçama focus, 1997-99

Name	Zone/village	Census	No. of people screened		No. of HAT cases			HAT prevalence %			
Mulemba 106 90° kd4.90 156° kd4.90 NAd 0 NA 0 0 NA Cacoba 154 73 kd4.00 102 kd6.20 NA 0 0 NA 0 0 NA Cabala 150 274° kd6.20 127 kd6.30 NA 0 1 NA 0 0.7 NA Cabululo 692 416 kd6.30 309 kd6.00 2 1 0 0.5 0.3 0 Bumba 137 kd5.00 81 kd6.00 53.00 1 1 0 1.5 1.2 0 Catondof 262 317° kd4.60 81 kd6.30 330 1 1 0 1.5 1.2 0 Catondof 262 317° kd7.00 485° kd2.00 1 1 0 0.2 0.2 0.2 Muxima³ 60 470° kd7.00 455° kd2.00 492° kd2.00 1 1 0 1.7 0.2 0 Caiy 467 3189 kd6.30 782.0		n	Year			Year			Year		
Mulembla 106 96° (147.1) (147.1) NA ^d 0 0 NA 0 0 NA Cacoba 154 73 (47.4) (166.2) NA 0 0 NA 0 0 NA 0 0 NA Cabala 150 274° (182.6) 127 (182.6) NA 0 1 NA 0 0.7 NA Cabululo 692 416 (182.6) 309 (60.1) (446.6) (37.5) 2 1 0 0.5 0.3 0 Bumba 137 (65.8) 81 (53.1) (38.6) 1 1 0 1.5 1.2 0 Catondol 262 (317° (27.8) (38.0) (83.2) 11 5 2 3.5 1.9 0 0 Catondol 260 (78.3) (78.3) 485° (82.0) 1 1 0 0.2 0.2 0.2 0 Muxima³ 60 (62.5) (78.3) (78.0) (82.0) (82.0) 1 1 0 0.7 0.2 0 0 0			97	98	99	97	98	99	97	98	99
Cacoba 154 73 102 102 103 104 105 102 103 10	Kwanza River zone	e									
Cabala 150 274° 127 184 127 184 127 184 186 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184 185 184	Mulemba	106			NA ^d	0	0	NA	0	0	NA
Cahululo	Cacoba	154			NA	0	0	NA	0	0	NA
Bumba 137 656 811 53 11 1 0 1.5 1.2 0	Cabala	150			NA	0	1	NA	0	0.7	NA
Catondo Cato	Cahululo	692				2	1	0	0.5	0.3	0
Muxima ⁹	Bumba	137				1	1	0	1.5	1.2	0
Pital	Catondo ^f	262				11	5	2	3.5	1.9	0.9
Caju 467 319 (68.3) 337 (72.2) 285 (61.0) 20 0 6.3 0 0 Km 12 42 118 (68.3) 49 (72.2) 46 (109.5) 1 0 0 0.8 0 0 Gando 181 170 (94.0) 69 (38.1) 51 (28.1) 8 2 0 4.7 2.9 0 Candole 248 172 (89.3) 181 (73.3) 160 (64.5) 5 1 0 2.9 0.5 0 Cabonda 217 (193) (142 (92.2) 7 3 1 3.6 2.1 0.5 Gandala 386 (93.9) (65.4) (93.0) 7 3 1 3.6 2.1 0.5 Gandala 386 (97.9) (46.8) (46.8) (48.2) 3 3 5 0.8 1.7 2.7 Total 3738 (92.3) (75.6) (65.6) 66 (93.6) (93.3) (75.6) (65.6) 19 8 1.91 (0.67) 0.67 0.36 Inland zone Catala 301 (285 (94.6) (70.4) (62.1) (62.1) (75.6) (85.6) 1 3 5.2 0.5 1.6 Cagimo	Muxima ^g	60				1	1	0	0.2	0.2	0
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(109.7) (39.1) (80.5) Total 1304 1301 979 1060 28 2 3 2.15 0.20 0.28 Total 5042 4753 3806 3245 94 21 11 1.97 0.55 0.33	Chio	283				5	1	0	1.8	0.4	0
(99.8) (75.0) (81.2) Total 5042 4753 3806 3245 94 21 11 1.97 0.55 0.33	Chaca	195 ⁱ				0	0	0	0	0	0
Total 5042 4753 3806 3245 94 21 11 1.97 0.55 0.33 (94.3) (75.4) (70.0)	Total	1304			1060 (81.2)	28	2	3	2.15	0.20	0.28
	Total	5042			3245 (70.0)	94	21	11	1.97	0.55	0.33

