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Hypoglycaemic hemiparesis in a Nigerian alcoholic

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Summary

Alcoholism is rarely reported as a clinical problem in Nigerians. This report is of a Nigerian alcoholic who presented with chest infection and alcohol withdrawal symptoms and subsequently developed transient hemiparesis which responded to correction of hypoglycaemia. Hypoglycaemia should be considered in all alcoholics presenting with hemiparesis.

Keywords: *Alcohol, hypoglycaemia, hemiparesis, withdrawal, Nigerian*

Résumé

L'alcoolisme est rarement rapportécomme une probléme clinique chez les Nigérians. Ce rapport est basé sur tel individu qui se presente avec l'infection de la cage thoracique, les symptomes alcooliques, et ensuite developpe une hémiparesie qui répondait à la correction de l'hypoglycémie. L'hypoglycémie pourrait etre considerée chez tous les alcooliques se presentant avec l'hémiparesie.

Case report

J D. is a 36-year-old civil servant who was admitted on 10th of March 2002 with left-sided pleuritic chest pain, breathlessness and high-grade fever of three days duration. He also had cough productive of mucoid sputum as well as throbbing headaches. He had fluctuating episodes of confusion and incoherent speech as well as inappropriate defecation and micturition. He had well formed visual hallucinations of a man looking at him from the roof holding a stick and wearing a red shirt, as well as fixed delusions of being a divorcee with three children (his friends and relatives insist that he had never been married).

There was no past or family history of psychiatric illness and he had no preceding history of trauma. He had a background history of significant alcohol ingestion of about three to five bottles of beer a day for about twenty years associated with poor eating habits. Drinking took primacy in his daily routine. He started drinking regularly at 10.00 a.m. when his local drinking bar opened. The CAGE screening test however did not demonstrate alcohol-related problems as he denied having the need to cut down or have an 'eye-opener', and he also denied feelings of guilt or anger related to his drinking. There was no history of consumption of illicit drugs. He was unmarried and his relatives had withdrawn from him on account of his attachment to alcohol.

On examination, he was sweating and had a fever of 38 degrees centigrade. He had a regular heart rate of 120/minute with a blood pressure of 90/70 mmHg with normal heart sounds. He was tachypnoeic with a respiratory rate of 40/minute and also had bronchial breath sounds in his right lower lung zone. He was conscious but had frequent episodes of confusion and incoherent speech but there were no focal motor or sensory deficits. A chest X-Ray done showed bilateral lower lobe consolidation while sputum culture grew coliform species. Liver function tests showed a markedly elevated gamma- glutamyl transferase of 190 u/L. Aspartate transaminase was 17 iu/L with an AST: ALT ratio of about 2:1. Alkaline phosphatase was also marginally elevated at 226 iu/L. Haematological tests showed a low white blood cell (WBC) count of 1.5×10^3, an elevated mean corpuscular volume (MCV) of 104 fL and a rapid erythrocyte sedimentation rate (ESR) of 70mm/Hr. Random blood sugar was 80mg/dL on admission.

He was admitted to the ward and was advised on appropriate feeding. He was commenced on antibiotics (pefloxacin, gentamicin and metronidazole) with significant resolution of respiratory symptoms. He continued to have inappropriate behaviour, irrational speech and hallucinations for which he was placed on haloperidol. Thiamine supplementation was instituted.

Six days after admission, confusion worsened and he was discovered to have left sided weakness. It was reported then that he had not been feeding adequately due to financial constraints. On examination, he was dysphasic with a right hemiparesis (power grade 3). Sensory deficits were difficult to assess. He had no meningeal signs. A random blood sugar done then was 25mg/dL. He was placed on oral and intravenous glucose

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with resolution of hemiparesis within 30 minutes. A brain computerised tomographic (CT) scan done the following day showed no abnormality. He had no recurrence of neurological features thereafter. He made a successful recovery and was discharged three weeks after admission.

