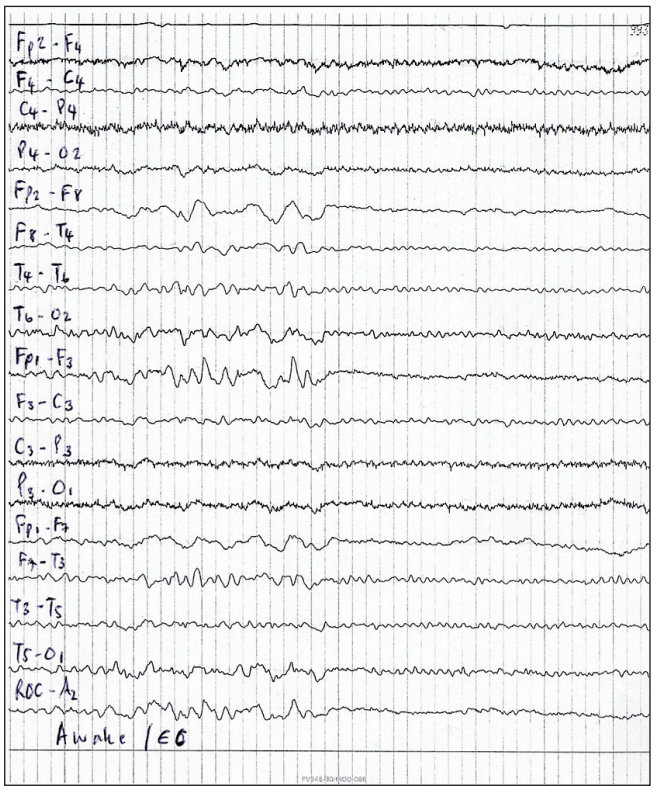

Fig. 2. EEG of case 1 showing mixed-frequency low-voltage background activity with frontally dominant bursts of high-voltage irregular delta activity.

Discussion

WAT, caused by *T. b. gambiense*, is transmitted by tsetse flies inhabiting the forests of Central and West Africa. EAT, caused by *T. b. rhodesiense*, is transmitted by tsetse flies found in the savannah and woodland areas of Central and East Africa.[3] Patients present with a two-phase illness – haemolymphatic and neurological. The course in WAT is more protracted and the outcomes usually more favourable than in EAT.

Giemsa-stained smears of peripheral blood, bone marrow, lymph node aspiration or chancre fluid are used to detect trypanosomes. Serial specimens should be examined as parasitaemia levels may vary.[4] Concentration methods increase the chances for detection (quantitative buffy coat, Becton-Dickenson, NJ).[4] Treatment decisions should be based on demonstration of the parasite since serological assays have variable sensitivity and specificity.[5]

In vivo inoculation of mice distinguishes between EAT and WAT as *T. b. rhodesiense* but not *T. b. gambiense* trypanosomes will be detected.[3]

The diagnosis of WAT in our first patient was surprising, as he had not travelled to Western endemic areas. This raises the possibility of cross-migration of tsetse fly species to other areas.

A lumbar puncture is mandatory after parasite clearance of peripheral blood to exclude stage II (neurological) disease and to individualise treatment. Suramin and pentamidine do not penetrate the central nervous system (CNS) adequately and are only used for the haemolymphatic stage.

The arsenic-based drug melarsoprol is currently the only effective treatment for second-stage EAT but causes an encephalopathic syndrome in 5 - 10% of patients, with a case fatality rate of 50%, and resistance has also been described.[3] Eflornithine should be used in WAT patients with CNS disease.

Development of treatment options is limited. Nifurtimox, a drug used to treat Chagas disease, may be effective, but has not yet been validated for use. Oral eflornithine, the diamidine prodrug DB 289, and the pre-clinical development of megazol seem promising.[5]

1. Cattand P, Jannin J, Lucas P. Sleeping sickness surveillance: an essential step towards elimination. *Trop Med Int Health* 2001; **6:** 348-361.
2. Burri C. Efficacy of new, concise schedule for melarsoprol in treatment of sleeping sickness caused by *Trypanosoma brucei gambiense:* a randomised trial. *Lancet* 2000; **355:** 1419-1425.
3. Kirchhoff LV. Agents of African trypanosomiasis (sleeping sickness). In: Mandell GL, Bennet JE, Dolin R, eds. *Principles and Practice of Infectious Diseases*. 6th ed. Philadelpia: Elsevier, 2005: 3165-3170.
4. Bailey JW, Smith DH. The use of the acridine orange QBC technique in the diagnosis of African trypanosomiasis. *Trans R Soc Trop Med Hyg* 1992; **86:** 630.
5. Fairlamb AH. Chemotherapy of human African trypanosomiasis: Current and future prospects. *Trends Parasitol* 2003; **19:** 488-494.