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Original Research Article

A Study of Hospital Based Epidemiology, Role of Routine Biochemistry & Evaluation of Causes Underlying New Onset Seizures Presenting to the Emergency Department

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Abstract: The patients who present to the emergency with seizures are usually in a state of altered sensorium or comatose and are not able to tell about the preceding events i.e. (aura progressing to seizure activity). Hence this factor leads on to difficulty in assessing the etiology of seizures. The aim was to study hospital based epidemiology of new onset seizures presenting to the Emergency Department, to evaluate causes underlying new onset seizures and to evaluate role of routine biochemistry in establishing etiology of new onset seizures in emergency setting. In this study, all the patients above 12 years of age presenting to the emergency department with onset of new seizures, within 72 hours prior to presentation, were included for a period of 1½ years. It was concluded that seizures are one of the common presenting complaints in emergency department. Overall, maximum numbers of patients (50%) were less than 39 years of age and elderly people > 65 years of age contributed to only 13.6% of total patients studied. Neurocysticercosis and CNS infections are important causes of new onset seizures in developing countries like ours. Routine laboratory investigations play an important role in establishing etiology of new onset seizures in emergency setting.
Keywords: CNS Infections, Electrolyte Abnormalities, Epilepsy, Laboratory Investigations, Neurocysticercosis, Seizures

INTRODUCTION

Seizures are common presenting problem to emergency department. First time generalized seizures account for nearly 1% of all emergency department visits in the adult patients [1, 2]. As many as 1 in 20 of the general population will suffer a seizure at some point in their lifetime.

A single seizure is not synonymous with epilepsy, which is defined as recurrent unprovoked seizures. Seizures, which occur in emergency department are often provoked by some underlying acute disease process metabolic, infective or vascular and are called as acute symptomatic seizures. Such seizures are often provoked situation related seizure and occur in patients who don't have epilepsy [3].

Acute symptomatic seizures occur in close temporal relationship to a systemic or neurologic insult and occur as indirect or direct consequences of this insult.

For systemically ill patients with seizures, it is important to exclude a primary neurological cause. The etiology of first time seizures presenting to emergency department is a vexing problem and requires full clinical assessment.

The etiological profile of seizure presenting to emergency departments depends upon various factors. One of the most important factors in the etiology of new onset seizures is geographical profile. Incidence of various infections e.g. Neurocysticercosis is common in

certain locations and its incidence is more in tropical countries.

The etiological profile also depends upon the type of sample studied eg. Patients may suffer only a single episode of seizure (First seizure), or new onset epilepsy (incidental epilepsy) or chronic prevalent epilepsy. Any of the provoking factors that cause acute symptomatic seizures in individuals without epilepsy can also aggravate seizure in those with epilepsy.

The etiological profile of seizures also differs between patients presenting to emergency department and patients presenting to outpatient setting. The patients who present to emergency with seizures are usually in a state of altered sensorium or comatose and are not able to tell about the preceding events i.e. (aura progressing to seizure activity). Hence this factor also leads on to difficulty in assessing the etiology of seizures.

The patients might have suffered from similar episode in past which the attendants of patients may not be aware off. So there is a difficulty in classifying these patients into various epileptic syndromes. However patients who are seen in emergency setting as a case of new onset seizures are fully able to tell the preceding events before seizures and hence it helps to classify the type of seizures.

Because seizures have a profound psychological concern for the patient and family and also the further therapeutic decisions regarding treating these patients and for how long to treat these patients with anti-epileptic drugs depend upon etiological profile of new onset seizures.

Seizures are one of the common problems encountered in emergency setting. An individual lifetime risk of having a seizure is approximately 10%. Seizures account for almost 1% of emergency room visits and they prompt more concern and investigations than does many other conditions [1].

Seizures may be benign and self-limited and require little further treatment or they may be the initial sign of a serious medical illness or lead to status epilepticus. The evaluation of seizures in emergency department should include a sequence of steps aimed at identifying the etiology of the event.

When a patient presents to the emergency department with history of seizures, it is important to get an accurate description of the event. The first step is to determine whether the event was truly a seizure and which diagnostic studies are needed [4].

A proper history should be taken and seizures must be differentiated from confusional states, syncope, arrhythmias or pseudoseizures.

Patients in convulsive status epilepticus and those patients who are not actively convulsing but are persistently post-ictal require comprehensive diagnostic testing which includes determination of serum glucose, electrolytes, urea nitrogen, creatinine, magnesium, calcium, phosphate, CBC, LFT and drug of abuse screen. An arterial blood gas analysis in a convulsing patient may show an anion gap acidosis, which is usually secondary to lactic acidosis. ABG may provide information regarding hypercarbia and oxygenation.

The second step is to find out the underlying precipitant for seizures. Seizures may be manifestation of an underlying medical illness requiring specific treatment. The third step is to decide whether drug treatment is necessary. When a patient with new onset seizure comes in emergency, is usually seen by the attending physician or internist. There may be difficulty in describing the event leading on to seizure. Also a distinction is to be made between epileptic seizures and epileptic syndromes. Semiological seizure classification is based on ictal clinical semiology and stresses the differences between epileptic seizures and epileptic syndromes [5].

