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Causes of Medical Coma in Adult Patients at the University College Hospital, Ibadan Nigeria

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Summary

Aims and objectives*: Diseases of medical origin leading to coma account for 3 -15% of emergency hospital admissions in developed countries. There is dearth of data on causes of medical coma in adults in Nigeria in particular and Africa in general. This study is to*
determine the causes of coma in adult patients admitted at the medical emergency unit and wards of the University College Hospital (UCH) Ibadan.
Patients and methods*: A prospective study of two hundred consecutive adult unconscious patients seen at the medical emergency unit of UCH, Ibadan, from August 2004 to March 2005, was undertaken using a structured clinical history and physical examination protocol, and results of relevant diagnostic investigations, including post-mortem.*
Results*: Medical causes of coma constituted 10% of all emergencies and 3% of total hospital admissions respectively during the 8- month period. Sixty six percent were males. The age group 20-59 years were affected most (76.5%). Four commonest causes were: Acute stroke (33%), diabetic emergencies (12.5%), uraemic encephalopathy and meningitides (11% each). Four least causes were cerebral malaria (1.0%), hypertensive encephalopathy, alcohol and gamalline poisoning (0.5% each). Four common predisposing factors which also had significant male predominance were systemic hypertension (38.5%), diabetes mellitus (14%), alcohol and substance abuse (12.5%), and HIV/AIDS (11.5%).*
Conclusions*: Hypertensive stroke and diabetic coma constituted the commonest medical causes of coma. Thus preventive measures such as public health enlightenment campaigns for lifestyle modifications, routine blood pressure and glucose examinations are necessary to avert their disastrous consequences.*

Keywords*: Glasgow coma scale, medical causes of coma, medical emergency.*

Introduction

Coma is a deep sleep-like state in which the patient is totally unaware of self and environment and has no sleep-wake cycle, although there may be inappropriate response to painful stimuli[1]. Clinically a patient is said to be in coma when the Glasgow coma scale score is ≤ 8 [2]. Current biological and clinical evidence indicates that coma occurs when the connection between the cerebral cortices and the brainstem ascending reticular activating system (ARAS) specifically

the ARAS-thalamo-cortical pathway is disrupted, lost or deactivated, by diffuse insults to both cerebral cortices or upper brainstem from exogenous or endogenous toxins, hypoxia, extensive trauma, widespread vascular damage and raised intracranial pressure [3].

Coma may occur as a progression or complication of an underlying illness such as brain infections, stroke, hypo- and hyperglycemic emergencies, hypo- and hypernatremia, hypo- and hyperthyroidism, seizures, drug intoxication and poisoning, sepsis, hypertensive crises and emergencies, cardiac arrest, hypovolemic shock, leukemias and lymphomas, respiratory failure, hepatic and uraemic encephalopathies [4]. In these conditions, coma occurs in response to increased intracranial pressure and subsequent compression of the brainstem by either supratentorial lesions, meningeal infections or subarachnoid haemorrhage ; infratentorial lesions in the posterior fossa; a metabolic, endocrine, or anoxic encephalopathy with diffuse involvement of the cerebral hemispheres; or generalised tonic-clonic seizures[5].

The evaluation of the acutely comatose patient is a common situation encountered in the emergency room. The examination should begin by attempting to obtain information from those who are familiar with or who have had some contact with the patient, including relatives,

neigbours, police, and the emergency services team where available. A history of systemic hypertension, heart disease, diabetes melitus, drug abuse, epilepsy, or recent head trauma is invaluable[6-7].

Hospital admission figures for medical coma are as disparate as the causes of coma and are affected by the socio-demographic and geographical locations of the subjects. For example, a multi-center study of causes of coma in 310 patients between 1973 and 1976 by Bates et al[8], in Europe and United States of America (USA) showed that while 58% of the American patients had hypoxia-ischaemia, 57% of the British patients had cerebrovascular disease, including subarachnoid haemorrhage. This result was said to be a reflection of the different emergency systems adopted by each country. For instance, the USA is known to have an efficient and accessible cardiopulmonary resuscitation system which does not depend on a referral system unlike in Europe[8]. Other causes of coma reported in that study were liver cirrhosis (22 cases), paracetamol-induced hepatitis (5 cases), acute viral hepatitis (3 cases), other hepatic abnormalities (3 cases), uraemia (6 cases), acid-base, water, or electrolyte abnormalities (3 cases), mass lesions with haerniation syndromes (7 cases), meningitis and encephalitis (6 cases) and hypothermia (1 case). Extension of the study to 500 patients by Levy et al[9] over a 4 year period revealed that medical coma accounted for 15% of emergency admissions and was mainly due to hypoxia-ischaemia (210 cases), cerebrovascular disease (181 cases), hepatic encephalopathy (51 cases), metabolic disorders (19 cases), infections (16 cases), hypoglycaemia (12 cases) and mass lesions in the brain (11 cases).

