*A Dissertation on*

# "THE CLINICAL AND ELECTROENCEPHALOGRAPHIC PROFILE OF SEIZURES IN PATIENTS WITH ALCOHOL DEPENDENCE"

*Submitted to*

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# CERTIFICATE

This is to certify that the dissertation entitled **"The clinical and electroencephalographic profile of seizures in patients with alcohol dependence**" is a genuine work done by Dr. P.Vijayashankar for the partial fulfilment of the requirements for D.M. (Neurology), examination of the **The Tamilnadu Dr.M.G.R. Medical University** to be held in August 2014, under the able guidance and supervision of **Prof. Dr.S.GOBINATHAN, M.D.,** D.M.,(Neurology), Professor and Head, Department of Neurology, Government Stanley Medical College and Hospital, Chennai.

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# DECLARATION

I, **Dr.P.Vijayashankar,** Solemnly declare that the dissertation **"The clinical and electroencephalographic profile of seizures in patients with alcohol dependence,**" is a bonafide work done by me during the period of January 2012 to January 2014 at the Government Stanley Medical College and Hospital, Chennai under the expert supervision of **Prof. Dr. S. GOBINATHAN., M.D, D.M., (Neurology)**, Professor and Head, Department of Neurology, Government Stanley Medical College and Hospital, Chennai.

This thesis is submitted to **The Tamilnadu Dr.M.G.R. Medical University** in partial fulfilment of the requirements for D.M. (Neurology), examination to be held in August 2014

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Date:

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# CONTENTS

# The study of the clinico-electroencephalographic profile of seizures in patients with alcohol dependence

## Introduction

In 400 BC Hippocrates described “alcohol overuse cause seizures”.[1] In the Roman period the epilepsy was called as “Morbus Convivialis” a disorder related to feast.[2] In 1851swedish physician Huss named the diseases including seizures caused by chronic alcohol consumption as “alcoholismus chronicus”.[3]

Alcohol is used in almost all the countries as a social drink. The prevalence of alcohol abuse is more common in developed countries.[4,5] Now a days there is a increasing trend was seen in developing countries also. In the United States and Australia 80% of men and 60% of women consumes alcohol at some point of time in their lifetime.[4]

Alcohol abuse was one of the five most important risk factors causing global burden of disease and disability was associated with epilepsy.[6]

Epilepsy is one of the most common neurologic presentations. Worldwide, there are 50 million people living with epilepsy and most of them (80%) were living in developing countries.[9] An annual incidence

of 40 -70 per 1,00,000 people were in industrialized countries.[7] Epilepsy contributed 0.5% of the global disease burden and more than 7 million disability adjusted life years [8.]

The incidence of unprovoked seizures was 370 per 1, 00,000 in Iceland and 800 per 1,00,000 in Poland .[7] The most common age for first independent drinking without a family member is about 15 years. It varies among countries and depends up on the culture, but it has not changed much in decades. Heavy drinking was commonly observed in the adolescent age group of 18 years to 22 years.

Alcohol withdrawal seizures occur most frequently within 6 -48 hours of abstinence from alcohol. The recurrent alcohol withdrawal seizures reduces the threshold for seizures by kindling effect.

Seizures were very common in the alcohol withdrawal state. Hence seizures in an alcoholic were usually considered as alcohol withdrawal seizures. Recent literatures suggest that the seizures in alcoholics were not only due to alcohol withdrawal, it may be due to alcohol dependence. It can be easily differentiated by a detailed drinking history, clinical examination and to do the necessary supportive investigations such as EEG, MRI Brain to evaluate the cause of the seizures and treat the patient appropriately.

Alcohol related seizures can be associated due to various diseases involving central nervous system such as

**Infections:**

-neurosyphilis

-HIV

-Meningitis

-Cerebral abscess.

**Metabolic causes:**

-Hypoglycemia

**Toxins**

**-Trauma**

-Intracranial hematoma

-Subarachnoid hemorrhage

**-Cerebro vascular accident**

**Neoplasm**

## Pathophysiology of alcohol related seizures

Acute alcohol intoxication with the steadily increasing serum alcohol level could also precipitate the seizures due to its excitatory effects.[8] Chronic alcohol consumption gradually produces tolerance and dependence for alcohol. Hence, alcohol was considered as having both anticonvulsive effect and proconvulsive effect.[9] When this balance of anticonvulsive effect is tilted by the alcohol withdrawal they mostly go for proconvulsive state which is more pronounced during alcohol dependence but it is not provoked by binge of drinking for a minimal duration or by a small quantity of alcohol. So, Alcohol related seizure is an indicator for alcohol dependence

## Alcohol drinking pattern

Alcohol use was classified as the following types

1) Healthy drinking

2) Alcohol dependence

## Standard healthy drinking

8gm of ethanol in United Kingdom and about 10 gm in U.S.A was considered as standard healthy drinking.

## Hazardous drinking

Any individual who consumes more than 3 or more standard drink per day is considered as hazardous drinking.

## Alcohol dependence

### ICD 10 DCR criteria for alcohol dependence

A definite diagnosis of dependence should usually be made only if three or more of the following have been present together at some time during the previous year:

(a) A strong desire or sense of compulsion to take the substance.

(b) Difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use.

(c) A physiological withdrawal state when substance use has ceased or been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms.

(d) Evidence of tolerance, such that increased doses of the psychoactive Substances are required in order to achieve effects originally produced by lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill non tolerant users).

(e) Progressive neglect of alternative pleasures or interests because of Psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects.

(f) Persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm.

**Binge drinking**

Binge drinking is characterized as the rapid increase in blood alcohol concentration (BAC) to 0.08 gram percent or above. Binge drinking is defined as acute intake of alcohol within 2 hours. It may be 4 or more drinks for adult women and 5 or more drinks for adult men.

A “drink” refers to half an ounce of alcohol (e.g., one 12-oz. Beer, one 5-oz. Glass of wine, or one 1.5-oz. Shot of distilled spirits)

**Alcohol metabolism**

2-10% of the ingested alcohol was excreted via the lungs, urine and sweat. The rest is metabolized as acetaldehyde by the enzyme alcohol dehydrogenase (ADH). It was rapidly converted to carbon dioxide and water by the enzyme aldehyde dehydrogenase (ALDH).

The genetic variations in the enzyme alcohol dehydrogenase - ADH (ADH1B*2 and ADH1C*1) rapidly breaks down alcohol and produces acetaldehyde. 40% of Asian ethnic people such as Japanese, Koreans and Chinese have ALDH2*2 mutations which produce more acetaldehyde than normally, it has to produce with a regular drink. [9,10]

## Laboratory evaluation

Liver function tests such as Gamma glutamyltransferase, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST) were markers of heavy drinking. The elevation of these enzymes indicates regular heavy alcohol consumption such as 5 or more standard drinks/day for 5 days or more. These enzymes return to normal baseline when alcohol was stopped for several weeks. It can also be considered for monitor the adherence to treatment.

Gamma glutamyltransferase is present in the endoplasmic reticulum and the bile duct epithelial cells. It is important for amino acid transport. It has a specificity of 60% in men and 50% in women.

The serum aminotransferases are a sensitive indicator of liver injury and can be helpful in identifying heavy drinking. It has a sensitivity of 25-45% and specificity of 90% for alcohol intake. [12]

## Electroencephalography

Electroencephalography is utilized to differentiate between epilepsy syndrome and symptomatic seizures in alcohol dependence seizures. The electroencephalography is frequently normal in most of the alcohol dependence seizures patients. If it is abnormal it is

suggestive of a epilepsy syndrome. The important post ictal EEG pattern was Normal Low amplitude.[13] A dose dependent relative reduction in the 24 HZ photic response was also observed. [14]

With the above investigations imaging also helps to rule out infections, stroke, neoplasm, Trauma and other causes.

**Follow up**

All the patients after registration were followed up as outpatient regularly at an interval of every three months and their drug compliance were monitored.

# AIM OF THE STUDY

To study the clinical and electroencephalographic profile of seizures in patients with alcohol dependence.

# REVIEW OF LITERATURE

Ten million people were living in India were having epilepsy. The prevalence rate was about 1% in Our Indian population.[15] The prevalence rate is more in the rural population (1.9%) than it was compared to urban population (0.6%).[16,17] The Urban Rural Neuro-epidemiological Survey (BURNS), which was conducted in Bangalore revealed that the prevalence rate of epilepsy was 8.8/1000 people in urban community and it was 11.9/1000 people in rural communities. It indicated that the epilepsy was two times more prevalent in rural areas than the urban areas.[18]

According to W.H.O 2005, there are 88% of adults consumed spirits, 10% consumed beer, 2% consumed wine. There is an increasing trend in the alcohol consumption comparing to previous years.[20]

Various Indian studies showed that the overall prevalence of epilepsy in India is 5.59-10 per 1000. The prevalence ratio of epilepsy in Kerala is 4.7 per 1000. The prevalence rate in males was (5.1 per 1000) was higher than that of females (2.2 per 100).[15,18,19]

**N-Methyl –D-aspartate**

Chronic alcohol consumption enhances the binding of glutamate with N-methyl –D-aspartate. Snell et al observed that chronic consumption of alcohol enhances the NMDA receptor subunit protein -NR1 in the hippocampus and cerebellum. It also enhances the NMDA receptor subunit protein -NR2A in the hippocampus and cortex.[21] Kalluri et al observed that the 2B subunit was involved in the up regulation of the NR1 &NR2A subunits. During acute alcohol intoxication, the above mentioned receptors were inhibited and during alcohol withdrawal rebound excitations occur.[22]

**Gamma -Aminobutyric acid (GABA)**

Alcohol enhances the effect of GABA, specifically the α4- subunit and the δ subunit. The α4subunit has a differential response due to small GABA response, increased synaptic and less extra synaptic receptors.[23]

Chronic adaptations occur in NMDA and GABA receptors on during alcohol intoxication and hyper excitation of these receptors during alcohol withdrawal. Soyka et al in his study postulated that

α2 subunit gene was involved in alcohol dependence and also with alcohol withdrawal.[24]