The role of neurologist in the evaluation and management of new onset seizures varies among different institutions. Often the internist first sees the patient and later on neurologist is called. Ictal seizure semiology is identified and then seizures are classified in various categories according to classification proposed by international league against epilepsy [6].

Very few studies have been done so far to address this problem and to study the etiological profile of new onset seizures presenting to the emergency department.

A prospective study [7] was conducted to determine the utility of extensive laboratory workup in patients presenting to emergency department with history of new onset seizures. The results of this study showed that with the exception of serum glucose, extensive laboratory workup done for the evaluation of new onset seizures is unnecessary.

Etiological profile of new onset seizures:

Seizures are common in patients with various systemic and neurological illnesses. These seizures are often precipitated by an acute stressor or precipitant and are called situation related seizures. Medical illness has a profound psychological and physical stress and seizures in these settings complicate the illness [3].

Seizures may be convulsive or nonconvulsive and may be focal or generalized or initially focal with secondary generalization. There are many systemic causes of seizures. Management of seizures in medically ill patients is aimed at correction of the underlying cause and use of appropriate short-term anticonvulsant medication. Various causes of seizures are –

A) Electrolyte abnormalities:

1) Derangement in sodium levels: Among the common metabolic abnormalities, disturbance in serum sodium concentration is a frequent finding in some patients. Although low serum levels are seen in hospitalized patients, hyponatraemia can be a feature of patients presenting in emergency with new onset seizures.

However neurological effects due to low serum Na levels are usually seen when concentration of serum sodium falls below 120 meq/l. Hyponatraemia produces brain oedema and increases intra-cranial pressure, which may lead to seizures and other neurological sequelae including death.

Hyponatraemia is a common complication of SIADH, head injury, intracranial tumours, and intracranial infections and can also be seen following trans-sphenoidal surgery for pituitary, and can be due to drugs. Hypernatraemia is defined as sodium level > 145mmol/L. It can cause various neurological manifestations. In non-hospitalized patients, hypernatraemia is seen mainly in elderly age groups and can be a manifestation of some infection in the body. In hospitalized patients. About 1% of these patients more than 60 years of age group have been found to be hypernatraemic. Hospital mortality ranges from approximately 40% to more than 60% in admitted patients with hypernatraemia [8].

2) Disturbances in calcium levels

Hypocalcemia is an important cause of seizures and symptomatic hypocalcemia is related with low ionized calcium levels. Hypocalcemia can cause various neurological manifestations like carpopedal spasm, perioral paraesthesias and tetany. It can also cause derangement in sensorium and seizures. Hypocalcemia may occur due to various causes like sepsis, acute renal failure, chronic renal failure, vitamin D deficiency and can cause seizures when there are very low levels of serum calcium.

Hypercalcemia also can cause seizures. Common neurological manifestations are deranged sensorium, psychiatric complaints, muscle weakness and seizures, either generalized or focal. Most common cause of hypercalcemia is primary hyperparathyroidism. Other causes include many types of malignancies, vitamin D intoxication etc. Acute seizures in hypercalcemia can be a part of parathyroid storm [9].

3) Disturbances in magnesium level

Hypomagnesaemia can cause cardiac arrhythmias, tetany and seizures. Many signs and symptoms of hypomagnesaemia are not specific and may be attributable to other electrolyte abnormalities [10].

Hypomagnesemia is defined as serum magnesium level of less than 1.2meq/l. Serum magnesium levels do not accurately reflect the tissue magnesium stores because less than 2%of total magnesium is extracellular. Hypomagnesemia is usually associated with other deranged metabolic abnormalities like hypocalcaemia and hypokalaemia. Hypomagnesemia is an uncommon but correctable cause of acute intractable cause of seizures in humans [11].

4) Disturbances in blood sugar levels

Both hyperglycaemia and hypoglycaemia are important causes of seizures. Hypoglycaemia can be due to several endocrinological disorders like hypothalamic-pituitary insufficiency causing growth hormone, thyroid hormone deficiency, ACTH deficiency, and is seen in diabetic population on oral hypoglycaemic agents like chlorpropamide which can cause prolonged hypoglycaemic episodes because of its long half life.

Seizures are the most common presenting neurological problem of hypoglycaemia at any age. Hypoglycaemic seizures usually occur at serum glucose concentration lower than 40 mg/dl. Acute starvation for 48-72 hours increases seizures threshold. Increased ketone body formation is believed to be responsible for the elevation in seizure threshold.

B) Organ failure

1) Renal failure: -

Acute renal failure is characterised by rapid decline in renal functions causing decline in glomerular filtration rate while symptoms of chronic renal failure are manifested when GFR falls below 25%. Various electrolyte abnormalities seen in renal failure are hyperkalaemia, hyponatraemia, acidosis and secondary hyperparathyroidism. All these can attribute to development of uraemic encephalopathy and seizures.

Clinical neurological features of uraemic encephalopathy typically relate to rate of development of renal failure rather than to degree of laboratory abnormality.