Studies from some African countries found that cerebral infections and metabolic disorders were the main causes of coma[10-12]. Analysis of 170 causes of non-traumatic coma in the intensive care unit of a Lusaka hospital revealed that 33 (19.4%) cases were undiagnosed while stroke constituted 32 (18.8%) cases, among other causes which were organophosphorous poisoning 23, (13.5%), cerebral malaria and eclampsia 22, (12.9% each), meningitis 13,(7.6%), 8 miscellaneous causes (mass lesions, abscesses, infections), and 2 cases each of diabetic ketoacidosis, hepatic encephalopathy, hypoglycaemia [12]. In Ibadan, Bondi reported causes of childhood coma to be cerebral malaria (55%), meningitis (13%) and encephalitis (10%)[13].

The differences in the aetiology of coma in the Europe / USA and the African studies may be a reflection of the socioeconomic differences between the peoples of these continents, because while non-communicable disorders predominated in the former (developed economics), infections were more prevalent in the latter (developing economy)[14].

In whichever form, depth or severity of its presentation, and irrespective of its cause, medical coma rarely last for more than four weeks without an outcome, in which case, either the patient dies, or goes into a vegetative state or recovers with or without psychosocial and physical dependence[15]. Because of these, every health care provider must have an understanding of the common etiology or etiologies of medical coma in the locality, as well as the complexity of its presentation, in order to have a comprehensive and holistic approach to its management[6, 16]. This study was aimed at determining the common causes of medical coma in adult patients in a teaching hospital in Nigeria.

Patients and methods.

This was a prospective study of patients, aged 16 years and above, who presented to the medical emergency unit of the University College Hospital (U.C.H) Ibadan with strictly defined coma of medical origin (Glasgow coma scale score of ≤ 8), from August 2004 to March 2005. It was done with the permission of the Institutional Research Committee of the Hospital and the written consent of the patients' relations.

Study site: UCH Ibadan, which was founded in 1948, is the premier teaching hospital in Nigeria. It is a referral centre for Ibadan (said to be the largest town in West Africa with a population of more than one million people) and other major towns and villages in the Western part of Nigeria. The hospital has a well organised medical emergency unit headed by a physician with all the medical subspecialties represented. All emergencies, including unconscious patients, were first evaluated and investigated at the hospital emergency unit where a presumptive diagnosis must be made before such patients were transferred to the appropriate medical subspecialties or intensive care unit (ICU) usually within 24 hours of presentation. All the coma patients in this study were co-managed by the neurology unit (where the authors belonged) and any of the following subspecialties: cardiology, dermatology, endocrinology, gastroenterology, hematology, nephrology and respiratory medicine depending on the clinical indications. Post-mortem examinations were compulsorily performed if death occurred within 24 hours of presentation to the hospital to ascertain the cause(s) of death.

Sample size determination[17]: The sample size of 200 was determined from the formula,

$$N = z^2 Pq / d^2, \text{ where}$$

N= the desired sample size (when population is greater than 10,000)

z = the standard normal deviate at 95% confidence interval (1.96)

P= admission rate of coma in the literature (15%)[5]

d^2 = degree of accuracy at 0.05

q = 1.0- p (1.0- 0.15) = 0.85

Therefore, N= (1.96^2 x 0.15 x 0.85 / $0.05^{2)}$ = 0.489804 / 0.0025 = 195.9 approximated to 200,

to take care of losses or referrals.

Exclusion criteria.

The following patients were excluded from the study: Patients below 15 years; traumatic coma; altered sensorium with GCS score above 8; and inconclusive diagnosis.

Study procedure

On presentation to the emergency unit, each patient was evaluated according to methods described by Caronna and Simon, 1979 [6], and Malik and Hess, 2002 [7], including detailed history, physical and neurological examinations. Predisposing factor (s), co morbidities and intervening complication(s) associated with each presenting illness were documented and presumptive diagnosis made using standard criteria for each condition. Patients were then monitored daily for a maximum of 28 days for outcome[15]. During this period results of ancillary investigations were documented and used to confirm the cause of coma. However, a study was terminated if a patient died before the 28th day.

