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RESEARCH ARTICLE

NEUROPSYCHIATRIC MANIFESTATIONS AFTER AWAKE ETHYLIC COMAS AT THE SERVICE NEUROPSYCHIATRY UNIVERSITY HOSPITAL BEFELATANANA, ANTANANARIVO

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ABSTRACT

This is a retrospective study from January 2017 to December 2017 at the Department of Neuropsychiatry, University Hospital Joseph Raseta Befelatanana Antananarivo, Madagascar. This study aims to describe the clinical aspects after waking up of ethyl coma. We included all woken-up comas after resuscitation with neuropsychiatric symptoms. But excluded, unknown patients, incomplete files or released against medical advice. We collected 123 patients, one hundred and ten (89%) are male versus thirteen (11%) female. The middle age is 37 years old. Farmers and low education represent the majority of our patients. Seventy-six patients (61.79%) were married, thirty-three (26.83%) were single, and fourteen (11.38%) were divorced and widowed. The main neurological clinical manifestations were withdrawal syndrome (n = 90), seizure and status epilepticus (n = 60), Gayet Wernicke's encephalopathy (n = 22), korsakoff syndrome (n = 12), pellagous encephalopathy (n = 7), cerebellar syndrome (n = 30), alcoholic polyneuropathy (n = 40), optic neuropathy (n = 3), alcoholic dementia (n = 2). The Psychiatric manifestations were mainly depression (n = 16) and alcoholic psychosis (n = 7). At the end of this study, we can conclude that wakeful comatose patients often have serious neuropsychiatric complications that are still a problem of management.

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INTRODUCTION

Since Antiquity, humanity has never been separated from alcohol. Until today, drinking alcoholic beverages has been a part of their daily lives. However, alcoholism is a social disease that is both a public health problem and an economic problem. Excessive alcohol consumption generally leads to alcohol coma when the blood alcohol level exceeds 3 to 5 grams per litre (Raveloson *et al*., 2009). After awakening, the patient develops neuropsychiatric disorders or signs of varying degrees. This is how we chose this study in order to describe the neuropsychiatric manifestations in survivors after alcohol coma in order to be able to consider treatment.

MATERIALS AND METHODS

This is a retrospective study of consecutive medical records seen at Neuropsychiatrie, Centre Hospitalier Universitaire (CHU) de Befelatanana, 12 months, from January 2017 to December 2017. Included were patients who experienced alcohol comas with neuropsychiatric symptoms referred to Neuropsychiatry after resuscitation. However, it does not include patients with no psychological or neurological manifestations on waking and excludes unknown patients brought in by firefighters, patients with incomplete records and patients discharged against medical advice.

RESULTS

Out of 1304 patients seen in the neuropsychiatry department during the study period, we collected 123 medical records, or 9.4% of admission to the neuropsychiatry department, CHU Befelatanana. Of these, one hundred and ten (89%) are male compared to thirteen (11%) female. Our selected patients ranged in age from 15 to 77 with an average age of 37 years. The majority of our patients are farmers and the low level of education. Seventy-six patients (61.79%) were married, thirty-three (26.83%) were single, and fourteen (11.38%) were divorced and widowed.

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After coma ethyl, the main neurological clinical manifestations were withdrawal syndrome (n = 90), convulsive seizure and status epilepticus (n = 60), encephalopathy Gayet Wernicke (n = 22), the syndrome korsakoff (n = 12), pellagous encephalopathy (n = 7), cerebellar syndrome (n = 30), alcoholic polyneuropathy (n = 40), optic neuropathy (n = 3), alcoholic dementia (n = 6). Psychiatric manifestations were primarily depression (n = 16) and alcoholic psychosis (n = 10)

DISCUSSION

In our study, during a 12-month period (from January 2017 to December 2017), in the service of Neuropsychiatrie CHU Befelatanana, we identified 123 cases of alcohol comas awakened after resuscitation and then complicated neuropsychiatric problems. The age of our patients with neuropsychiatric disorders after an alcohol coma ranges from 15 to 77 years. The most vulnerable age group is 30 to 40 years, which represents 40% of cases. This could be explained by the fact that this age group corresponds to the maximum professional activity. Thus, alcohol often becomes a habit after the stressful time of work. It is considered as a means of release to rid problems and to relieve fatigue. Also, young people consider it a product of "pleasure", and adolescence is often the age of experimentation with psychoactive products, especially alcohol. Young people tend to come together to create a drinking atmosphere. Predictors of adolescent alcohol use and abuse are exercised by peer groups. Raveloson and his teams also found a result close to our study whose average age was 48 years with extreme from 16 to 80 years (Raveloson, 2009). The male dominance was found again, it constitutes the 89% of our patients. Indeed, several publications determine that men generally remain more frequent drinkers than women (Raveloson, 2009; Broucker, 2013). Low incomes and the unemployed are the most found. The cause-and-effect relationship can be explained by the fact that heavy drinkers are at greater risk of losing their jobs and unemployment often leads to increased alcohol consumption. Another explanation is that the absence of daily occupation or responsibility among the unemployed leads to a daily frequentation in the world of ethylism which evolves towards daily consumption, dependence and chronicity (Manandromalaza, 2004). Among our patients, seventy-six patients (61.79%) were married, thirty-three (26.83%) were single and fourteen (11.38%) were divorced and widowed. These percentages explain the importance of socio-economic factors in alcoholism. Marital conflict, family problems and professional problems promote this alcoholism. 1, 3) After awakening, the ethyl comatose patients had presented withdrawal syndromes that evolve in three stage (Mantz, 2011; Patrice, 2002). The first is simple weaning. The patient presents behavioral disorders with psychomotor agitation and instability; violent anxiety, anxiety, nightmares; insomnia. Neurological disorders such as rapid, ample tremors of the extremities, especially in the hand; muscle cramps; oral language disorder to dysarthria type. Neurovegetative disorders marked by profuse sweating; tachycardia, high blood pressure and digestive disorders especially abdominal pain, diarrhea, anorexia, nausea and vomiting. The second stage is the pre-delirium tremens marked by behavioural disorders a type of confuso-oniric state associating mental confusion, delirium and onirism, significant psychomotor agitation an obnubilation of consciousness; hallucinatory disorders with zoopsies type i.e. the patient sees monstrous animals, rats, spiders that they assault; multisensory hallucinations auditory or olfactory.