2) Hepatic failure: -

Patients with acute hepatic failure due to various causes like viral hepatitis, drug exposure, and toxin exposure can present with seizures. Acute hepatic failure is associated with changes in glucose metabolism, ammonia production and ability of the liver to remove metabolites and toxins from circulation. Progressive

encephalopathy results in cerebral oedema, which produces seizures. Seizures are a rare manifestation of hepatic encephalopathy [12].

C) Alcohol withdrawal seizures: -

Alcohol withdrawal is one the most important cause of adult new onset seizures. Alcohol withdrawal seizures are usually

- Generalized tonic clonic type
- Occur within 6 to 48 hours of cessation of alcohol
- Occur often in binge drinkers
- Occur abruptly without warning

However alcohol withdrawal seizures can occur as long as seven days afterwards. Alcohol acts as a CNS anaesthetic and has anticonvulsant properties. When blood concentration of alcohol is reduced, CNS of habituated alcoholics becomes excitable and produces signs of withdrawal. Alcohol may produce seizures by its partial or absolute withdrawal following a period of chronic intake by a combination of neurotoxic effects directly or its metabolites or associated metabolic conditions such as hypoglycaemia.

D) Eclampsia: -

Eclampsia is a common problem encountered in gynaecological practice and is characterised by hypertension, oedema and proteinuria. It may occur antepartum, intrapartum or postpartum. Eclampsia may present suddenly with seizures and may not always occur as a clinical continuum from pre-eclampsia. Maternal age less than 20 years is a strong risk factor for pre-eclampsia and eclampsia. Patients with eclampsia have CT scan findings of cortical white matter oedema, loss of sulci and cortical venous thrombosis.

E) Stroke: -

Cerebrovascular disease is a common cause of seizures in adults more than 50 years. Upto 10-15% of patients with acute stroke or TIA will present with a seizure and in some studies, twice that number will suffer a seizure within 24-48 hours after their initial insult [13].

Seizures can occur later after stroke and even after months or years and are due to result of structural brain lesions leading to development of an epileptic focus. The risk is higher in cases of venous sinus thrombosis e.g. cortical vein thrombosis and sagittal sinus thrombosis.

F) Intracranial hemorrhage

Seizures may complicate intracerebral haemorrhage in as many as 15-20% of cases. Sung et al [14] studied 1402 patients with intracerebral haemorrhage. Among these patients, seizures occurred in 64(4.6%) of patients. Seizures were the first manifestation of ICH in 19 patients. Status epilepticus occurred in 11(17%) of patients and it was the initial presentation of ICH in six (9%). The majority had simple partial seizures that were predominantly focal and motor.

G) Infections: -

Many bacterial, viral, fungal and parasitic infections can produce seizures. Cortical damage may accompany infections with a number of viruses including herpes, rubella, measles and human immunodeficiency virus. Cerebral malaria is very frequently accompanied by seizures. Acute seizures associated with CNS infection followed the patterns of CNS infections incidence [15]. The highest incidence occurred during the first year of life and in children aged <15 years: incidence is lower in adults.

1) Viral encephalitis: - Viral encephalitis is usually associated with development of new onset seizures. Various viruses found to be causing encephalitis include Herpes virus, West Nile virus and virus causing Japanese encephalitis.

2) Meningitis: - Both TB meningitis and pyogenic meningitis may be associated with seizures. Incidence of TB meningitis varies among different countries and is more in tropical countries. Patients usually present with headache, low-grade fever and altered sensorium. As disease progresses, patients may develop seizures and focal neurological deficits.

Pyogenic meningitis also may be associated with occurrence of seizures, however here also, seizures usually occur as a late manifestation. Inflammatory process involving small arteries and veins causing cortical damage and infarction in pyogenic meningitis may be responsible for focal neurological deficits and seizures in these patients [16].

3) Neuro cysticercosis: -Neuro cysticercosis is the most frequent parasitosis of the CNS in tropical countries. In Asia, the disease is widely distributed particularly in China, India and Thailand. Seizures are the most common manifestation of Neurocysticercosis.

Neuro cysticercosis has been suggested as the cause of many cases of epilepsy in Southern India as well, but disease is prevalent in northern and north-western regions of the country. Neurocysticercosis cases have also been reported in other countries as well.

Cerebral cysticercosis is the result of infestation of the brain with the larval stage of the intestinal tapeworm Taenia solium. The brain is a site of predilection for this infestation, where it results in meningeal racemose, parenchymal ventricular or mixed type of lesions [17].

In view of the above literature, we can see that very few studies have been done so far to study the

etiology of seizures in patients presenting to emergency department. Some of the studies have suggested routine biochemical investigations in these subsets of patients, while others have advised detailed laboratory workup in these patients. We undertook this study to evaluate the clinical and etiological profile of patients presenting to emergency departments with new onset seizures. So we undertook this study to do detailed clinical workup in patients presenting to emergency department with new onset seizures along with routine biochemical investigations in these patients.

AIMS AND OBJECTIVES

The aims and objectives of the study were

1) To study "hospital based epidemiology of new onset seizures" presenting to the Emergency Department.
2) To evaluate causes underlying new onset seizures presenting to the Emergency Department.
3) To evaluate role of routine biochemistry in establishing etiology of new onset seizures in emergency setting.