Diagnostic criteria for various causes of coma

i. Acute intracerebral haemorrhagic and large infarctive stroke: fifty five patients had computerized tomographic (CT) scan of the brain confirmation of diagnosis, 4 died within 24 hours of presentation and so had autopsy, while diagnosis was based on clinical grounds in the remaining 7 patients[18].

ii. Hypertensive encephalopathy: severe headache and rapid loss of consciousness, convulsions, recent onset severe rise in blood pressure (systolic > 220mmHg and diastolic > 130 mmHg), papilledema, diffuse cerebral edema on CT scan and granular casts on urine microscopy[19]

iii. Uremic coma: features of chronic renal failure, ± history of renal dialyses, abnormal serum electrolytes (bicarbonate < 15 mmol/ L, potassium > 5 mmol/L), creatinine (> 900 µmol/L)[20,21].

iv. Diabetic coma: ± history of diabetes mellitus, signs of severe dehydration, ± acetone breath and ketonuria, plasma glucose > 22 mmol/L and serum osmolality > 320 mOsm/ kg[21,22]

v. Hepatic coma: features of acute fulminant hepatitis and hepatic failure; deranged liver function tests, prothrombin time and serum proteins; abnormal liver size and architecture on ultrasonography[23,24].

vi. Hypoglycaemic coma: history of oral hypoglycaemic agent (OHA) ingestion or insulin injection in a known diabetic; diaphoresis, tachycardia ± seizures, and plasma glucose of < 2.2 mmol/L[25].

vii. Meningitides (acute pyogenic, tuberculous and cryptococcal) : acute pyogenic meningitis was recognized by the presence of classic triad of fever, headache with photophobia and neck stiffness; signs of meningeal irritation; characteristic cerebrospinal fluid (CSF) findings and positive culture of Streptococcal pneumoniae[26, 27]. Tuberculous meningitis was confirmed from a combination of clinical evaluations, characteristic CSF (although Ziehl-Neelsen stains for acid fast bacilli were negative); chest radiology and CT scan findings[28]. Cryptococcal meningitides were confirmed in HIV/AIDS patients with positive Indian ink stain for Cryptococcus neoformans in the CSF[29].

viii. Acute lymphoblastic meningitis was also confirmed from peripheral blood and bone marrow films, and CSF cytology[30].

ix. HIV encephalopathy: HIV seropositivity, low CD4+ cell counts, clear CSF with lymphocyte pleocytosis, negative bacterial cultures and no other identifiable cause except HIV (although plasma HIV load, viral cultures and titres for acute and convalescent sera were not done) [31].

x. HIV- associated brain lymphoma: headaches, progressive focal neurological deficit, HIV seropositivity, low CD4+ cell counts and characteristic high density contrast enhancing mass (es) with surrounding massive oedema[32].

xi. Cerebral malaria: Hyperpyrexia with rapid loss of consciousness, asexual forms of Plasmodium falciparum in peripheral blood films, normal CSF, negative blood, urine and stool cultures[33].

xii. Cardiac arrest: 6 patients had clinical, electrocardiographic and echocardiographic evidences of acute myocardial infarction[34], while 2 others had postmortem confirmation of diagnosis.

xiii. Sepsis syndrome: clinical presentations were variable, but diagnoses were based on the presence of temperature > 38 ^{0}C or < 36 ^{0}C, heart rate > 100 beats per minute, respiratory rate > 20 breaths per minute, leucocytosis, anemia, hypotension, oliguria, multiple organ dysfunctions and microbial culture results[35,36].

xiv. Alcohol intoxication and Gamalline poisoning: diagnoses were based on historical evidences and identification of Gamalline by the Pharmacy department.

Statistical analysis

Data was subjected to frequency distribution, Student't' test, Chi-Square and multiple response analyses using the EPI INFO 6 statistical software. Level of significance (p-value) was accepted as less than 0.05.

Results

Medical coma constituted 9.8% (200/2033) of emergency and 3.1% (200/6548) of total hospital admissions during the study period. The study population comprised 132 (66%) males and 68 (34%) females, with a male to female ratio of 2:1. The mean age of the patients was 50.0 ±18years, the females being slightly older than the males (t=-0.514, P>0.05). Their age groups were distributed as follows: 40-59 years, middle aged [100 (50%)], > 60 years, elderly [46 (23%)] and 20-39 years, youth [53 (26.5%)]. The only adolescent (< 20 years) was a male (0.5%) (Table I).

