PRINCIPAL CAUSES OF COMA IN MEDICAL UNITS OF A TERTIARY CARE HOSPITAL OF PESHAWAR

Hamzullah Khan, Muhammad Ishaq Khattak, Muhammad Zarif

Department of Medicine,
Khyber Medical College, Khyber Teaching Hospital, Peshawar, Pakistan.

ABSTRACT

Objective: *To determine the principal causes of coma in patients admitted to the medical units of a tertiary care hospital of Peshawar.*

Material and Methods: *This cross sectional study was conducted at department of Medicine, Khyber Teaching Hospital Peshawar, from July 2006 to August 2007. All patients were randomly selected. Relevant information was recorded on a questionnaire prepared in accordance with the objectives of the study.*

Results: *A total of 124 patients with coma, 96(77.41%) males and 28(22.58%) females were included in the study. The age range of patients was from 12 years to 72 year with mean age of 50.5 years. The principal causes of coma were: cerebrovascular accident (CVA) 73.38% (n=91), epilepsy in 10.48% (n=13), head injury 5.64% (n=11), hepatic coma 2.41% (n=3), central nervous system(CNS) infections 2.41% (n=3), metabolic acidosis, drug abuse (1.61%) (n=2) each and cerebral malaria, hypoglycemia and uremia 0.8% (n=1) each. Risk factors for stroke recorded were hypertension in 46.2% (n=42/91), diabetes in 15.4% (n=14/91), ischemic heart disease 12.1% (n=11/91), smoking 5.5% (n=5/91), hyperlipedemia 3.3% (n=3/91) and atrial fibrillation 1.1% (n=1/91). Scoring on Glasgow coma scale showed that 74.2% (n=92) patients scored 3-8, 25.8% (n=32) scored 9-12 and none of the patients scored 13-15.*

Conclusion: *In our setup CVA is the most common cause of coma followed by epilepsy, and head injury Other minor causes recorded were metabolic acidosis, drug abuse, central nervous system infections, cerebral malaria, hypoglycemia and uremia.*

Key word: *Coma, Principal Causes, Peshawar.*

INTRODUCTION

Coma, derived from the Greek word "***koma***," meaning *deep sleep*, is a state of extreme unresponsiveness, in which an individual exhibits no voluntary movement or behavior. Furthermore, in a deep coma, even painful stimuli (actions which, when performed on a healthy individual, result in reactions) are unable to affect any response, and normal reflexes may be lost[1]. Cerebrovascular accident (CVA) is a major cause of coma. CVA is a clinical syndrome characterized by rapidly developing symptoms and/or signs of focal, and at times global (for patients in Coma) loss of cerebral functions, with symptoms lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin[2]. According to World Health Organization report 2002, total mortality due to stroke in Pakistan was 78512.[3]

Other major causes of coma reported in the literature are epilepsy or convulsive disorders[4], trauma and injuries to head especially to the skull base[5] and cerebral hematoma or dried blood spots in brain[6]. Rare causes of coma are metabolic disorders[7], hypothyroidism[8], various bleeding disorders like bleeding associated with super selective thrombolysis in a pre-adolescent with diabetic ketoacidosis[9], subarachnoid hemorrhage (SAH)[10] and central nervous system infections[11] etc. Present study was designed to determine the principal causes of coma in patients admitted to the medical units of a tertiary care hospital in Peshawar.

MATERIAL AND METHODS

A cross sectional study was conducted in medical wards of Khyber Teaching hospital Peshawar from July 2006 to August 2007. A total

of one hundred and twenty four patients with established diagnosis of coma were randomly selected. Coma was defined as a state of extreme unresponsiveness, in which an individual exhibits no voluntary movement or behavior[1]. Patients meeting the criteria for coma, irrespective of ages and sex were included. A detailed history regarding fever, trauma, fits, any infection, hypertension, diabetes, hypercholestrolemia, smoking, oral pills etc was recorded. Past history of any CNS infection, fits, risk factors of cerebrovascular disease like hypertension, diabetes, cholesterol and smoking was recorded. Investigation reports regarding CT scan brain, blood culture (for CNS infection), lumber puncture, blood pressure, fasting blood sugars and random blood sugars, cholesterol and triglyceride levels were also recorded from the ward record of the patients. Electroencephalogram (EEG) findings of patients with fits, also recorded. History of use of warfarin, heparin and aspirin was also recorded if were there.

CVA was defined as focal neurological deficit due to vascular lesions that may be cerebral infarction or hemorrhage, confirmed on C.T scan, resulting in partial or complete loss of motor and sensory activities.[12] The neurological status and prognosis of the disease were graded according to modified Glasgow Coma Scale[13]. Glasgow Coma Scale has a 15 point scoring system for parameters like eye opening, verbal and motor responses. Data was analyzed and association of risk factors with coma was studied.

RESULTS

We included a total of 124 patients meeting the criteria of coma. Ninety six (77.41%) were male patients and 28 (22.58%) were females. The age range of patients was from 12 years to 72 year with mean age of 50.5 years.