The frequency of alcohol dependent seizure is fairly related to the dose and the duration. In the study conducted by Ng et al showed that the people who consumed 50 -100gm/ day of alcohol had a 2.8 % risk of seizures. Those who drink 101-200 g /day and more than 200 gram/day had 7.9% and 19.5% risk of seizures.[24] Leone et al also observed a similar percentage of risk in his study. He observed 3.0% risk in patients who consumed 50-100gm/day of alcohol. 7.9% and 16.6% risk of seizures in patients consumed 101-200 gm/day and in more than 200 gm/day of alcohol. In both the studies the non-drinkers had very low risk (1%) of seizures.[25]

Sandeep et al evaluated 100 consecutive patients presented with seizures related to alcohol intake, he observed that the mean age was 43.7 years and the median age was 45 years. The mean duration of alcohol intake was 17 years. Their mean daily intake of alcohol in the previous one month duration was 280gms and the mean alcohol consumption in the bout before seizures were 398 grams. They also observed that the average number of seizures were 2.26, 22% had

clusters of seizures. 8% of the first degree relatives of the patients had seizures.[26]

Pratima Murthy et al evaluated 381 consecutive male patients with alcohol dependents observed that mean age was 37.5 years and mean age of initiation of alcohol intake was 22.1 years. They had an average daily alcohol consumption of 17. The daily alcohol consumption in the previous month prior to seizures was 50%. The mean onset of first seizures was 33 years. Seizure onset prior to alcohol use was 3%. Median number of seizures was 3%. They also observed 59% of their patients had tonic clonic seizures, 4% had partial seizures, 1% had status epilepticus, 11% had clusters of seizures. 22% had a family history of alcohol dependence and 7% had alcohol dependence and seizures.[27]

Bajaj et al in his study of 320 patients with alcohol dependence observed 120 (37.5%) patients had seizures. Mean age was 35.5 years, mean age of onset of alcohol consumption was 25.25 years, 90% had consumed alcohol daily and mean age of onset of first seizures was28 years, 5% of patients had status epilepticus. He also observed that 25% of patients had a family history of alcohol dependence and 10% had a

family history of seizures. 75% of patients MRI brain showed cortical atrophy and cerebellar atrophy was found in 33% of patients.[28]

Niels K. Rathlev et al in his 105 patients presented to the emergency department after six hours of administration of placebo who had alcohol related seizures observed that patients who had an ethanol level higher than 100 mg/dl didn't have recurrent seizures. 36% of his patients who had a level lower than 100 mg/ ml had recurrent seizures. Alcohol is the important causative factor in 15 to 24% of patients admitted with status epilepticus. Victor and Brausch observed that 85% of alcohol related seizure patients developed their first and last seizure within 6 hours duration.[29]

Morton et al and DaryananiHE, et al also reported in their study that a previous incidence of alcohol related seizures are a strong predictor of further future events. [30,31]

Andriy V. Samokhvalov et al in his meta-analysis observed that consistent alcohol consumption was associated with epilepsy / unprovoked seizures. He also observed that the consumption of daily alcohol has a significant dose dependent response relationship with the onset of probable epilepsy. Those who are consuming an average of

four, six and eight drinks per day have a significant relative risk compared to non-drinkers.[32]

Leone et al evaluated 237 first seizures patients and observed that the risk of first generalized seizures were higher in alcoholic men than in non-alcoholics. The author observed an odds ratio, 6.8%; a confidence limit was 95% in men and in women it was 6.8%, 1.6 to 32.6. The odds ratio in his patients who consumed an average alcohol of 1 -25gm/day was 1.2 (0.8 to1. 8). It was 3.0 (1.7 to 5.4) in those who consumed alcohol of 51-100gm/day, 7.9 (2.9 to 21.9) in those consumed alcohol of 101 to 200gm/day and 16.6 (1.9 to 373.4) for those consumed more than 200 mg/day. He found that chronic alcohol consumption is a risk factor for developing first generalized epilepsy in both male and females over 15 years.[33]

Brathen et al observed that 12% of epilepsy outpatients had harmful and hazardous consumption of alcohol and 35% of patients were admitted with seizures in the emergency department. He also observed that 51% of his patients were presented with partial onset seizures with secondary generalization. Sandeep et al observed that 88% of his patients had generalized seizures and 12% of patients had partial seizures.[34]

Murthy et al observed that 18% of their patients had Clusters of seizures. Sandeep et al observed that 22% of his patients had clusters of seizures.[26,27]

Pilke A et al, Lowenstein et al, Leppik IE et al observed that the incidence of status epilepticus varies between 10-24 % of patients with alcohol abuse. Avdaloff et al observed that repeated seizures and status epilepticus are frequent encountered in alcohol dependent patients particularly in the late onset patients.[36]

Neils. K. Rathlev observed that 50% of seizures in alcohol dependent patients were not due to alcohol withdrawal related. 15.7% of patients were having the idiopathic seizure disorder. They were also presented with various etiologies such as significant head trauma in 25.7%, cerebro vascular accident in 5.7%, non traumatic intracranial lesion in 3.6% toxic and metabolic abnormalities in 2.9% of patients.[29]

Bajaj et al observed that 80% of his patients had an elevated Liver function tests. Murthy et al observed that the liver function tests were elevated in all the patients in her series.[28]

Coutin and churchman observed that 8.6% of his patients had normal EEG. 42.4% of patient had decreased power in theta and delta

waves and increase in beta waves. 17.2% had reduction only in theta and delta waves. 15.2% increase in beta waves, 14.7% had reduction in slow waves and alpha waves, 3.1% had reduction in alpha waves. They speculated that reduction in slow waves may be due to cerebral atrophy and increase in beta waves may be due to antiepileptic drugs.[37]

Victor and Brausch observed that photo paroxysmal EEG responses and photo-myoclonic responses during alcohol withdrawal.[38]

T. sand et al in his study observed that a definitely abnormal EEG suggests epilepsy or symptomatic seizures which were unrelated to alcohol. The predictive value of a normal EEG is limited, but the typical post-ictal finding in alcohol related seizures is nevertheless a normal low-amplitude EEG record.[39]

Prathima Murthy et al in her study of 381 patients observed 25% of patients had a family history of seizures. In his series Sandeep et al observed that 8% of patients with alcohol related seizures had a family history of seizures. Schumen et al in his 289 patients observed an odds ratio of 2.45 in the relatives of alcohol induced seizures. He suggested that the genetic predisposition could be the cause for the family history of seizures. [26,27]

Dam et al observed that 74% of patients with a history of long term alcohol use with seizures were having cortical atrophy due to chronic alcohol intake.[40] Sandeep et al observed a 27% of cortical atrophy in the C.T scan in his series. Bajaj et al observed 75% of patients with alcohol induced seizures had cortical atrophy, which was predominantly involved the frontal, parietal and temporal regions. Carlen et al observed in his patients that nonspecific cortical atrophy was observed in alcoholics who have consumed alcohol more than ten years.[26,28] kriel et al in his patients observed that the frontal lobe was predominantly involved secondary to alcohol consumption.

The predictors for first alcohol related seizures were previous history of such events, prior detoxification admissions and clinical institute withdrawal score of more than 15. Earlier studies have shown that the alcohol abuse for more than 10 years has a higher risk of developing alcohol related seizures. The Seizure incidence was not documented in less than 5 years of alcohol abuse patients. A prior history of alcohol related seizures is a strong predictor of future episodes.[41]

## PATHOPHYSIOLOGY

### Genetic causes

Recent advances in genetic analysis have shown us that multiple genes were associated with alcohol abuse.40 - 60 % of the risk for developing Alcohol related disorders was explained by its genetic association and other environmental factors.

1) Lower risk for alcohol related disorders was associated with enzymes that metabolize alcohol, which increases the sensitivity to alcohol. A variant of aldehyde dehydrogenase (ALDH2*2 allele) was associated with aversion to alcohol.

2) Polymorphisms associated with variations in the receptors of Gamma- amino butyric acid (GABRA2), acetylcholine (CHRM2), and dopamine (DRD2) were associated with impulsivity, disinhibition and vulnerability to alcohol in people with type 2 and type B personality.

3) Some people were associated with low response to alcohol. Hence, they will drink more alcohol to obtain the adequate desired response, which leads to alcohol abuse. Serotonin transporter (SLC6A4), potassium channels (KCNMA1), Gamma anion butyric acid variations (GABRA6), second messenger systems (AC9), genes

affecting glutamate receptors were associated with this reduced alcohol response.[12]

Alcohol withdrawal seizures occur most frequently within 6 -48 hours of abstinence from alcohol. The recurrent alcohol withdrawal seizures reduces the threshold for seizures by kindling effect.

**Acute effects**

After ingestion of alcohol the binding of glutamate increases avidly binds to N-methyl-D-aspartate (NMDA) receptors and increases the Gamma amino butyric acid (GABA) effects particularly effect of its delta subunit. These sub units were distributed in the cerebellum; cortex, thalamic circuits, and the brain stem are the specific centres which mediate the intoxicating effects of alcohol.

**Chronic effects**

The chronic consumption of alcohol produces tolerance and dependence towards alcohol. This adaptive effect of the central nervous system was temporary phenomenon; once alcohol intake was stopped this effect will disappear. The prolonged consumption of alcohol increases the NMDA proteins with tonic inhibition of these receptors. It leads to rebound activation during alcohol withdrawal. The

consumption of alcohol potentiates the excitotoxic effect of the glutamate, aspartate and homocysteine in the circulation. During the alcohol withdrawal, it increases the homocysteine level and increases the seizure risk.[42]

**Treatment**

Treatment for alcohol abusers is always difficult due to poor drug compliance, drug overuse and the interaction between drug and alcohol. Few studies have reported that Carbamazepine, sodium valproate, gabapentin ad gabapentin reduces the alcohol intake. The newer AED Topiramate reduces the craving for alcohol. Prophylactic antiepileptic drugs should be offered to the patients who were having seizures that were not related to alcohol consumption. Status epilepticus due to alcoholism has a better prognosis. It is also a predictor for subsequent epilepsy. Injection Lorazepam was the drug of choice for alcohol induced status epilepticus. Diazepam is the alternative for Lorazepam.[9,43]

# MATERIALS AND METHODS

**Study Design** **:** Prospective study

**Setting** **:** Govt. Stanley Medical College and Hospital, Chennai

**Study Period** **:** January 2012 to January 2014.

**Sample size** **:** 44 patients.

**Study Population** : Alcohol dependence patients who had seizures admitted/referred to the Neurology Department, Govt.Stanley Medical College and Hospital

All Patients with seizures in patients with alcohol dependence

**Inclusion criteria**

1) All patients who fulfil the criteria of ICD-10 DCR for alcohol dependence and have developed seizures.