MATERIAL AND METHODS

In this study, all the emergency patients above 12 years of age presenting to the emergency department with onset of new seizures, within 72 hours prior to presentation, were included for a period of 1½ years.

All the patients were questioned about duration of onset of seizures, numbers of seizures and seizure semiology was noted as per Luders classification **[5].** An attempt was made to assign an electro-clinical syndrome to the seizures according to the 1989 ILAE classification when possible. Seizure clusters were noted within 24 hours, 48 hours and 72 hours and history regarding post ictal neurological deficit, psychosis and postictal headache was obtained.

Pertinent neurological examination was carried out noting the time of examination and the contributory history to find out the cause of seizures was taken. Patients were subjected to all routine investigations i.e. Haemogram, Urine R/E, Stool R/E, blood sugar, LFT, RFT, Serum calcium, serum phosphorus, serum magnesium and ABG. Patients were investigated about any medications taken and EEG was carried out when possible. CSF analysis was done in those seizures patients who had persistently altered mental status, infectious symptoms, elevated WBC count or fever.

Patients were subjected to CT scan head and other investigations, if indicated, were carried out based on clinical history and examination

Final diagnosis was made based on ictal, interictal seizure semiology, neurological examination, clinical history and electroclinical classification.

Any treatment given in the form of antiepileptics and other treatment given was noted.

Inclusion Criteria:

1. Age >12 years.
2. New onset seizures in 72 hours prior presentation to Emergency department.

Exclusion criteria

1. Patients having history of seizures of more than 72 hours prior to presentation in the Emergency department.
2. Diagnosis of non-epileptic seizures.

The patient cohort was analyzed for demographic trends, seizure semiology patterns, Electroclinical classification, aetiological diagnosis and EEG or imaging abnormalities. For purpose of analysis appropriate statistical tests were applied.

OBSERVATIONS

In this study, total of 110 consecutive patients with new onset seizures who presented to emergency department were included. Patients with age <12 years and with history of head trauma were excluded from the study. The study was conducted for period of one and half-years. The patients were subjected to routine biochemical investigations including renal function tests, liver function tests, computerized tomography scan head, magnetic resonance imaging brain (where ever needed) and electroencephalography (if possible).

Demographic profile:

Total number of patients who attended the emergency department during this period was 10659 and total number of patients with history of new onset seizures was 110. So 1.03% of total patients who came to emergency during this period had new onset seizures. Among 110 patients, 62 (56.34%) were males and 48 (43.6%) patients were females. Mean age of entire sample was found to be 41± 4 years (males 40 ± 5 years; females 40 ± 5 years).

Distribution of patients in different age groups

The patients were categorized in three different age groups (Table 1)

Table 1

Age Groups (in years)	Total Patients	Males	Females
15-39	55(50%)	29(26.3%)	26(23.6%)
40-64	40(36.3%)	26(23.6%)	14(12.7%)
≥ 65	15(13.6%)	7(6.3%)	8(7.2%)

We observed that about one half of all patients with new onset seizures were less than 39 years of age. 36.3% of patients were in age group of 40-64 and persons who were above 65 years of age contributed 13.6% of the patients.

Clinical profile:

Table 2

	GTC seizures	Focal Seizures	Focal seizures with gen.	Myoclonic seizures	Seizure clusters			Neurol. deficit	Encephalopathy
					Within 24 hours	Within 48 hours	Within 72 hours		
No. of patients	97(88.2%)	6(5.4%)	6(5.4%)	1(0.9%)	61(55.4%)	2(2%)	0%	13(12%)	47(43%)

We observed that GTC seizures were seen in 97(88.2%) of total patients. Focal seizures were seen in 6(5.4%) patients and focal seizures with secondary generalization were observed in 6(5.4%) patients. Seizure clusters within 24 hours were seen in 61(55.4%) of patients and within 48 hours in only 2(2%) patients. Neurological deficit was seen in 13(12%) patients and 47(43%) patients had encephalopathy.

Etiological diagnosis in patients who had seizure clusters

Table 3

Etiological diagnosis	Number of patients with seizure clusters	
	Within 24 hours	Within 48 hours
Unestablished	16(25.3%)	0
Neurocysticercosis	8(13.1%)	0
Acute infarct	6(10%)	0
Uraemic seizures	5(8.2%)	1
Alcohol withdrawal seizures	4(6.5%)	0
Presumed viral encephalitis	3(5%)	0
Post-partum eclampsia	3(5%)	1
Intracranial hemorrhage	2(3.3%)	0
Organo-chlorine poisoning	2(3.3%)	0
ADEM	2(3.3%)	0
Post- stroke seizures	2(3.3%)	0
Hyponatraemia	2(3.3%)	0
Venous thrombosis	1(1.6%)	0
Myoclonic seizures	1(1.6%)	0
OPC poisoning	1(1.6%)	0
Intracranial tumour extension	1(1.6%)	0
Hypernatremia	1(1.6%)	0
Drug withdrawal seizures	1(1.6%)	0
Total patients	**61**	**2**