A total of twenty medical causes of coma were identified. These include: 33% (66 patients) with acute stroke made up of 26.0% (52 patients) with intra-cerebral hemorrhage and 7.0 % (14 patients) with infarctive stroke, followed by 17.5% (35 patients) with hyper- and hypoglycemic emergencies, 12.0% (24 patients) with uraemic coma. The other causes were meningitides in 11.0% (22 patients), sepsis syndrome in 10.5% (21 patients), hepatic coma in 6.0% (12 patients), cardiac arrest in 4.0% (8 patients), HIV-associated brain disorders in 3.5% (7 patients) and cerebral malaria in 1.0% (2 patients) patients respectively. Hypertensive encephalopathy, alcohol and pesticide (Gamalline) poisoning were seen in 1(0.5%) patient each respectively.

Table II showed that apart from diabetic coma and acute lymphoblastic meningitis with a female predominance, and cerebral malaria with equal sex distribution, other causes had overwhelming male predominance (P< 0.05).

The distribution of coma according to patients' age groups showed that for the elderly:- 13 (6.5%) patients had HONK , 11 (5.5%) had acute infarctive stroke and 5 (2.5%) had OHA-induced hypoglycemia. Forty-five (22.5%) of the middle- aged patients had acute intracerebral haemorrhagic stroke, 8 (4.0%) had sepsis syndrome, 6 (3.0%) had hepatic failure, 4 (2.0%) had Cryptococcal meningitis, 5 (2.5%) had acute lymphoblastic meningitis, 3 (1.5%) had HIV-associated CNS lymphoma and 1 patient (0.5%) had alcohol intoxication. Thirteen (6.5%) young patients were affected by uraemic encephalopathy , 5 (2.5%) each by tuberculous and Streptococcal pneumoniae meningitides respectively, 4 (2.0%) by cardiac arrest , 3 (1.5%) each by DKA and insulin-induced hypoglycemia respectively and 1 (0.5%) patient by Gamalline poisoning. Other causes such as hypertensive encephalopathy and cerebral malaria (2

patients, 1.0% each) were uniformly distributed between the middle-aged and the youth (Table II).

Results of multiple response analyses showed that all the predisposing factors were significantly higher in the males ($p < 0.05$). Their distribution in terms of age groups showed that while systemic hypertension, diabetes mellitus and obesity were most prominent in both the elderly and middle aged; alcohol/substance abuse and hepatitis B surface seropositivity were more in the middle aged patients while HIV was the most prominent factor in the youth (Table III).

Table i:
Age and sex distribution of patients

Age (years)	Male N (%)	Female N (%)	Total N (%)
Adolescence (< 20)	1 (0.5)	0 (0.0)	1 (0.5)
Young age (20 -39)	30 (15.0)	23(11.5)	53(26.5)
Middle age (40 -59)	83(41.5)	17 (8.5)	100(50.0)
Elderly (> 60)	18(9.0)	28(14.0)	46(23.0)
Total	132(66.0)	68(34.0)	200(100.0)
Mean	49.5±16.1	50.9±20.9	50±17.8
	t = -0.514,	p -value 0.61	