**Exclusion criteria**

Other substance abuse

CNS infections

Road traffic accident / head injuries.

Seizures due to tuberculoma and other granulomas.

Encephalopathy due to other causes

# METHODOLOGY

A prospective study of patients with alcohol dependence who developed seizures. Demographic and clinical data of these patients were collected. Detailed history, sleep EEG, MRI Brain, electrolytes, random blood sugar, liver function test were done for these patients. Cerebrospinal fluid analysis was done for patients who developed fever.

Patients with alcohol dependence who developed seizures were included in the study. Demographic details of the patients were collected and the results were analyzed.

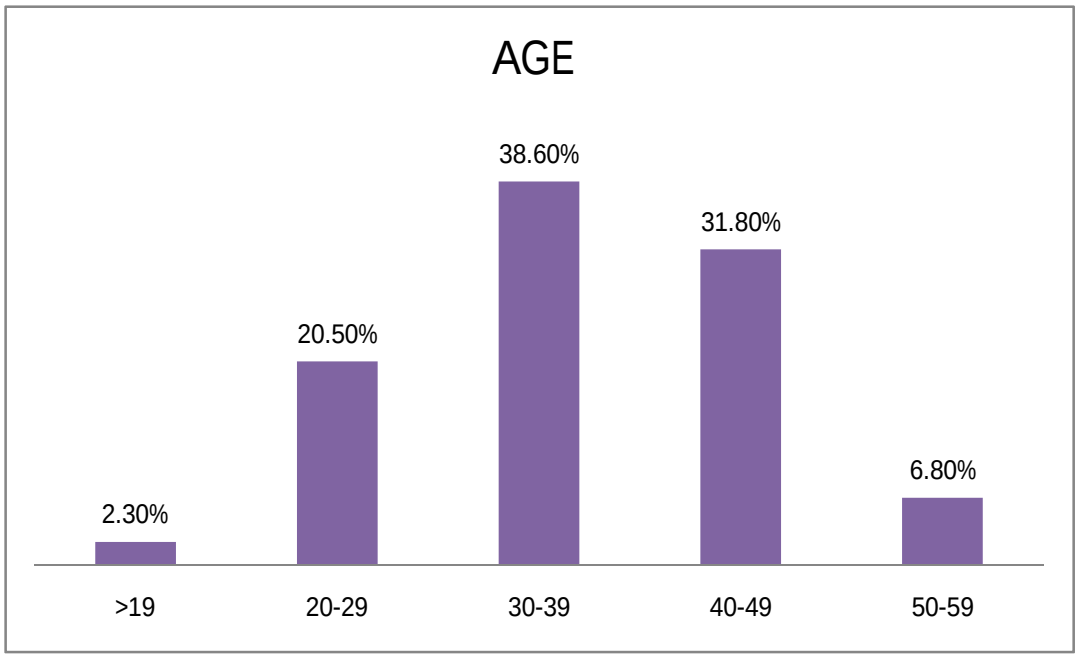
# OBSERVATION AND RESULTS

The total number of patients included in the study were forty four in number.

## Age group

The youngest patient's age was 19 years and the oldest patient's age was 57 years. The mean age of the patients was 35.65 and the median age was 35. One Patient (2.27%) was between 10 and 19 years of age, 9 patients (20.45%) were between 20 and 29 years of age, 17 patients (38.63%) were between 30 and 39 years of age, 14 patients (31.81%) were between 40 and 49 years of age, 3 patients (6.81%) were between 50 and 59 years of age.

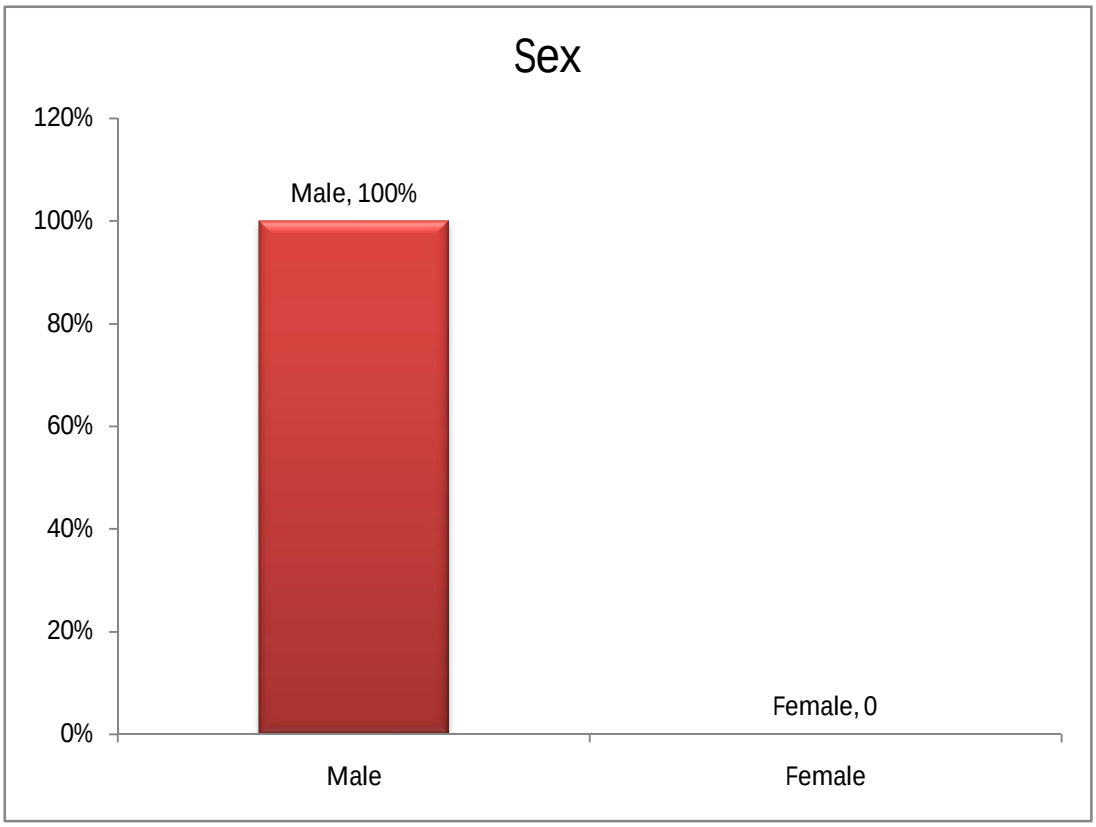
| Age of the patient | Number of patients (%) |
|---|---|
| 10 - 19 years | 1 (2.27) |
| 20 - 29 years | 9 (20.45) |
| 30 – 39 years | 17 (38.63) |
| 40 -49 years | 14 (31.81) |
| 50 – 59 years | 3 (6.81) |




**The diagram indicates alcohol dependence seizures affected the most productive age group of patients.**

**Sex**

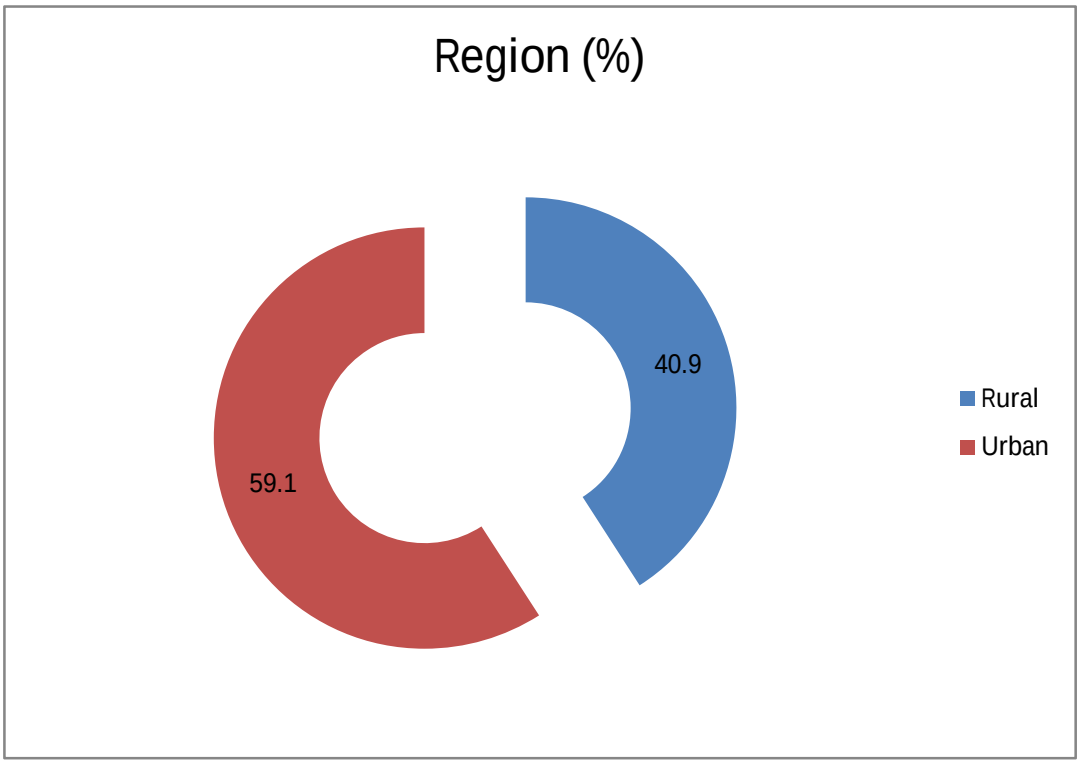
In this study, all patients were males.



**Residence**

In our study group, 26 (59.10%) patients were from urban area and 18 (40.90 %) patients were from rural area.