Etiological diagnosis in patients with no seizure clusters

Table 4

Etiological diagnosis	**Number of patients**
Unestablished	11(23.4%)
Neurocysticercosis	6(12.7%)
Hyponatraemia	4(8.5%)
Acute infarct	3(6.4%
Hypernatremia	3(6.4%)
Presumed viral encephalitis	3(6.4%)
Uraemic seizures	2(4.2%)
Intracranial haemorrhage	2(4.2%)
Subarachnoid haemorrhage	2(4.2%)
Hypothyroid coma	1(2.1%)
Venous thrombosis	1(2.1%)
Hypertensive encephalopathy	1(2.1%)
Pyogenic meningitis	1(2.1%)
Tubercular meningitis	2(4.2%)
Hyperosmolar coma	1(2.1%)
Post stroke seizure	1(2.1%)
Hepatic encephalopathy	1(2.1%)
Hypocalcemia	1(2.1%)
Drug withdrawal seizures	1(2.1%)
Total patients	**47**

Table 3 and table 4 show grouping of patients according to presence of seizure clusters and etiological diagnosis in these patients. We noted that 63(57.2%) patients had seizure clusters. Out of these, 61 (97%) patients had seizure clusters in 24 hours and 2(3%) had seizure cluster in 48 hours. The cause of seizures was unestablished in 16(25.3%) of patients who had seizure clusters in 24hours. Neurocysticercosis accounted for 13.1% of these cases and seizure clusters were observed in 10% and 8.2% of patients with diagnosis of acute infarct and uraemic seizures respectively.

We also noted that 47 patients had no seizure clusters and presented with single episode of seizures. Out of these, cause was unestablished in 11(23.4%) patients. Neurocysticercosis was diagnosed in 12.7% of this category of patients. Hyponatraemia and hypernatraemia accounted for 8.5% and 6.4% of these cases respectively.

Etiological diagnosis in patients with neurological deficit

Table 5

Etiological diagnosis	**Number of patients**
Acute infarct	9(69.2%)
Intracranial hemorrhage	2(15.3%)
ADEM	2(15.3%)
Total patients	**13**

In our study total of 13 patients had neurological deficit. Out of these, 9(69.2%) had acute infarct, 2(15.3%) had ICH and 2(15.3%) had ADEM.

Etiological diagnosis in patients with encephalopathy

Table 6

Etiological diagnosis	Number of patients
Unestablished	14(30%)
Presumed viral encephalitis	5(10.6%)
Uraemic seizures	3(6.4%)
Hypernatremia	3(6.4%)
Hyponatraemia	2(4.2%)
Alcohol withdrawal seizures	2(4.2%)
Tubercular meningitis	2(4.2%)
Post-partum eclampsia	2(4.2%)
Organo- chlorine poisoning	2(4.2%)
OPC poisoning	1(2.1%)
Venous thrombosis	1(2.1%)
Subarachnoid haemorrhage	1(2.1%)
Tumour	1(2.1%)
Hyperosmolar coma	1(2.1%)
Hypocalcaemia	1(2.1%)
Hepatic encephalopathy	1(2.1%)
Drug withdrawal seizures	1(2.1%)
Hypothyroid coma	1(2.1%)
Hypertensive encephalopathy	1(2.1%)
Myoclonic seizures	1(2.1%)
Total patients	**47**

We noted that total of 47(43%) patients out of 110 had encephalopathy. The etiology of seizures was unestablished in 14(30%) of these patients. Five patients (10.6%) had viral encephalitis. Other causes of seizures in these patients were uraemia, alcohol withdrawal, hypernatraemia, hyponatraemia, poisonings, drug withdrawal and hepatic encephalopathy.

Diagnosis in patients with seizure clusters and encephalopathy

Table 7

Diagnosis	Number of patients
Unestablished	7(29.2%)
Viral encephalitis	3(12.5%)
Alcohol withdrawal seizures	2(8.3%)
Uraemic seizures	2(8.3%)
Post-partum eclampsia	2(8.3%)
Organo-chlorine poisoning	2(8.3%)
OPC poisoning	1(4.2%)
Post stroke seizures	1(4.2%)
Myoclonic seizures	1(4.2%)
Tumour	1(4.2%)
Hyponatraemia	1(4.2%)
Hypernatremia	1(4.2%)
Total patients	**24(2%)**

We noticed that 24 patients had seizure clusters and were in encephalopathy. The cause of seizures was unestablished in 7(29.2%) of these patients. Three (12.5%) patients had viral encephalitis and two had uraemic seizures. Other causes were organo-chlorine compound poisoning, post partum eclampsia, hyponatraemia and hypernatraemia

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Diagnosis in patients with seizure clusters without encephalopathy

Table 8

Etiological diagnosis	**Number of patients**
Unestablished	9(23.1%)
Neurocysticercosis	8(20.5%)
Acute infarct	6(15.4%)
Uraemic seizures	4(10.2%)
Alcohol withdrawal seizures	2(5.1%)
Intracranial haemorrhage	2(5.1%)
Post partum eclampsia	2(5.1%)
Post stroke seizures	1(2.5%)
Venous thrombosis	1(2.5%)
Drug withdrawal seizures	1(2.5%)
Hyponatraemia	1(2.5%)
ADEM	2(5.1%)
Total patients	**39(35.4%)**

We found thirty-nine cases with seizure clusters that did not had encephalopathy. The cause of seizures was unestablished in 9(23.1%) of these patients. 20.5% patients had neurocysticercosis, 15.4% had acute infarct and 5.1% patients had intracranial hemorrhage. Other causes were post stroke seizures, drug withdrawal seizures, hyponatraemia, venous thrombosis and ADEM.