 ^a HAT = human African trypanosomiasis.
 ^b Figures in parentheses are screening rate in percentages.
 ^c People from neighbouring villages came to the survey.
 ^d NA = not available. In 1999, these villages could not be visited due to security reasons.

^e The survey was undertaken on market day.

f A suburb of Muxima.

⁹ Capital of the municipality.

All pupils from different schools coming from neighbouring villages were screened on the same day in Muxima.

During the harvest period many people from the neighbouring villages work in Km 12.

Census was incorrect: some people refused to be registered.

cell count of ≤ 5 cells/ μ l and no trypanosomes in the CSF had been applied, the percentage of cases in first stage would have been 56.8% (71/125). By comparison, among 5699 people actively screened in Cacuso (Malange) during the 1997 by the National Programme for Trypanosomiasis Control in Angola, 70 of 224 detected cases (31%) were in the first stage of the disease, using the classical criteria (T. Josenando, personal communication, 1997). In 1990 there were 37% (213/583) first-stage cases among the 160 167 people screened, using the classical criteria (11). The higher proportion of individuals in the first stage of the disease (56.8%) in Quiçama could be explained by the fact that second-stage patients feel sick and they probably sought treatment at the relatively close Viana NSSC. Moreover, in a historical focus such as Quiçama, with a long parasite-host coexistence, the disease could be slow to evolve.

In general, we found that adults were more affected than children. We ascribe this to the cumulative risk of exposure to infection over time, and to the specific activities performed locally. We estimate that men's main activities (fishing and hunting) do not involve more risk than the agricultural and water transportation activities carried out by women. Instead, anthropophylic vectors such as *G. palpalis palpalis* are universally distributed and transmission occurs everywhere. However, the initial prevalence of the disease in the area (1.97%) led us to select active case-finding as the sole control method, dismissing vector control activities altogether.

As observed in other countries (12–14), active case-finding alone could significantly reduce disease prevalence (by 83.3% in this study). The proportion of patients from the

Quiçama municipality attending the Viana NSSC decreased, as they were treated in situ and had no further need to go to the NSSC. This decreases the work burden for the NSSC and the patients save money on transportation costs.

The 3.5% melarsoprol treatment failure rate is consistent with the 3–9% failure rate observed in many countries (15). The 4.7% relapse rate for patients with a CSF white cell count of 6-10 cells/µl and who were treated with pentamidine is lower than the 6% rate seen in patients with a CSF white cell count of 6-20 cells/µl (8). In our results, there was no statistical significant difference in relapse rates between the two groups treated with pentamidine and who had a CSF white cell count of 0-5 cells/ μ l or 6–10 cells/ μ l. Thus, a CSF white cell count of \leq 10 cells/ μ l is a more suitable criterion for initiating pentamidine treatment than that of 5 cells/µl, and could save the National Programme in both drug costs and hospital expenses. It would also significantly reduce the number of patients who would otherwise be exposed to the risk of arsenical toxicity. Further studies with a larger number patients and a longer follow-up periods should now be carried out.

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Résumé

Lutte contre la trypanosomiase humaine africaine dans le foyer de Quiçama (Angola)

Objectif Réexaminer la situation épidémiologique de la trypanosomiase humaine africaine (THA), connue également sous le nom de maladie du sommeil, dans le foyer de Quiçama, province de Bengo (Angola), et mettre en place un programme de lutte contre cette maladie.