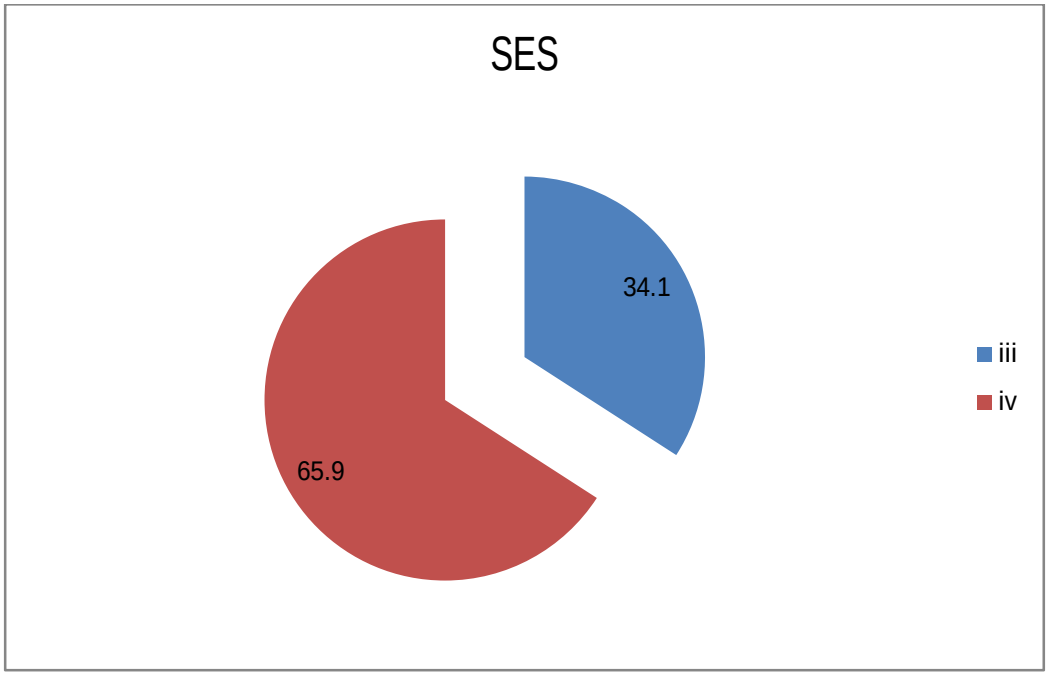
| Residence | No of patients |
|---|---|
| **Urban** | 26 (59.10) |
| **Rural** | 18 (40.90) |



**Socioeconomic status**

As per updated Kuppuswamy's scale (2007)15 (65.9 %) the patients belong to class IV socioeconomic status and 29 (34.1 %) the patients belong to class III socioeconomic status in our study.

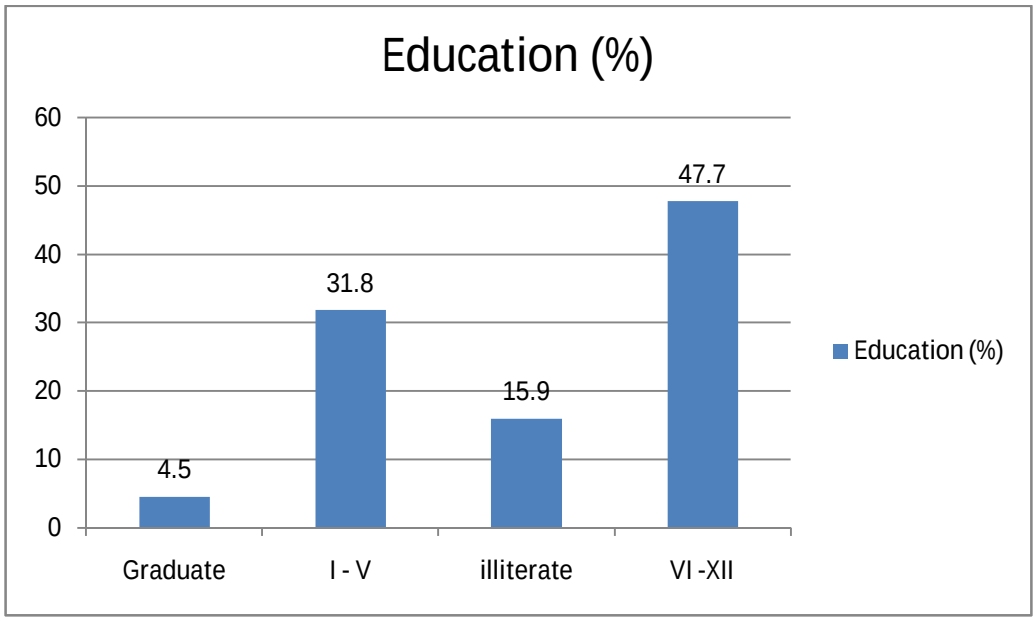
| Socioeconomic Status: | Number of patients (%) |
|---|---|
| Class iii | 15 (34.1%) |
| Class iv | 29 (65.9%) |



## Literacy

In our study population 7 (15.90 %) patient did not know to read or write,14 (31.81 %) patients completed their primary school, 21 (47.72 %) patients completed VI-XII standard, 2 (4.54 %) patients graduated.

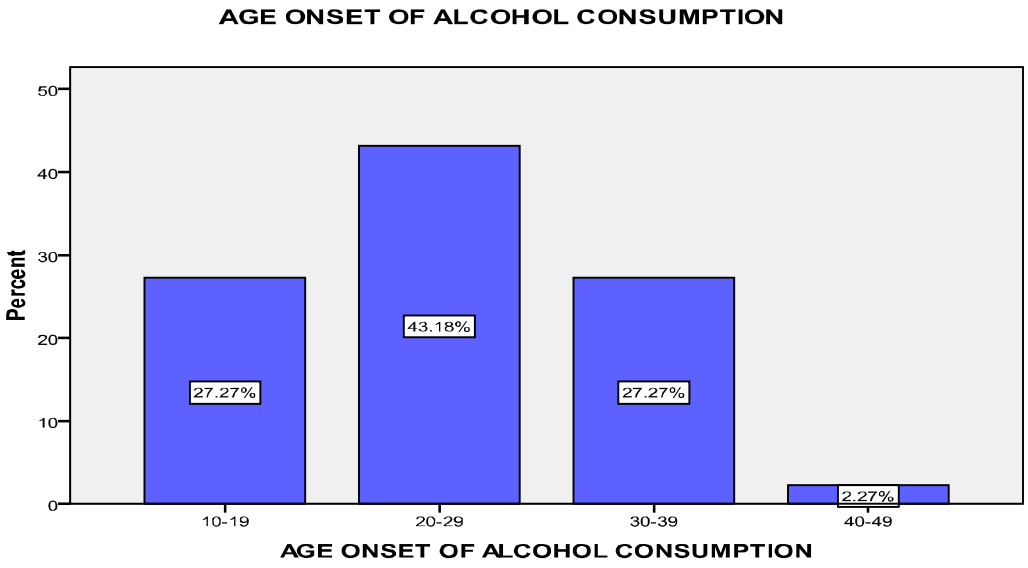
| Literacy | Number of patients (%) |
|---|---|
| Illiterate | 7 (15.90) |
| I – V STD | 14 (31.81) |
| VI-XII STD | 21 (47.72) |
| Graduated | 2 (4.54) |



## Age of onset of alcohol consumption

In our study Twelve (27.3 %) patients started consumption of alcohol between 10 and 19 years of age, 19 (43.2 %) patients started consumption of alcohol at the age between 20 and 19 years of age, 12 (27.3 %) patients started consumption of alcohol at the age between 30 and 39 years of age 1 (2.3 %) patient started consumption of alcohol between 40 and 49 years of age.

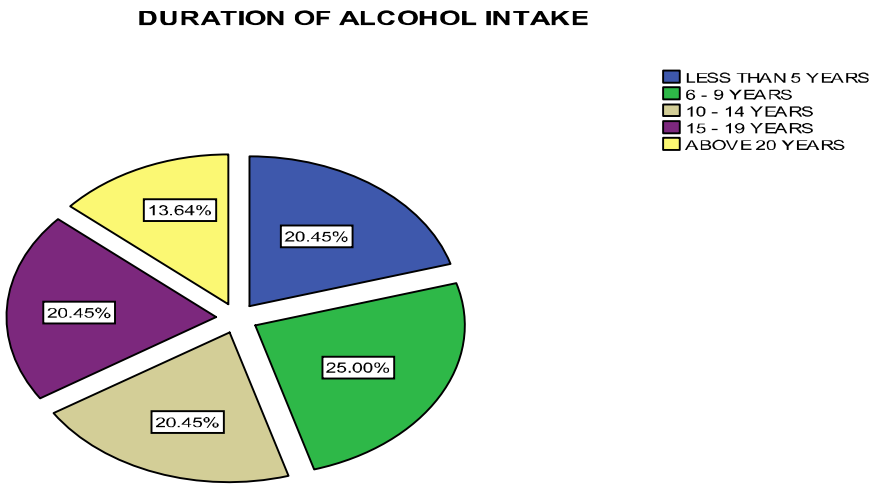
| Age of onset of alcohol consumption | Number of patients (%) |
|---|---|
| 10 -19 years | 12 (27.3) |
| 20 -29 years | 19 (43.2) |
| 30 -39 years | 12 (27.3) |
| 40 -49 years | 1 (2.3) |



**Duration of alcohol intake**

In our study 9 (20.5 %) patients consumed alcohol for less than 5 years, 11 (25.0 %) patients consumed alcohol between 6 to 10 years, 9 (20.5 %) patients consumed alcohol between 11 and 15 years, 9 (20.5 %) patients consumed alcohol between 16 and 20 years, 6 (13.6 %) patients consumed alcohol for more than 20 years.