N= number of patients

% = percentage of patients in bracket

Table ii:
Distribution of causes of medical coma by sex and age groups

S.No	Cause of coma	Gender*		Age groups (years)			Total N (%)
		Male N (%)	Female N (%)	≥ 60 N (%)	40-59 N (%)	≤ 39 N (%)	
1	Acute intracerebral hemorrhagic stroke	43 (21.5)	9 (4.5)	0 (0.0)	45 (22.5)	7 (3.5)	52 (26.0)
2	Uremic coma	15 (7.5)	9 (4.5)	4 (2.0)	7 (3.5)	13 (6.5)	24(12.0)
3	Hyperosmolar nonketotic coma (HONK)	9 (4.5)	12 (6.0)	13 (6.5)	6 (3.0)	2 (1.0)	21 (10.5)
4	Sepsis syndrome	13 (6.5)	8 (4.0)	8 (4.0)	10 (5.0)	3 (1.5)	21 (10.5)
5	Acute infarctive stroke	7 (3.5)	7 (3.5)	11 (5.5)	3 (1.5)	0 (0.0)	14 (7.0)
6	Hepatic failure	9 (4.5)	3 (1.5)	3 (1.5)	6 (3.0)	3 (1.5)	12 (6.0)
7	Cardiac arrest	5 (2.5)	3 (1.5)	2 (1.0)	2 (1.0)	4 (2.0)	8 (4.0)
8	Oral hypoglycemic agent(OHA) -induced hypoglycemia	6 (3.0)	1 (0.5)	5 (2.5)	2 (1.0)	0 (0.0)	7 (3.5)
9	Tuberculous meningitis	5 (2.5)	2 (1.0)	0 (0.0)	2 (1.0)	5 (2.5)	7 (3.5)
10	Streptococcal pneumoniae meningitis	3 (1.5)	2 (1.0)	0 (0.0)	0 (0.0)	5 (2.5)	5 (2.5)
11	Cryptococcal meningitis	2 (1.0)	3 (1.5)	0 (0.0)	4 (2.0)	1 (0.5)	5 (2.5)
12	Acute lymphoblastic meningitis	0 (0.0)	5 (2.5)	0 (0.0)	5 (2.5)	0 (0.0)	5 (2.5)
13	HIV- associated CNS lymphoma	5 (2.5)	0 (0.0)	0 (0.0)	3 (1.5)	2 (1.0)	5 (2.5)
14	Diabetic ketoacidotic coma (DKA)	2 (1.0)	2 (1.0)	0 (0.0)	1 (0.5)	3 (1.5)	4 (2.0)
15	Insulin-induced hypoglycemia	3 (1.5)	0 (0.0)	0 (0.0)	0 (0.0)	3 (1.5)	3 (1.5)
16	HIV encephalopathy	1 (0.5)	3 (0.5)	0 (0.0)	1 (0.5)	1 (0.5)	2 (1.0)
17	Plasmodium falciparum cerebral malaria	1 (0.5)	3 (0.5)	0 (0.0)	1 (0.5)	1 (0.5)	2 (1.0)
18	Hypertensive encephalopathy	1 (0.5)	0 (0.0)	0 (0.0)	1 (0.5)	0 (0.0)	1 (0.5)
19	Alcohol intoxication	1 (0.5)	0 (0.0)	0 (0.0)	1 (0.5)	0 (0.0)	1 (0.5)
20	Gamalline poisoning (3, 17-dihydroxy pancuronium)	1 (0.5)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.5)	1 (0.5)
	TOTAL	**132 (66)**	**68 (34)**	**46 (23.0)**	**100 (50.0)**	**54 (27.0)**	**200 (100.0)**

Chi square (X^2) = 11. 54, *$p < 0.05$ (males > females)
N= number of patients
% = percentage of patients in bracket

Table iii:
Distribution of predisposing factors by sex and age groups

S.No	Predisposing factor	Gender*		Age group (years)			Total N (%)
		Male N (%)	Female N (%)	≥ 60 N (%)	40-59 N (%)	≤ 39 N (%)	
1	Systemic hypertension	55(27.5)	22 (11.0)	37 (18.5)	31(15.5)	9 (4.5)	77 (38.5)
2	Diabetes mellitus	18 (9.0)	28 (14.0)	16 (8.0)	9 (4.5)	3 (1.5)	28 (14.0)
3	HIV	19 (9.5)	9 (4.5)	0 (0.0)	11 (5.5)	12 (6.0)	23 (11.5)
4	Alcohol abuse	16 (8.0)	7 (3.5)	2 (1.0)	12 (6.0)	3 (1.5)	17 (8.5)
5	Obesity	17 (8.5)	0 (0.0)	8 (4.0)	8 (4.0)	0 (0.0)	16 (8.0)
6	Hepatitis B surface antigen seropositive	9 (4.5)	7 (3.5)	3 (1.5)	6 (3.0)	3 (1.5)	12 (6.0)
7	Substance abuse	8 (4.0)	4 (2.0)	3 (1.5)	3 (1.5)	2 (1.0)	8 (4.0)

Chi square (X^2) = 10. 06, *p < 0.05(males > females)
N= number of patients, % = percentage of patients in bracket

Discussion

Medical coma accounted for about 10% of medical emergencies and 3% of total hospital admissions. The 10% of emergency admission due to medical coma in this study was lower than the 15% reported by Levy et al[9] in their multi-centre study in Europe and USA on nontraumatic coma. Some factors may be responsible. While our study was carried out in a single hospital for a period of eight months only, Levy and his colleagues did a multi-center study involving 4 hospitals in two countries over a period of 4 years. The male to female population ratio of 2:1 in this study, however, agreed with most studies in non traumatic coma all over the world [9-13]. A major reason for this is the higher risky lifestyle of males as compared to females.