Distribution of patients according to ILAE, 1989 classification

The patients were classified according to ILAE 1989 classification and in this study; distribution of patients in different categories of ILAE is shown in following table. (Table 9)

Table 9

ILAE, 1989 Category	**Number of patients**
4.13 (Acute symptomatic seizures)	79(71.8%)
4.12 (Isolated seizures)	28(25.4%)
1.22 (Remote symptomatic seizures)	3(2.7%)

Seventy-nine patients (71.8%) were categorized as acute symptomatic seizures and were placed in category ILAE, 4.13. Twenty-eight (25.4%) patients were grouped in category of isolated seizures as ILAE, 4.12. Only three (2.7%) patients were placed in category ILAE, 1.22 and were classified as remote symptomatic seizures. This group comprised of those subjects in whom a definite categorization according to 1989 ILAE classification was not possible. Although, customarily a single seizure does not amount to epilepsy according to definition, an attempt was made to assign ILAE category to cases presenting with single seizure.

Distribution of patients according to etiological diagnosis

The following table (Table 10) shows etiological diagnosis of patients and their relative frequencies along with male/female distribution. We observed that 14 patients were diagnosed as cases of neurocysticercosis, which constituted about 12.8% of total patients, included in the study. Acute infarct was seen in 9 (8.2%) patients. Other important causes were intracranial hemorrhage, uraemic seizures, alcohol withdrawal seizures, hyponatraemia and hypernatraemia. Post stroke seizures, which were classified as remote symptomatic seizures,

were seen in only three (2.7%) patients. The cause of seizures remained unestablished in 27(24.5%) patients.

Table 10

Acute Symp. Seizures	Etiological diagnosis	Total patients	Number of males	Number of females
1	Neurocysticercosis	14(12.7%)	10(71.5%)	4(28.5%)
2	Acute infarct	9(8.2%)	6(66.3%)	3(33.3%)
3	Intracranial hemorrhage	4(3.6%)	4(100%)	0
4	Subarachnoid hemorrhage	2(1.8%)	2(50%)	1(50%)
5	Presumed viral encephalitis	6(5.4%)	3(50%)	3(50%)
6	Alcohol withdrawal seizures	4(3.6%)	4(100%)	0
7	Uraemic seizures	8(7.2%)	4(50%)	4(50%)
8	Hyponatraemia	6(5.4%)	1(16.6%)	5(83.3%)
9	Hypernatremia	4(3.6%)	3(75%)	1(25%)
10	Postpartum eclampsia	4(3.6%)	0	4(100%)
11	TB meningitis	2(1.8%)	1(50%)	1(50%)
12	ADEM	2(1.8%)	0	2(100%)
13	OPC poisoning	1(0.9%)	1(100%)	0
14	Organo-chlorine poisoning	2(1.8%)	2(100%)	0
15	Hyperosmolar coma	1(0.9%)	0	1(100%)
16	HT encephalopathy	1(0.9%)	1(100%)	0
17	Drug withdrawal seizures	2(1.8%)	2(100%)	0
18	Hypothyroidism	1(0.9%)	0	1(100%)
19	Status myoclonus	1(0.9%)	0	1(100%)
20	Pyogenic meningitis	1(0.9%)	1(100%)	0
21	Venous thrombosis	2(1.8%)	1(50%)	1(50%)
22	Tumour extension	1(0.9%)	0	1(100%)
23	Hepatic encephalopathy	1(0.9%)	0	1(100%)
24	Hypocalcaemia	1(0.9%)	0	1(100%)
25	Unestablished	27(24.5%)	17(63%)	10(41%)
26 Rem. Symp. seizures	Post stroke seizures	3(2.7%)	1(33.3%)	2(66.3%)

Role of routine biochemistry

Table 11

Biochemical abnormality	Test performed in number of patients	Number of patients with abnormal result
Increased serum creatinine	108(98.2%)	8(7.4%)
Hyponatraemia	109(99.1%)	6(5.5%)
Hypernatremia	109(99.1%)	4(3.7%)
Hypocalcaemia	84(76.4%)	1(1.2%)
Total patients	110	19(17.3%)

Out of 110 patients, 19(17.3%) patients showed some form of metabolic abnormality. Serum creatinine levels were measured in 98.2% of patients. Out of these, 7.4% had raised levels of creatinine. Sodium levels were done in 109 patients and 10(9.17%) patients had deranged serum sodium levels. Hyponatraemia was observed in 6(5.5%) patients and hypernatraemia in 4(3.7%) patients. Serum calcium levels were measured in 84(76.4%) patients and only one (1.2%) patient had hypocalcaemia.

DISCUSSION

In the present study, we included 110 consecutive patients with history of new-onset seizures who presented to emergency department. We excluded patients who were less then 12 years of age and also had seizures in more than 72 hours prior to admission and with diagnosis of non-epileptic seizures. Patients with history of head trauma were also excluded from the study.