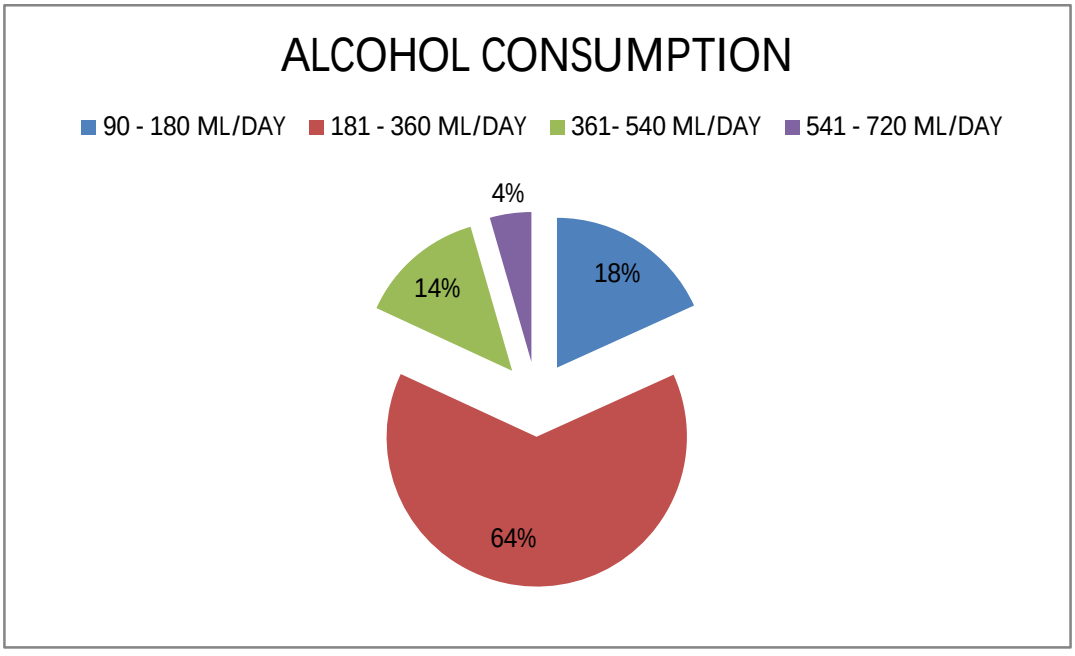
| Duration of alcohol intake | Number of patients (%) |
|---|---|
| Less than 5 years | 9 (20.5) |
| 6 - 10 years | 11 (25.0) |
| 11 -15 years | 9 (20.5) |
| 16 -20 years | 9 (20.5) |
| Above 20 years | 6 (13.6) |



**Quantity of alcohol consumption**

In our study 8 (18.2 %) patients consumed 90-180 ml/day, 28( 63.6%) consumed 181-360 ml/day, 6 (13.6%) patients consumed 361-540 ml/day, 2 (4.5%) patients consumed 541-720 ml/day.

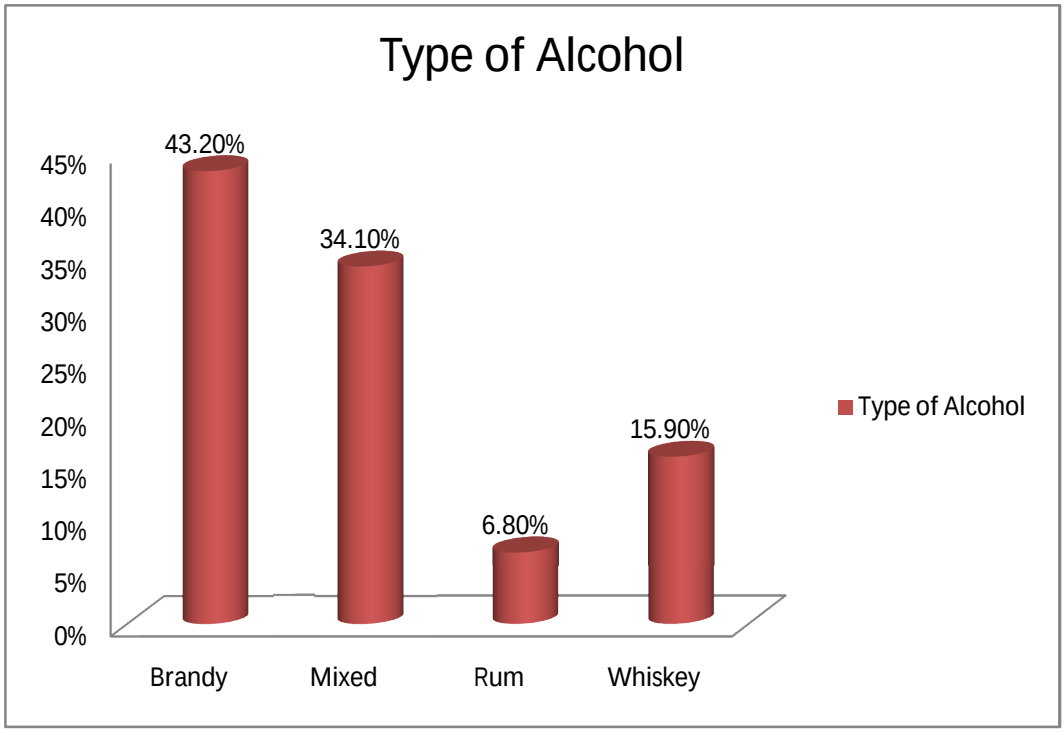
| Quantity of alcohol consumption | Number of patients (%) |
|---|---|
| 90 - 180 ml/day | 8 (18.2) |
| 181 ml - 360 ml/day | 28 (63.6) |
| 361 - 540 ml/day | 6 (13.6) |
| 541 - 720 ml/day | 2 (4.5) |



**Type of alcohol consumption**

In our study, 19 (43.2%) patients consumed brandy, 7 (15.9%) patients consumed whisky, 3(6.1%) patients consumed rum, 15 (34.1 %) patients consumed mixed alcoholic beverages.

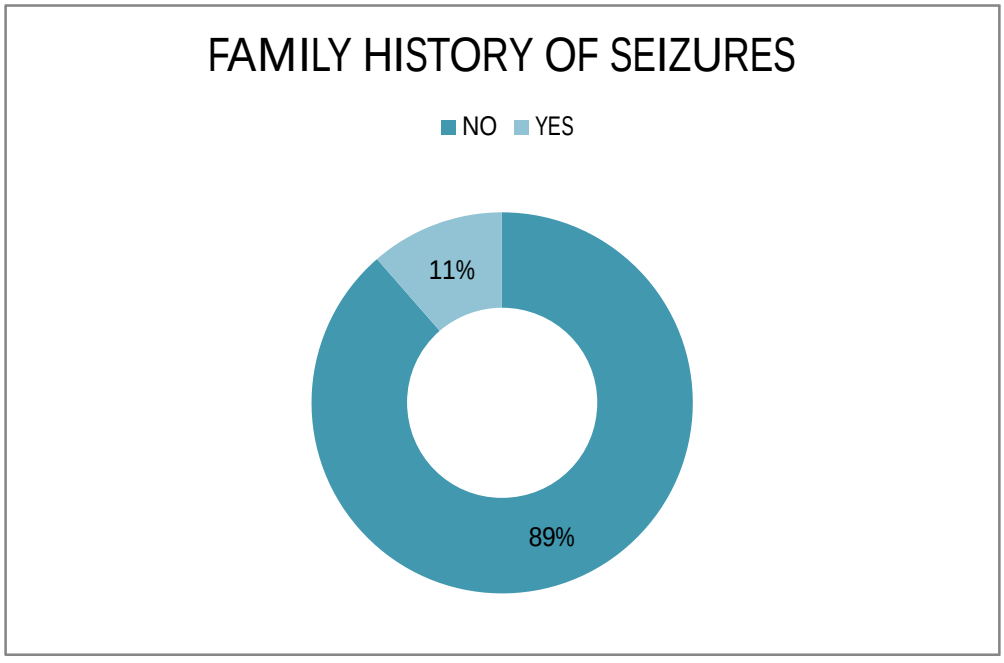
| Type of alcohol consumption | Number of patients (%) |
|---|---|
| Brandy | 19 (43.2) |
| Whisky | 7 (15.9) |
| Rum | 3 (6.1) |
| Mixed alcoholic beverages | 15 (34.1) |



**Family history of seizures**

In our study, 5 (11.4 %) patients had a family history of seizures. 39 (88.6 %) patients did not have family history of seizures.

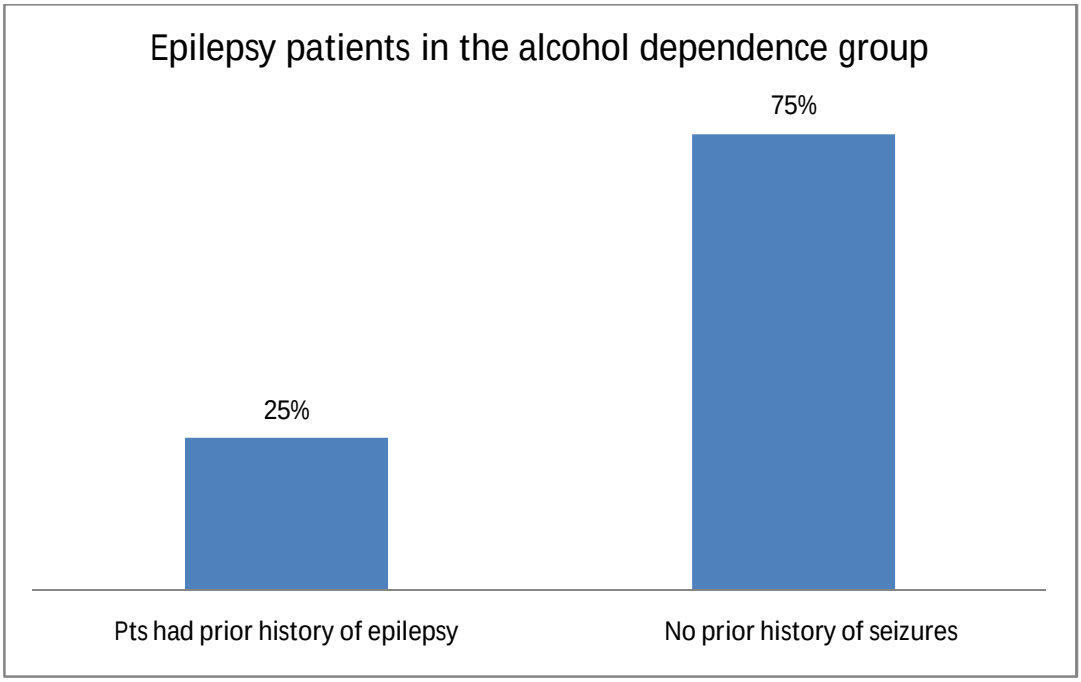
| **Family history of seizures** | **Number of patients (%)** |
|---|---|
| Family history of seizures | 5 (11.4) |
| No family history of seizures | 39 (88.6) |



**Epilepsy patients in the alcohol dependence group**

In our study, 11 (25%) patients were diagnosed to have epilepsy prior to alcohol consumption and 33 (75%) patients did not have seizures prior to alcohol consumption.