The principal causes of coma were: cerebrovascular accident 73.38% (n=91), epilepsy 10.48% (n=13), head injury 5.64% (n=11), hepatic coma due to hepatic failure 2.41% (n=3), central nervous system infections 2.41% (n=3), metabolic acidosis, drug abuse (1.61%) (n=2) each and cerebral malaria, hypoglycemia and uremia 0.8% (n=1) each (Table 1). Risk factors of stroke recorded were hypertension 46.2% (n=42/91), diabetes 15.4% (n=14/91), ischemic heart disease 12.1% (n=11/91), smoking 5.5% (n=5/91), hyperlipedemia 3.3% (n=3/91) and atrial fibrillation 1.1% (n=1/91) {Table 2}.

The neurological status and prognosis of the disease were graded according to the Glascow Coma Scale (Table 1). We divided our patients in to three groups. Group A (severe) with score 3-8, Group B (moderate) with score between 9-12 and Group C (mild) with score 13-15. Scoring on Glasgow coma scale showed that 74.2% (n=92) patients scored 3-8, 25.8% (n=32) scored 9-12 and none of the patients scored 13-15. (Table 4).

DISCUSSION

One of the most common causes of coma is stroke. Treatment varies depending on the cause. Overall, in coma cases, damage to the brain's "thinking and life support centers" has occurred [14]. In our part of the world cerebrovascular diseases are common because of heavy burden of factors

PRINCIPAL CAUSES OF COMA

Causes of coma		Number of patients (n=124)	Percentage %
Cerebrovasclar Accident (CVA)	Total	91	73.38
	Cerebral infarction	76	61.29
	Intracerebral haemorrhage	11	8.87
	Subarachnoid hemorrhage	4	3.22
Epilepsy		13	10.48
Head Injury		7	5.64
Hepatic coma		3	2.41
CNS infections	Total	3	2.41
	meningitis	2	1.61
	encephalitis	1	0.8
Metabolic acidosis		2	1.61
Drug abuse		2	1.61
Uremia		1	0.8
Cerebral malaria		1	0.8
Hypoglycemia		1	0.8

Table 1

STRATIFICATION OF RISK FACTORS OF CVA IN OUR SELECTED PATIENTS

Risk Factors of stroke	Number of patients (n=91)	% age
Hypertension	42	46.2
Diabetes mellitus	14	15.4
Ischemic heart disease	11	12.1
Smoking	5	5.5
Hyperlipedemia	3	3.3
Atrial fibrillation	1	1. 1

Table 2

like hypertension and diabetes. In present study majority (73.38%) of cases were attributed to stroke. A local study from Karachi showed that leading cause for coma was cerebrovascular diseases followed by metabolic and infectious diseases.[15] A study form India reported that cerebrovascular diseases (33%) are major contributor to coma followed by CNS infections (21%), and hepatic encephalopathy (18%), with the first two carrying relatively poor prognosis [16]. Barsic B et al [17] also correlate stoke with coma depending on the prognosis of stroke which alters the consciousness level adversely. Prognosis of stroke is well assessed by Glasgow coma scale, poorer the score higher is the rate of morbidity and mortality. In our study majority (74.2%) of patients scored 3-8 (severe coma) and none of the patients scored 13-15 (mild coma). Poor outcome was also associated with low GCS score and absence of brainstem reflexes especially absent pupillary, oculocephalic and oculovestibular responses.[16,17] In present study epilepsy was recorded in 10.48% cases as prime cause of coma. Our findings match that of Solomont T et al.[18]

In western countries trauma is a major most cause of coma as they usually have low incidence of other risks like stroke, hypertension and diabetes. In most mild traumatic brain injury (mTBI) patients suffer from several post-concussion symptoms suggestive of thalamic involvement [19]. The risk of associated blunt neurovascular injury appears to be significant in level 1 trauma patients in whom a diagnosis of skull base fracture has been made using CT. The incidence of neurovascular trauma is particularly high in patients with clival fractures. The authors recommend neurovascular imaging for Level 1 trauma patients with a high-risk fracture pattern of the central skull base to rule out cerebrovascular injuries[20]. In our study there were only 5.64% cases of traumatic head injury; the reason may be that this study was conducted primarily in medical units of the hospital. And such cases are usually managed by neurosurgical departments of other hospitals of the city.

We recorded 3(2.41%) cases of coma associated with CNS infections, 2 (1.6%) with meningitis and one (0.8%) with encephalitis. Another study conducted on the same theme has reported that patients have high risk for unfavorable outcome in adults with bacterial meningitis.[21] Kawakami A et al[22] demonstrated metabolic diseases as the leading causes of coma after CVA.

CONCLUSION

In our setup CVA is the most common cause of coma followed by epilepsy, and head injury. Other minor causes recorded were metabolic acidosis, drug abuse, central nervous system infections, cerebral malaria, hypoglycemia and uremia. GCS provides the best parameters to study the prognosis in patients with coma irrespective of cause.