Frequency of seizures in the emergency department:

Seizures are one of the common presenting complaints in emergency department. Previous literature shows that seizures account for 1 to 2% of emergency department visits. Krumholz *et al.*[1] noted that seizures specifically accounted for nearly 1% of all ED visits.

Ullman *et al.* [2] found that seizures or probable epilepsy are responsible for 1-2% of all non-trauma visits to urban hospitals. These studies however did not differentiate between new onset seizures and seizures due to chronic epilepsy per se.

In our study new onset seizures contributed 1.03% of total emergency visits, a figure that is consistent with published literature. The profile of first seizures and seizures presentation to the emergency department in context of known chronic epilepsy is likely to be different. Patients with pre- existent epilepsy are less likely to attend the emergency department in event of seizures. We sought to specifically study the profile of new onset seizures

Demographics:

In our study, mean age of entire sample was 41 + 4 years. Overall, maximum number of patients (50%) was between ages of 15-39 years, 40% patients were in age group of 40-64 years and only 13.6% patients were above 65 years of age. A community based study by Annegers *et al.* [18] showed incidence rate of acute symptomatic seizures increased with age and was highest in age group of > 75 yrs.

However in our study, most of the patients were in age group of 15-39 years. This difference in age distribution may be due to referral characteristics in our hospital and the fact that the study by Annegers *et al.* [18] was a community-based study while ours is a facility based. It is likely that in the Indian community, elderly patients with seizures are not brought to hospital.

Krumholz *et al.* [1] in their hospital based study reported that 31% of total 200 patients were in age group of 30-44 and 16% of patients were in age group of 19-29. So results of our study are consistent with that study.

Clinical profile:

Patients with new onset seizures may present with generalized or focal seizures. Krumholz *et al.* [1] reported GTC seizures in 61.5% of patients studied and other type of seizures including partial or absence seizures in 8.5% of patients.

Sempere *et al.* [19**]** in a prospective study of 98 consecutive patients with new onset seizures found GTC seizures in 68.4% of patients, focal with secondary generalization in 19.4% of patients, simple partial in 10.2% of patients and complex partial in 2% of patients.

Baumhacl *et al.* **[**20] in a prospective study of 327 adult patients with first epileptic seizures reported GTC seizures in 75.5% of patients, focal seizures in 11.6% of patients and focal with secondary generalization in 11% of patients.

In our study, 88.2% of patients with new onset seizures had GTC seizures, 5.4% had focal seizures, 5.4% had focal with secondary generalization and one patient presented with myoclonic seizures. We noted seizure clusters within 24 hours in 61(55.4%) and of patients and within 48 hours in 2(2%) of patients. 13(12%) patients had neurological deficit. These patients with neurological deficit had either acute infarct or intracranial haemorrhage or ADEM. We noticed seizure clusters in 63 patients. Twenty-four patients had seizure clusters with encephalopathy and causes of seizures in this category of patients included viral encephalitis, alcohol withdrawal seizures, uraemic seizures and poisonings by organo-chlorine and organophosphorus compounds. Thirty-nine patients had seizure clusters without encephalopathy. We noticed eight patients with neurocysticercosis, six patients with acute infarct and four patients with uraemic seizures in this subset of patients.

Syndromic Classification:

In our study, we attempted to categorize our patients according to guidelines provided by ILAE, 1989 and placed our patients in different categories of epileptic syndromes.

Eighty patients (72.3%) were grouped in ILAE category 4.13 of acute symptomatic seizures, 27 patients (24.5%) in ILAE category 4.12 of isolated seizures and 3 patients (27%) in ILAE category 1.22 of remote symptomatic seizures. Patients with neurocysticercosis were placed in category of acute symptomatic seizures.

However Murthy and Yangala [21] grouped 8% of patients in ILAE category 4.1 and 92% in ILAE category 1.2 in a study conducted on patients with acute symptomatic seizures. However that study was conducted on both in-patients and outpatients in a hospital.

Etiological profile:

Etiological spectrum of new-onset seizures varies among different population groups. Acute symptomatic seizures occur due to some underlying medical problem causing acute CNS insult while remote symptomatic seizures occur in patients with past history of trauma, stroke or some other structural abnormality in brain. Previous literature has reported alcohol withdrawal and cerebral infarction as two leading causes of new onset seizures. The cause of seizures was established in our study in 82(74.5%) patients and remained unestablished in 25.5% of patients.

Sempere *et al.* [19] could find cause of seizures in 72.4% of patients with new onset seizures and Ramirez-Lassepas *et al.* [22] were able to find etiology of seizures in 48% of patients.