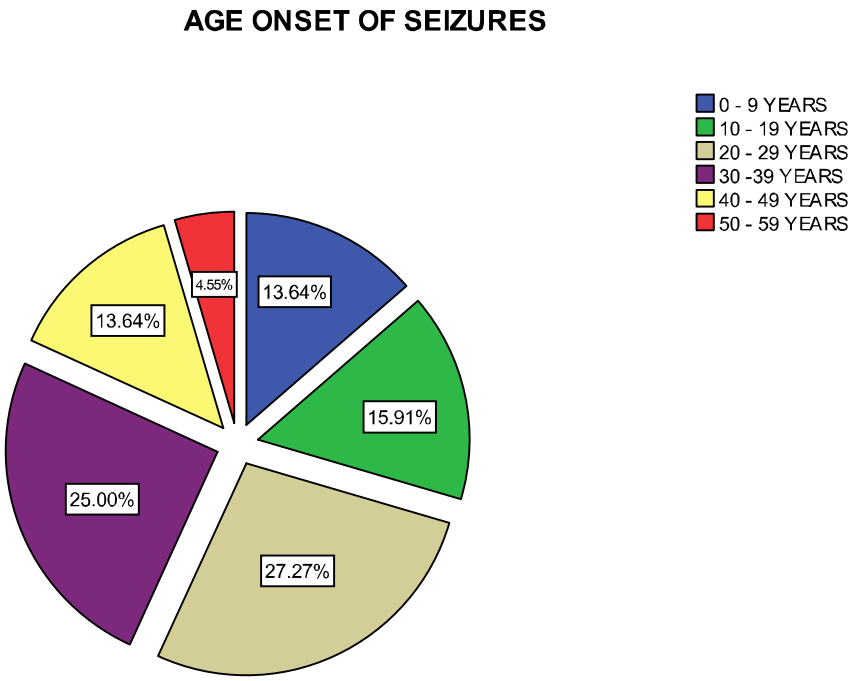
| Prior history of epilepsy | Number of patients (%) |
|---|---|
| Pts had prior history of epilepsy | 11 (25) |
| No prior history of seizures | 33 (75) |



**Age of onset of seizures**

In our study, 6 (13.6%) patients had onset of seizures between 0 and 9 years of age 7 (15.9) patients had onset of seizures between 10 and 19 years of age, 12 (27.3) patients had onset of seizures between the age of 20 and 29 years, 11 (25 %) patients had onset of seizures between the age of 30 and 39 years, 6 (13.6) % patients had onset of seizures between the age of 40 and 49 years, 2(4.5) patients had onset of seizures between the age of 50-59 years.

| **Age of onset of seizures** | **Number of patients (%)** |
|---|---|
| 0 – 9 years | 6 (13.6) |
| 10 - 19 years | 7 (15.9) |
| 20 -29 years | 12 (27.3) |
| 30 -39 years | 11 (25.0) |
| 40 -49 years | 6 (13.6) |
| 50 -59 years | 2(4.5) |



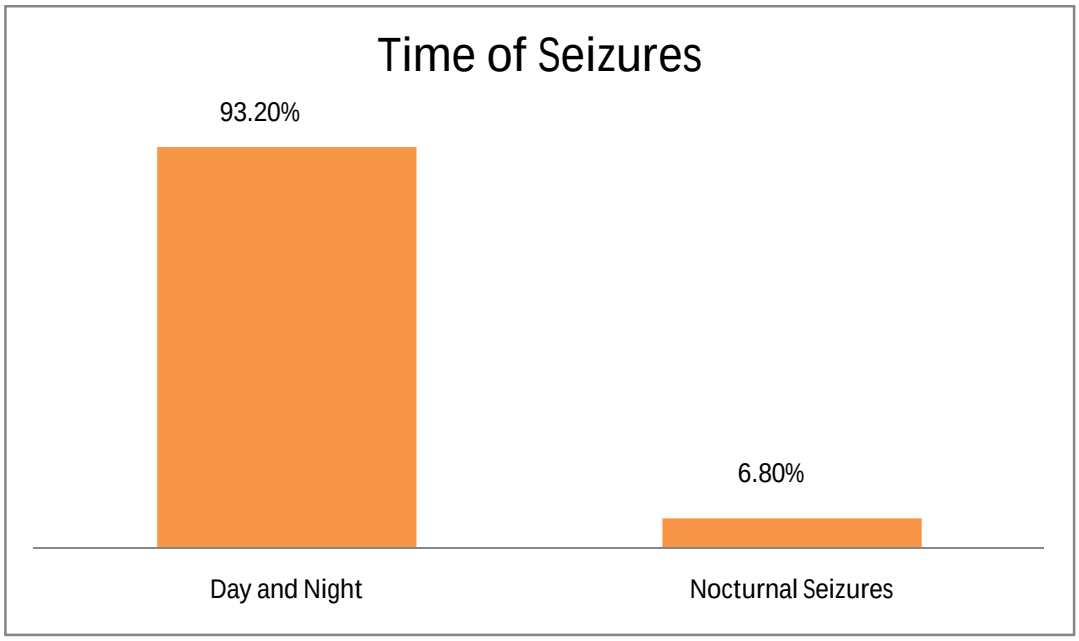

## Neurological deficits

No patients had prior Neurological deficits in our study.

**Habitual seizure characteristics**

In our study, 6 (13.63 %) patients had cluster of seizures, 3 (6.82 %) patients had nocturnal seizures and 41 (93.18 %) patients had both day and night time seizures.

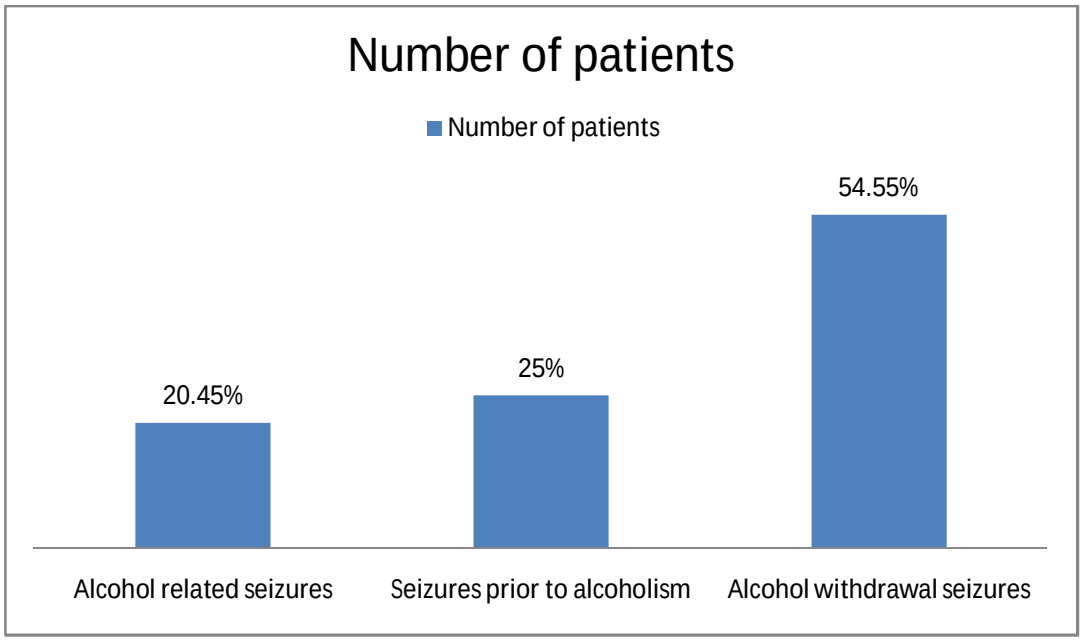
| Seizures Characters | Number of Patients (%) |
|---|---|
| Clusters | 6 (13.63) |
| Nocturnal Seizures | 3( 6.82) |
| Day and night time Seizures | 41 (93.18) |



**Alcohol dependent seizues**

In our alcohol dependent seizure patients, 9 (20.45%) patients had epilepsy prior to alcohol consumption, 11 (25%) patients had alcohol withdrawal seizures, 24(54.55%) patients had alcohol related seizures (excluding the alcohol withdrawal group).