Discussion

Alcoholic beverages are commonly used in Nigeria with about 50% of primary care patients reporting regular consumption[1]. While alcohol abuse was documented in only about 1.7% of alcohol imbibers, 28% have been reported to have alcohol-related medical problems [1,2]. The neurological complications of alcoholism have however not been well-characterised in Nigerians. Our patient presented with a spectrum of clinical features which highlight some of the major manifestations of alcohol consumption.

The patient had biochemical and haematological evidence of significant alcohol ingestion in the form of a raised gamma glutamyl transferase (GGT) and an increased mean corpuscular volume (MCV). Alcohol intake is an important determinant of raised serum GGT activity and this is more frequently so in Nigerian than in European alcoholics[3,4]. The subject in this case did not have other conditions which could have accounted for his raised GGT levels such as obesity, hypertension, and myocardial infarction[5,6]. The 2:1 ratio of AST to ALT in the patient was also suggestive of alcohol suppression of ALT activity [7] . Both AST and ALT levels were however within normal limits. The normal CAGE test in our patient emphasises the fact that most screening tools for alcohol related problems are more useful for detecting alcohol abuse than for detecting harmful or hazardous drinking[1,8,9].

The main clinical presentation of the patient was with confusion, sweating, hallucinations and delusions. These are typical features of delirium tremens although this has been reported to be relatively uncommon in Nigerian alcoholics [4,10]. It is therefore possible that hypoxaemia from chest infection was also contributing to his presenting symptoms.

The development of hemiparesis heralded hypoglycaemia in this patient. All patients with severe hypoglycemia have been reported to experience neurological manifestations and these include confusion, abnormal behaviour, convulsions, coma, transient and rarely permanent hemiparesis[11]. Hypoglycaemic hemiplegia is most commonly seen diabetics due to the frequent use of hypoglycaemic medications [12-15]. In alcoholics, hypoglycaemia is less common and the aetiology is thought to be alcohol-induced inhibition of gluconeogenesis along with starvation resulting from poor eating habits [16,17]. There is a reported predilection for hypoglycaemic hemiplegia to be right-sided and this was the case in this patient [18,19]. Correction of the hypoglycaemia also typically results in rapid clearance of the hemiplegia as demonstrated in this subject[18]. Hypoglycaemic hemiplegia can occur repetitively [20-22]. Because of this, a high index of suspicion is needed to exclude vascular transient is chaemic attacks (TIA), seizures, migraine, anxiety and hypotension [23]. Hypoglycaemia can also present with just dysphasia without hemiplegia, making the diagnosis more difficult [24].

No underlying brain disease has been reported in the vast majority of subjects presenting with hypoglyaemic hemiplegia [18]. While selective neuronal vulnerability has been suggested as the most likely cause, vasospasm, regional blood flow disturbances and underlying ischaemic disease may play a part [18,21,25-27]. The CT scan done in our patient was normal and this is in keeping with the fact that neuro-radiological investigation of hypoglycaemic hemiplegia frequently fails to show any abnormalities [28]. During episodes of hypoglycaemic hemiplegia however, hypodensities and hypoperfusion have been demonstrated by CT and single photon emission computerised tomography (SPECT)[19,25]. Electroencephalogram (EEG) has also demonstrated abnormalities in the form of continuous bilateral fronto-parietal slow wave activities during an episode of hypoglycaemic hemiplegia [28]. All these changes however were reversible with correction of hypoglycaemia.

The outcome of hypoglycaemic hemiplegia is generally good but permanent brain damage can occur if it is not recognised early[17]. Death due to the hypoglycaemia itself is rare and residual focal deficits were found in only a few instances [29]. To improve the outcome, all alcoholics presenting with neurological manifestations should also have thiamine deficiency corrected [30]. Alcohol withdrawal is best treated with benzodiazepines although beta-blockers, clonidine, and carbamazepine are also useful[31]. To prevent recurrence, the best modality is abstinence from alcohol which can be achieved with disulfiram, naltrexone or acamprostate[31-34]. These drugs are however not readily available in Nigeria.

There is a need to consider hypoglycaemia in any patient presenting with neurological features, and the

blood glucose concentration needs to be monitored and corrected as a routine in alcoholic subjects even if the screening test does not suggest alcohol abuse.

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