Méthodes En 1997, un examen sérologique de recherche de *Trypanosoma brucei gambiense*, l'agent étiologique de la THA, a été pratiqué chez 8796 personnes (soit la population de 31 villages). En 1998 et 1999, des enquêtes ont été réalisées dans les villages où des cas de THA avait été identifiés en 1997. On a d'abord réalisé un test d'agglutination sur carte pour la trypanosomiase (CATT), puis une recherche du parasite. Devant un test positif en CATT et quand la présence du parasite n'a pas pu être confirmée, on a refait le test CATT sur des dilutions de sérum et les patients ayant un titre positif final en anticorps égal ou supérieur à un quart ont été suivis. Quand la cellularité du liquide céphalorachidien (LCR) était inférieure ou égale à 10/µl et en l'absence de trypanosome dans le LCR, un diagnostic de premier stade de la

maladie a été porté. La lutte antivectorielle n'a pas été considérée comme nécessaire ou faisable.

Résultats Les zones principales de transmission se situent sur les berges du fleuve Kwanza, où la population s'élève à 5042 habitants. En 1997, la prévalence de la THA était de 1,97 %, mais est tombé à 0,55 % en 1998 et 0,33 % en 1999. Le taux de rechute était de 3 % après traitement par la pentamidine et de 3,5 % après traitement par le mélarsoprol. Chez les patients traités par la pentamidine, le taux de rechute n'était pas différent quand la cellularité initiale du LCR était 0-5 cellules/μl ou 6-10 cellules/μl. Le taux de mortalité global était de 0,6 % et la fréquence des encéphalopathie réactionnelles à l'arsenic parmi les patients traités par le mélarsoprol était de 1,7 %.

Conclusion La situation épidémiologique de la maladie a été réexaminée et les secteurs de transmission ont été délimités. Les méthodes de lutte mises en œuvre ont permis de réduire la prévalence de la maladie.

Resumen

Control de la tripanosomiasis humana africana en el foco de Quiçama (Angola)

Objetivo Actualizar la información epidemiológica sobre la tripanosomiasis humana africana (THA), también conocida como enfermedad del sueño, en el foco de Quiçama, provincia de Bengo, Angola, y establecer un programa de control de la THA.

Métodos En 1997, 8796 personas (la población de 31 aldeas) fueron sometidas a cribado serológico para *Trypanosoma brucei gambiense*, el agente causante de la THA. En 1998 y 1999 se llevaron a cabo encuestas en las aldeas donde se habían

identificado casos de THA en 1997. Los individuos participantes fueron cribados mediante la prueba de aglutinación en tarjeta (CATT) para la tripanosomiasis, y examinados luego para confirmar la presencia del parásito. Los individuos CATT-positivos en los que no pudo confirmarse esa presencia fueron sometidos a análisis adicionales con la prueba CATT utilizando diluciones del suero, y aquellos con un título de anticuerpos de 1/4 o superior fueron objeto de seguimiento. Los pacientes con cifras $\leqslant 10$ leucocitos/µl y sin tripanosomas en el líquido cefalorraquídeo (LCR) fueron clasificados como afectados por la primera fase de la enfermedad. La adopción de medidas de lucha antivectorial se consideró innecesaria o inviable.

Resultados Las principales zonas de transmisión se hallaban en las riberas de Kwanza, con 5042 habitantes. En 1997, la

prevalencia de THA era del 1,97%, pero el porcentaje disminuyó al 0,55% en 1998 y al 0,33% en 1999. La tasa de recaída fue del 3% en los pacientes tratados con pentamidina, y del 3,5% en los tratados con melarsoprol. En los pacientes tratados con pentamidina no se observó ninguna diferencia en la tasa de recaídas entre los pacientes con recuentos leucocitarios iniciales en el LCR de 0-5 células/µl y los que presentaban 6-10 células/µl. La tasa de mortalidad global fue del 0,6%, y la incidencia de encefalopatía arsenical reactiva entre los pacientes tratados con melarsoprol fue del 1,7%.

Conclusión Se actualizó la información epidemiológica sobre la enfermedad y se delimitaron las zonas de transmisión. Los métodos de control aplicados permitieron reducir la prevalencia de la enfermedad.

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