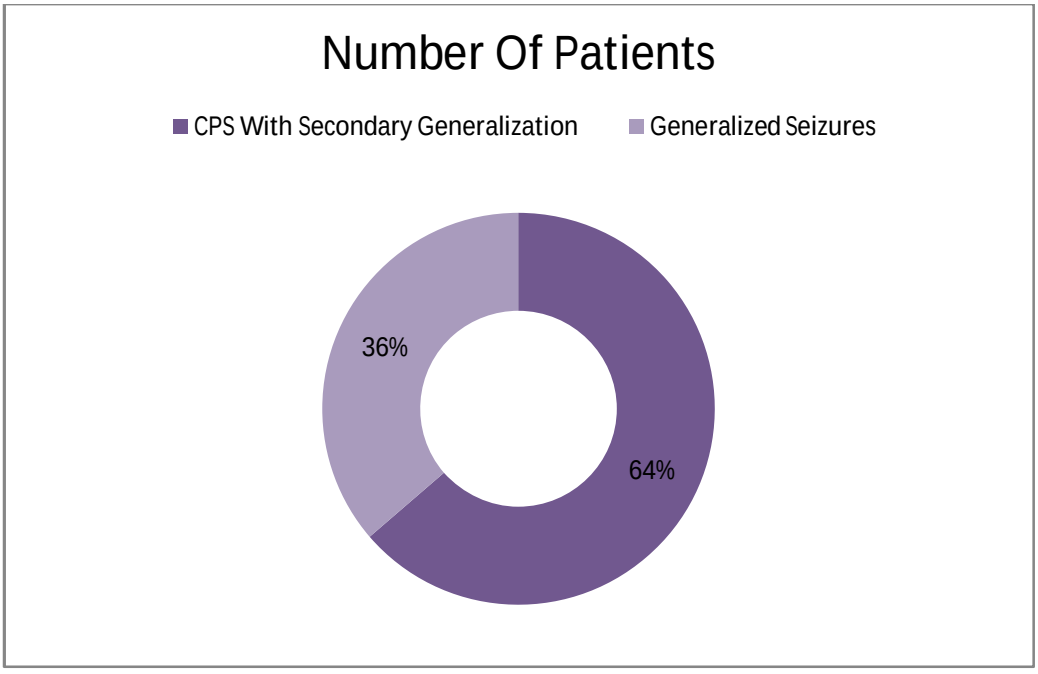
| Alcohol dependent seizues | Number of patients (%) |
|---|---|
| Alcohol related seizures | 9 (20.45) |
| Seizures prior to alcoholism | 11 (25) |
| Alcohol withdrawal seizures | 24 (54.55) |



**Type of seizures in epilepsy patients with alcohol dependence**

In our study, 7 (63.7%) patients had complex partial seizures with secondary generalization. 4 (36.3 %) patients had generalized seizures.

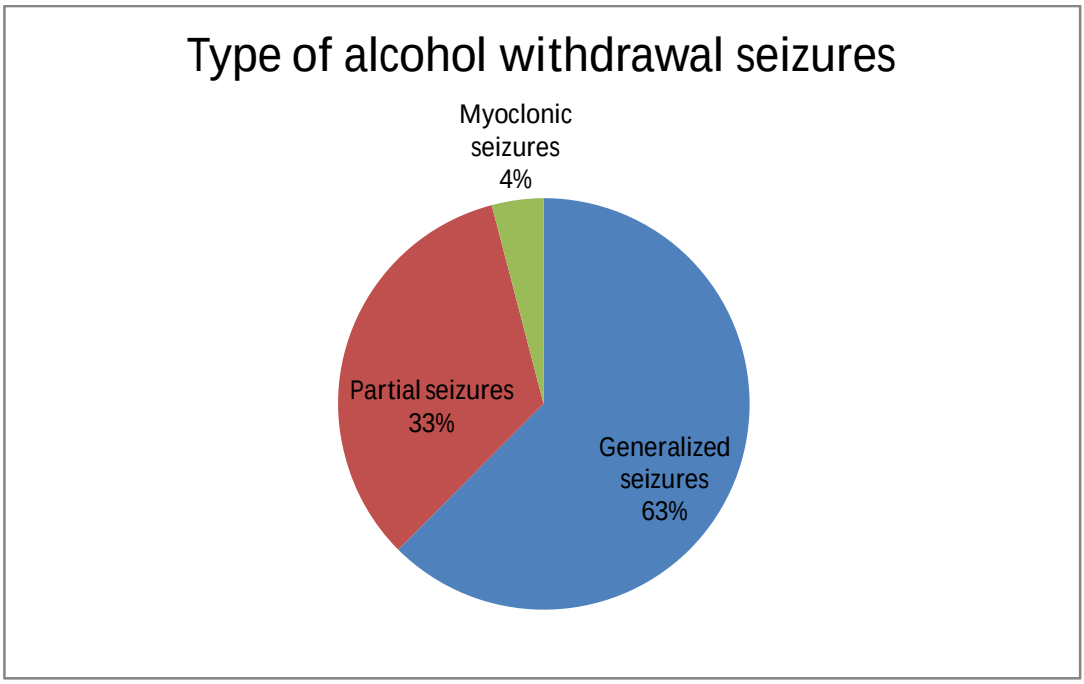
| **Type of seizures in epilepsy patients with alcohol dependence** | **Number of Patients (%) (N=11)** |
|---|---|
| CPS With Secondary Generalization | 7 (63.7) |
| Generalized Seizures | 4 (36.3) |



**Type of Alcohol withdrawal seizure**

In our patients with alcohol withdrawal seizures, 15 (62.5%) patients had generalized seizures, 8 (33.4%) patients had complex partial seizures with secondary generalization and 1 (4.1%) patient had myoclonic seizures.

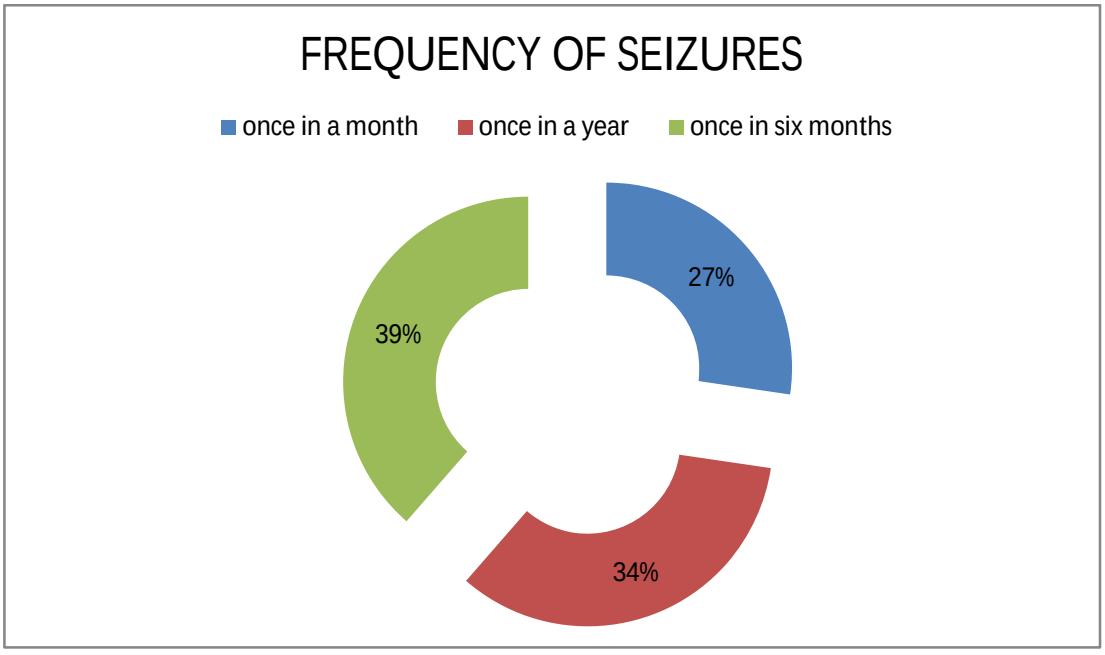
| Type of Alcohol withdrawal seizures | Number of patients (%) (N=24) |
|---|---|
| Generalized seizures | 15 (62.5) |
| Partial seizures | 8 (33.4) |
| Myoclonic seizures | 1 (4.1) |



**Frequency of seizures**

In our study, 12 (27.27 %) patients had at least one episode of seizure per month, 17 (38.63 %) patients had at least one episode of seizure every 6 months, 6 (13.63 %) patients had at least 1 episode of seizure every year.

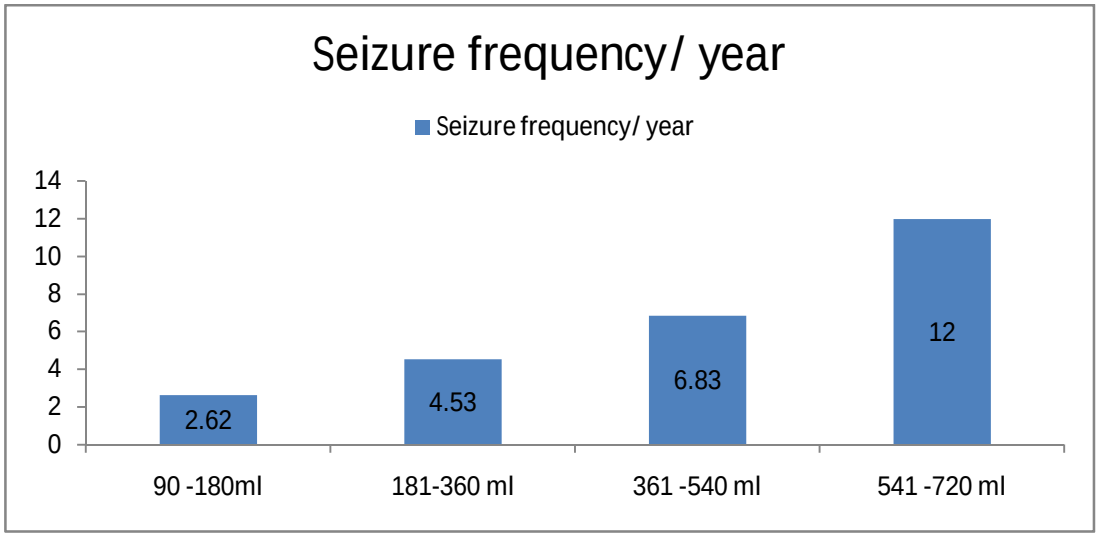
| Frequency Of Seizures | Number Of Patients (%) |
|---|---|
| At Least Once Per Month | 12 (27.27) |
| At Least Once In 6 Months | 17 (38.63) |
| At Least Once In A Year | 6 (13.63) |
| Alcohol related new onset seizures | 9 (20.45) |



**Quantity of alcohol consumption and frequency of seizures**

The mean frequency of seizures is 2.62/ year in patients who consumed alcohol between 90- 180 ml/day, the mean frequency of seizures is 4.53/year in patients who consumed alcohol between 181 - 360 ml/day, the mean frequency of seizures is 6.83/year in patients who consumed alcohol between 361 -540 ml/day, the mean frequency of seizures is 12/ year in patients who consumed alcohol between 542-720 ml/day.