In many societies in Africa, gender bias in the pattern of hospital attendance had been reported. The reason adduced for this is that the male being the breadwinner and the person who takes decisions on behalf of the family is usually valued, and therefore, the tendency is to bring the sick male to hospital more often than the female counterpart[37]. The part played by this observation in this study is difficult to discern accurately. Also, all the identifiable predisposing factors were significantly higher in the male patients. The middle and the young age groups were worst affected. These groups are not only the most active and productive segment of the society but are also the most explorative and therefore would be the most vulnerable, possibly due to higher risky lifestyle[38,39]

Haemorrhagic stroke as the commonest cause of coma in this study affected mainly middle aged males, a phenomenon reported in many studies on stroke [37, 38, 40, 41]. It accounted for 79% of all strokes in this study, a figure higher than the 65% reported by Ogun in a retrospective study on acute stroke mortality in Lagos [18]. The higher frequency in this study most probably resulted from complete documentation and utilisation of brain imaging unlike in Ogun's study. Acute stroke as an entity constituted 33% of the causes of coma thereby reaffirming it as a major cause of morbidity in this part of the world. This emerging trend has been attributed to an increase in the incidence of cardiovascular and cerebrovascular risk factors for stroke such as systemic hypertension [19, 38] diabetes mellitus, alcohol, substance abuse, HIV and obesity in our population[21,37-40].

Systemic hypertension and diabetes mellitus have been described as the most important predisposing factors for stroke in Nigerians and indeed Africans [37, 38]. Both are independently associated with a high rate of target organ complications which are particularly accentuated in the Negroid race [20, 21, 38]. Obesity, smoking, alcohol and substance abuse are newer factors probably emanating from the influence of or the adoption of Western way of life. HIV is now a strong risk factor for stroke in the young [39,40].

Diabetic coma as an entity constituted 12.5% of all causes of coma, second only to stroke and was also an important predisposing factor for other causes of coma. It has been described as the most important cause of end-stage renal failure and uraemic coma [20,21,42].

Causes of uremic coma in this study were chronic renal failure due to either hypertensive nephrosclerosis or diabetic nephropathy. In developed countries where chronic renal failure patients receive good prophylactic and therapeutic care in the form of dialysis and transplantation, uraemia coma is not as common as in our setting where factors such as ignorance, poverty, unorthodox medical practice, self-medication and drug abuse and limited renal dialysis services, deter patients from presenting to hospitals early[43]. Furthermore, in some hospitals in Nigeria, patients infected with HIV and/or hepatitis B and C are denied haemodialysis, a practice which is not internationally accepted [44].

HIV encephalopathy, CNS lymphoma, cryptococcal and tuberculous meningitides were the AIDS-defining opportunistic infections and malignancies recorded in our patients whose mean CD4+ cell counts were 100 cells/ μL[45,46]. HIV was also implicated as a cause of multiple cerebral thrombosis and mycotic intracerebral haemorrhages in two patients[47]. Pyogenic meningitides in our series were caused by streptococcal pneumoniae only. This was not surprising because it is still the commonest sporadic cause of bacterial meningitis, particularly in the adult[27].

Sepsis constituted 10.5% of causes of coma .It is an extremely serious condition that contributed to increased morbidity and mortality in the elderly patients and those

with diabetes mellitus, AIDS and alcoholic liver cirrhosis [31, 35, 36, 42].

Oral hypoglycaemic agents and insulin were the main causes of hypoglycaemia in our patients resulting from inappropriate dosing of insulin or the oral hypoglycaemic agents by the elderly, illiterate or ignorant patients or administration of the drugs in the absence of food [25, 48].

Liver failure was the major cause of coma in patients with background liver cirrhosis and primary liver cell carcinoma[23, 24]. Fortunately drug and alcohol poisoning were uncommon in our series as only 2 cases were recorded.

Conclusion

In this study, stroke and complications of diabetes mellitus constituted the most frequent causes of coma in adults in Ibadan. The conditions showed age and gender bias. All the predisposing factors, except age, are modifiable. This thus calls for extensive population screening programmes for early detection of systemic hypertension, diabetes mellitus, HIV and hepatitis B virus infections so that both preventive and curative measures could be instituted. Public health education and awareness campaigns against risky lifestyle behaviours, poor environmental sanitation and widespread immunisations and improvement in the socioeconomic status of the population will drastically reduce morbidity from infectious diseases, stroke, cardiovascular events, diabetes mellitus, renal and liver diseases.

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