From neurological disorders to a type of temporo-spatial disorientation, the patient recognizes neither the date, nor the place where he is. At this stage the patient seems lost and can carry out self-acting or hetero-aggressive passages to escape these hallucinatory experiences. The last stage is the delirium tremens proved, it is the aggravation of the patient's previous condition. It manifested itself in confuso-oniric disorders accompanied by a major central hyperthermia and resistant to antipyretics; tissue and central dehydration; metabolic disorders such as hypokalemia, hydro-electrolytic disorders, rhabdomyolysis. Rapidly, this serious condition can cause central neurological disorders such as seizures or even death. The seizure and/or epileptic seizure could be explained by electrical disorders in the brain. There is a balance between inhibitory Neurotransmitters represented by Gamma-Amino-Buturic Acid (GABA) and N-methyl-D-aspartate (NMDA) excitatory Neurotransmitters. But in case of acute alcohol intoxication, the alcohol binds to the GABA inhibitor by inhibiting Glutamate NMDA. Thus, the brain is in a state of hyperexcitability; this hyperexcitability of the brain neurons is the cause of seizures (OMS, 2007; Pic, 2015; Vandermersch, 2009). Korsakoff's syndrome is the chronic form of Gayet-Wernicke encephalopathy. It is characterized by a confounding syndrome consisting of: anterograde memory disorders, temporo-spatial disorientation, fabrication, and false recognition. Memory disorders are produced by the combined involvement of the mammary and thalamus bodies or could be caused by the involvement of the diencephalon and hippocampus following chronic ethylism (Vuadens, 1998). Pellar encephalopathy consisting of mental confusion, agitation with oppositional hypertonia and myoclonias; significant extrapyramidal rigidity (hypertonia of the neck with hypertonia in flexion of the 4 limbs, a skin rash with the presence of chronic pruritic dermatosis on the face, leg. It could be explained by the deficiency or lack of vitamin PP (nicotinic acid) complicated by severe malnutrition linked to chronic alcoholism (Vuadens, 1998; Laure et al., 2008; Phan, 2013; Jacobs, 2009). They also exist cerebellar attacks revealed by disorders of balance, a widening of the polygon of sustentation frequently improves gradually during their hospital stay but it can leave permanent sequelae. This is due to cerebellar incoordination during acute intoxication or cerebellar atrophy following chronicity of ethyl intoxication (Jacobs, 2009; Line, 2013).Oculomotor disorders appear following damage mainly to the VI nerve, sometimes the III nerve, or damage to the intrinsic musculature linked to a deficiency in vitamin B6. Supranuclear or inter-nuclear paralysis is also reported. Ethyl polyneuropathy is the demyelination of small fibers following chronic alcohol consumption in the context of a length-dependent polyneuropathy (Line, 2013; Vuadens, 1998). Optic neuropathy could also be explained by bilateral and asymmetric damage to the optic nerves, which is manifested by a decrease in visual acuity, sometimes dyschromatopsia. Alcoholic dementia is always linked to chronic intoxication. Alcohol consumed excessively over a long period of time can cause brain damage and symptoms similar to those of dementia. There are disturbances in memory, learning, psychomotor speed, space appreciation, and decreased reasoning ability. There is an absence of moral and social conscience and an absence of a sense of responsibility (14, 15). Alcohol depression is a common manifestation after an alcoholic coma. This depression is most often secondary to alcohol dependence in 70% of cases. Contrary to popular belief, chronic alcohol intake is a very bad antidepressant, it

even has a depressant effect (which causes depression) and we have seen this in our patients who have lived in a depressed mood with a dark face with an inexpressive and friendly face, cold tact and rare speeches. Their puffy, coppery and red brick faces characterize their chronic alcohol intoxications. Alcoholic psychosis is the most frequent form of chronic alcoholic delusions, the onset of which is brutal after an acute confuse-oniric state with hallucinatory mechanisms, jealousy themes. The evolution is towards a progressive mental regression in favour of an alcoholic psychosis (Lariviere, 2001; Ihadadene, 2015).

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