REFERENCES

1. Plum F. Disturbances of Consciousness and Arousal: In Cecil Textbook of Medicine, 19th edition, W.B. Saunders Co. Philadelphia 1992: 2048-63.
2. Davenport R, Denis M. Neurological emergencies: Acute stroke. J Neurol Neurosurg Psychiatry 2000; 68:277-88.
3. Singh RB, Suh IL, Singh VP, Chaithiraphan S, Laothavorn P, Sy RG, et al. Hypertension and stroke in Asia: prevalence, control and strategies in developing countries for prevention. J Hum Hypertens 2000; 14: 749-63.
4. Sugai K. Treatment of convulsive status epilepticus in infants and young children in Japan. Acta Neurol Scand 2007; 186:62-70.
5. Feiz-Erfan I, Horn EM, Theodore N, Zabramski JM, Klopfenstein JD, Lekovic GP, et al. Incidence and pattern of direct blunt neurovascular injury associated with trauma to the skull base. J Neurosurg 2007 ;107:364-9.
6. Weigel JF, Janzen N, Pfaffle RW, Thiery J, Kiess W, Ceglarek U. Tandem mass spectrometric determination of succinylacetone in dried blood spots enables presymptomatic

GLASGOW COMA SCORE

GROUP	Glasgow coma score	Number of patients (n=124)	% age
Group A	3-8	92	74.2
Group B	9-12	32	25.8
Group C	13-15	0	0

Table 3

detection in a case of hepatorenal tyrosinaemia. J Inherit Metab Dis 2007; 30:610.

7. Qian ZY, Miao Y. Therapeutic strategy for severe acute pancreatitis and pancreatic encephalopathy. Zhonghua Wai Ke Za Zhi 2007 ;45:740-1
8. Finora K, Greco D. Hypothyroidism and myxedema coma. Compend 2007 ;29(1):19-31.
9. Zerah M, Patterson R, Hansen I, Briones M, Dion J, Renfroe B. Resolution of severe sinus vein thrombosis with super selective thrombolysis in a pre-adolescent with diabetic ketoacidosis and a prothrombin gene mutation. J Pediatr Endocrinol Metab 2007;20:725-31.
10. Schmidt JM, Rincon F, Fernandez A, Resor C, Kowalski RG, Claassen J, Connolly ES, Fitzsimmons BF, Mayer SA. Cerebral infarction associated with acute subarachnoid hemorrhage. Neurocrit Care 2007;7(1):10-7.
11. Portolani M, Tamassia MG, Gennari W, Pecorari M, Beretti F, Alu M, Maiorana A, Migaldi M. Post-mortem diagnosis of encephalitis in a 75-year-old man associated with human herpesvirus-6 variant A. J Med Virol 2005 ;77(2):244-8.
12. Khan H, Afridi AK, Ashraf S. A hospital based study on stratification of risk factors of stroke in Peshawar. Pak J Med Sci 2006; 22: 304-7.
13. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974,2:81-84.
14. Engelter ST, Gostynski M, Papa S, Frei M, Born C, Ajdacic-Gross V, et al. Epidemiology of Aphasia Attributable to First Ischemic Stroke: Incidence, Severity, Fluency, Etiology, and Thrombolysis. Stroke 2006; 37: 1379 - 84.
15. Abdullah M, Chandnal A, Khan SM. Etiology and outcome of coma - A hospital based study. J Coll Physicians Surg Pak 2001;11(9):572-5.
16. Thacker AK, Singh BN, Sarkari NB, Mishra RK. Non-traumatic coma--profile and prognosis. J Assoc Physicians India 1997; 45:267-70.
17. Barsic B, Marton E, Himbele J, Ravlic Z. Evaluation of the Glasgow Coma Scale score in critically ill infectious disease patients. Infection 1996; 24:297-300.
18. Solomon T, Dung NM, Kneen R, Thao le TT, Gainsborough M, Nisalak A et al. Seizures and raised intracranial pressure in Vietnamese patients with Japanese encephalitis. Brain. 2002; 125:1084-93.
19. Kirov I, Fleysher L, Babb JS, Silver JM, Grossman RI, Gonen O. Characterizing 'mild' in traumatic brain injury with proton MR spectroscopy in the thalamus: Initial findings. Brain Inj 2007;13:1-8.
20. Feiz-Erfan I, Horn EM, Theodore N, Zabramski JM, Klopfenstein JD, Lekovic GP et al. Incidence and pattern of direct blunt neurovascular injury associated with trauma to the skull base. J Neurosurg 2007; 107:364-9.
21. Joffee AR. Lumbar puncture and brain herniation in acute bacterial meningitis: A review. J Intensive Care Med 2007; 22:194-207.
22. Kawakami A, Nakae Y, Toyoshima K, Imai Y, Kaneko E, Shimokado K. hyperglycemic hyperosmolar non-ketotic coma. Nippon Ronen Igakkai Zasshi. 2007;44(6):756-60.

Address for Correspondence:
Dr Hamzullah Khan
Department of General Medicine,
PGMI, Lady Reading Hospital,
Peshawar – Pakistan.