Table 12

Etiology	**Sempere *et al.*[19]; (prospective study**	**Krumholz *et al.*[1]; (hospital based study)**	**Henneman *et al.*[23]; (retrospective study)**	**Baumhacl *et al.*[20]; (prospective study)**	**Our study (prospective study)**
Idiopathic	27(27.5%)	28(14%)			
Unestablished	0	102(51%)	146(44%)		27(24.5%)
Cerebral infarction	23(23.4%)		38(11%)	48(14.7%)	9(8.2%)
Neurocysticercosis	0		41(12%)		14(12.7%)
Febrile seizures		30(15%)			
Alcohol related	11(11.2%)	13(6.5%)		119(36.3%)	4(3.6%)
CNS infection	9(9.1%)		10(3%)		8(7.2%)
Tumour	8(8.1%)		22(7%)	32(9.8%)	1(0.9%)
Trauma	4(4%)			26(7.9%)	
Drug toxicity	3(3%)				
Poisonings	0				3(2.7%)
Hyperglycaemia	2(2%)				
Hypocalcemia	0				1(0.9%)
Uraemia	1(1%)				8(7.2%)
Hyponatraemia	1(1%)				6(5.4%)
Hypernatremia	0				4(3.6%)
Post-partum eclampsia	0				4(3.6%)
Gelegenheitsanfalle	0			17(5.2%)	
Others	9((9.2%)	27(13.5%)	76(23.2%)	85(26%)	21(19.1%)
Total	98	200	333	327	110

Table 12 is showing etiology of new onset seizures and their relative frequencies as reported by various authors in comparison to our study results. Krumholz *et al.* [1] accounted 51% of total patients in their study with unknown etiology and 6.5% as cases of alcohol withdrawal.

Sempere *et al.* [19] reported that among 98 patients with new onset seizures, 23 (23.4%) patients had stroke, 11 (11.2%) patients had alcohol withdrawals and 9 (9.1%) patients had CNS infection.

Baumhacl *et al.* [20] evaluated 327 adult patients with a first epileptic seizure in respect to underlying causes. In 119 patients (36.3%), seizures were alcohol induced and in 48 patients (14.6%), vascular pathology predominated.

Our study reported neuro cysticercosis in 14 (12.7%) of patients, acute infarct in 9 (8.2%), presumed viral encephalitis in 5 (4.5%) patients and alcohol withdrawal seizures in 3.6% of patients. So our study results are not in consistence with earlier studies in which alcohol withdrawal and infarction were attributed are most common etiologies of new onset seizures. These differences may be due to different geographical location of our country where infections are commonly seen in our population.

Role of routine laboratory investigations:

When a patient comes to emergency with new onset seizures, he is usually subjected to routine laboratory investigations. There are no established guidelines for performing these investigations and only few studies have addressed this problem.

Ramirez *et al.;* [22] in their study on 184 patients for evaluation of first seizures reported metabolic abnormality in 16 (11%) patients.

Henneman *et al.* [23] retrospectively reviewed 333 adult patients with new onset seizures. He reported that 21 patients (6%) had some metabolic abnormality in form of deranged electrolytes, creatinine, glucose and he also reported 2 cases (1%) having abnormal calcium leads.

Sempere *et al.* [19] found abnormal biochemical tests in four patients out of total of 98 patients studied. These included two cases with hyperglycaemia, one with hyponatraemia and one with uraemia.

In our study, 19 patients (17.3%) showed some form of metabolic derangement. Among these 10 patients (9.1%) had abnormal sodium levels, six with hyponatraemia and four with hypernatraemia. Raised serum creatinine was found in 8 (17.4%) patients and only one patient (1.2%) had hypocalcaemia.

In conclusion, it appears from published literature that routine biochemical investigations contribute significantly to the tune of 5-10% of patients with seizures in emergency department. However our study showed some sort of metabolic abnormality either in serum sodium levels, creatinine or calcium levels in 17.3% of patients, which is relatively higher than results of above-mentioned studies. The higher proportion of abnormal results may be due to the fact that ours is a medical based emergency department rather than neurology based ED. In conclusion, we recommend that these biochemical investigations should be done routinely in patients presenting with new onset seizures in emergency department.

SUMMARY AND CONCLUSIONS

We included 110 consecutive patients with history of new onset seizures who attended emergency department.

- 1.03% of total patients who came to emergency during this period had new onset seizures.
- Overall, maximum numbers of patients (50%) were less than 39 years of age and elderly people > 65 years of age contributed to only 13.6% of total patients studied.
- Seventy-nine (71.8%) patients were diagnosed to have acute symptomatic seizures and were placed in ILAE category 4.13 and three patients were placed in ILAE category of remote symptomatic seizures.
- The cause of seizures was established in 82(74.5%) patients and remained unestablished in 28 (25.5%) patients.
- Fourteen (12.7%) patients were diagnosed to have neurocysticercosis. Other important causes were acute infarct, uremia, hyponatraemia, hypernatraemia, and viral encephalitis, and post-partum eclampsia, pyogenic and tubercular meningitis.
- Alcohol withdrawal seizures were seen in only 4(3.6%) patients.
- Metabolic derangements were seen in 19(17.3%) patients. We found no case of hypoglycemia as cause of new onset seizures.
- Seizure clusters were seen in patients with diagnosis of neurocysticercosis, acute infarct, uraemia, viral encephalitis and post partum eclampsia.
- No patient with neurocysticercosis was found to be in encephalopathy.

CONCLUSIONS

- Seizures are one of common presenting complaints in emergency department.
- Neurocysticercosis and CNS infections are important causes of new onset seizures in patients with new onset seizures in developing countries like ours.
- Routine laboratory investigations play an important role in establishing etiology of new onset seizures in emergency setting.

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