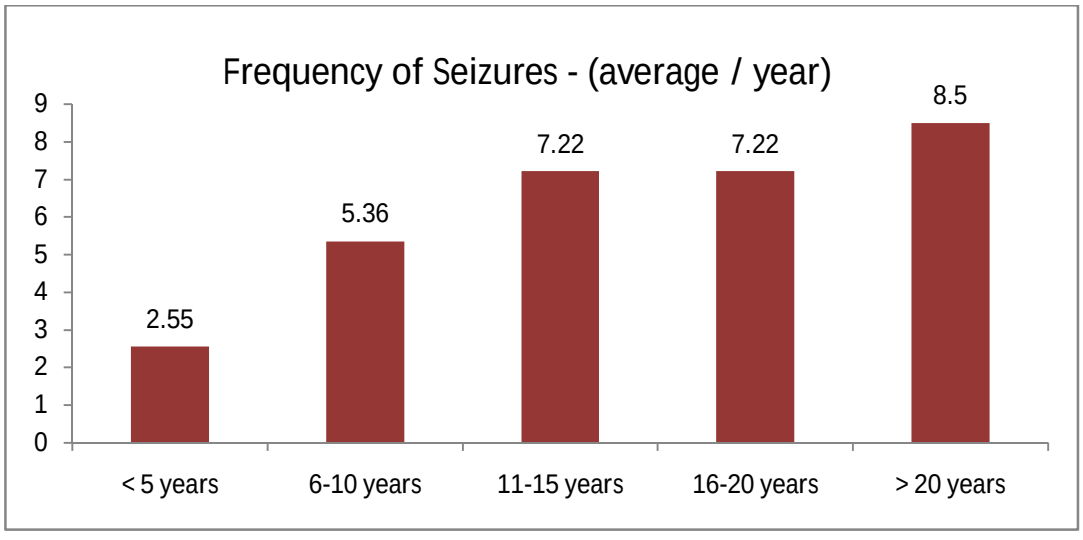
| **Quantity of alcohol consumption** | **Seizure frequency/ year** |
|---|---|
| 90 - 180 ml/day | 2.62 |
| 181 - 360 ml/day | 4.53 |
| 361 - 540 ml/day | 6.83 |
| 541 - 720 ml/day | 12 |



**Duration of alcohol consumption and frequency of seizures**

The mean frequency of seizures is 2.55/ year in patients consumed alcohol less than 5 years, the mean frequency of seizures is 5.36/ year in patients consumed alcohol between 6-10 years, the mean frequency of seizures is 7.22/year in patients consumed alcohol between 11-15 years, the mean frequency of seizures is 7.22/year in patients consumed alcohol between16-20 years, the mean frequency of seizures is 8.5/year in patients consumed alcohol more than 20 years

| **Duration of alcohol consumption** | **Frequency of seizures (average/ year)** |
|---|---|
| Less than 5 years | 2.55 |
| 6 -10 years | 5.36 |
| 11 -15 years | 7.22 |
| 16 – 20 years | 7.22 |
| More than 20 years | 8.5 |



**Liver function test**

29 (65.9 %) patients had elevated liver function test and 15 (34.1 %) patients didn't have an elevated liver function test.

| Liver Function Test | Number of Patients (%) |
|---|---|
| Elevated Liver Function Test | 29 (65.9) |
| Normal Liver Function Test | 15 (34.1) |

**Electroencephalography (EEG)**

41 (93.1 %) patients had normal EEG, 3 (6.82 %) patients had interictal epileptiform discharges.

| Electroencephalography | Number of Patients (%) |
|---|---|
| Normal EEG | 41 (93.18%) |
| Abnormal IED's | 3 (6.82%) |

# DISCUSSION

In our study 44 patients with alcohol dependence who presented with seizures were included. The mean age of patients in our group was around 35.93 years of age. Studies conducted by Bajaj et al and Pratima Murthy et al also showed that the mean age of patients in their study group was the same, suggesting that the alcohol dependence seizures affected the most productive age group of the above patients.

In our study all the patients were males and it is similar to the study conducted by Bajaj et al. The reason for this could be that in our Indian culture predominantly the males consume alcohol.

In our study 26 (59.1%) of patients were from urban area and 18 (40.9%) of patients were from rural area. The study population was predominantly from the urban sector probably because our hospital which is a tertiary referral centre is located in urban region.

According to updated Kuppusamy scale 29 (65.9 %) patients belong to class iv socio economic status and 15 (34.1 %) of patients belong to class iii socio economic status. Our hospital is a government hospital which treats the poorer section of the society free of costs could

be the reason for the high incidence of class iv category seen in our study.

As majority of patients were from class iv socio economic category most of them were school dropouts.

In our study the mean age of initiation of alcohol consumption was 25.18. It is similar to the study conducted by Bajaj et al which was 25.25 and pratima murthy et al which was 22.1.

9 (20.5 %) of patients have consumed alcohol for less than 5 years and 35 (79.5%) of patients have consumed alcohol for more than 5 years. The longer the duration of alcohol consumption the higher the frequency of seizures were observed.

A higher frequency of seizures was noted in patients who had consumed more than 180 ml of alcohol per day. The above two observations suggests that the higher frequency of seizures was noted in patients who consumed alcohol for a longer duration and who had consumed more amount of alcohol per day. Studies by Bajaj et al and Pratima Murthy et al also have shown similar results.

In our study 19 (43.2 %) of patients consumed brandy, 7(15.9 %) of patients consumed whisky, 3 (6.8 %) of patients consumed rum, 15 (34.1%) of patients consumed mixed alcoholic beverages.

Schaumenn et al observed the odds ratio of seizures to be 2.45 times among the relatives of patients with alcohol related seizures. 5 (11.4 %) of our patients had a family history of seizures. The higher percentage of family history of seizures could probably suggest a genetic etiology for the same.

11 (25%) patients had epilepsy prior to alcohol consumption. The high percentage of patients with epilepsy noted in our study could probably be due to the fact the most of our patients were daily wage labourers who consumed alcohol irrespective of their co-morbid conditions. The study by prathiba et al showed that only 10% of patients had epilepsy. The reason for the marked difference could be the different socio-economic strata of the two populations.

In our patients, 11(25%) of patients had epilepsy prior to alcohol consumption probably due to epilepsy syndrome, 24 (54.55%) of patients had alcohol withdrawal seizures and 9 (20.45%) of patients had alcohol related seizures (excluding the alcohol withdrawal group).

In the alcohol withdrawal seizures group, 15 (62.5%) of patients had generalized seizures and 8 (33.4%) of patients of alcohol dependent patients had complex partial seizures with secondary generalization. It is similar to the study conducted by pratima murthy et al was 59% of patients had generalized seizures and 4% had complex partial seizures.

In epilepsy patients with alcohol dependence group, 7 (63.7%) of patients had complex partial seizures with secondary generalization and 4 (36.3%) of patients of alcohol dependent patients had generalized seizures.

In the In patients with epilepsy who developed alcohol dependence a higher percentage of complex partial seizures (63.7%) were noted. In the alcohol withdrawal seizure group a higher percentage of patients had generalized seizures (62.5%) and a smaller percentage of patients developed complex partial seizures. This apparent difference in the pattern of seizures between the two groups makes it necessary to distinguish the two groups to tailor treatment accordingly.

The limitation of our study is very small sample size they may not be truly representative of the community sample.

# SUMMARY AND CONCLUSION

- Alcohol dependent patients have an increased propensity to develop seizures.
- Alcohol dependence seizures affected the most productive age group of males in our society as demonstrated in our study.
- Alcohol dependence seizures is not a homogenous entity, it is a heterogeneous entity consisting of patients with alcohol related seizures, alcohol withdrawal seizures and epilepsy patients who developed alcohol dependence later in their life.
- The duration of alcohol consumption is directly correlated with a higher frequency of seizures.
- The more the consumption of alcohol per day the higher the frequency of alcohol withdrawal seizures.
- Alcohol withdrawal seizure patients had a higher frequency of generalized seizures.
- Epilepsy patients who developed alcohol dependence later in their life had a higher frequency of complex partial seizures.

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# PROFORMA

Name: Age: Sex :

Marital status: Phone no:

Address: Residence:

Socio economic status: Study no/ Neuro no:

Literacy status: Degree:

occupation,:

consanguinity:

family history of seizures: Relation to index case:

family h/o febrile seizures: Relation to index case:

Place of delivery: 1) Home 2) Institutional

Mode of Delivery: Normal vaginal delivery / Assisted delivery (LSCS)

Developmental history: normal / abnormal

Motor:

Language:

Social / Behaviour:
H/O onset of Alcohol consumption :

Duration of alcohol consumption :

Consumption of alcohol/ Day : ml

Consumption of alcohol/week : days

Type of alcohol consumed:
Beer/Whisky/Brandy/Vodka/Jin/Rum/Arrack/Mixed

Any change in recent drinking pattern :

Any recent withdrawal of alcohol :

Any symptoms of alcohol withdrawal :

Any other drug abuse :

Any history of Road traffic accident :

Any history of head injury :

Age of onset of first seizures:

Febrile seizures / neonatal sepsis / head injury / encephalitis / others

Habitual seizures:

Aura type: detailed description:

Ictal semiology detailed description:

Tonic / Clonic /Myoclonic / Atonic / Versive / Epileptic spasm / Hypomotor /unclassified motor / Autonomic

Seizure type: SPS /CPS / CPS with secondary generalization / Generalized

Duration of each attack

Tongue bite /Urination:

Post ictal confusion / Post ictal weakness:

No. of seizure type: one / two / three /four / more

Seizure free period between 1st seizure and habitual seizures:

Clusters:

Nocturnal seizures only / day alone / day and night seizures:

Status epilepticus history:

Frequency of seizures before regular treatment: detailed description:

Present medication:

Name of the drug: dose in mg:

No of drugs:

Seizures episodes on medication:

Adverse effects of drugs:

Treatment of co morbid condition

Examination:

Ht: Wt: HC: Handedness:

Focal neurological deficits: Details:

Others:

MRI Brain:

Interictal EEG:

Liver function tests:

HIV/ VDRL:

Other investigations:

Follow up:

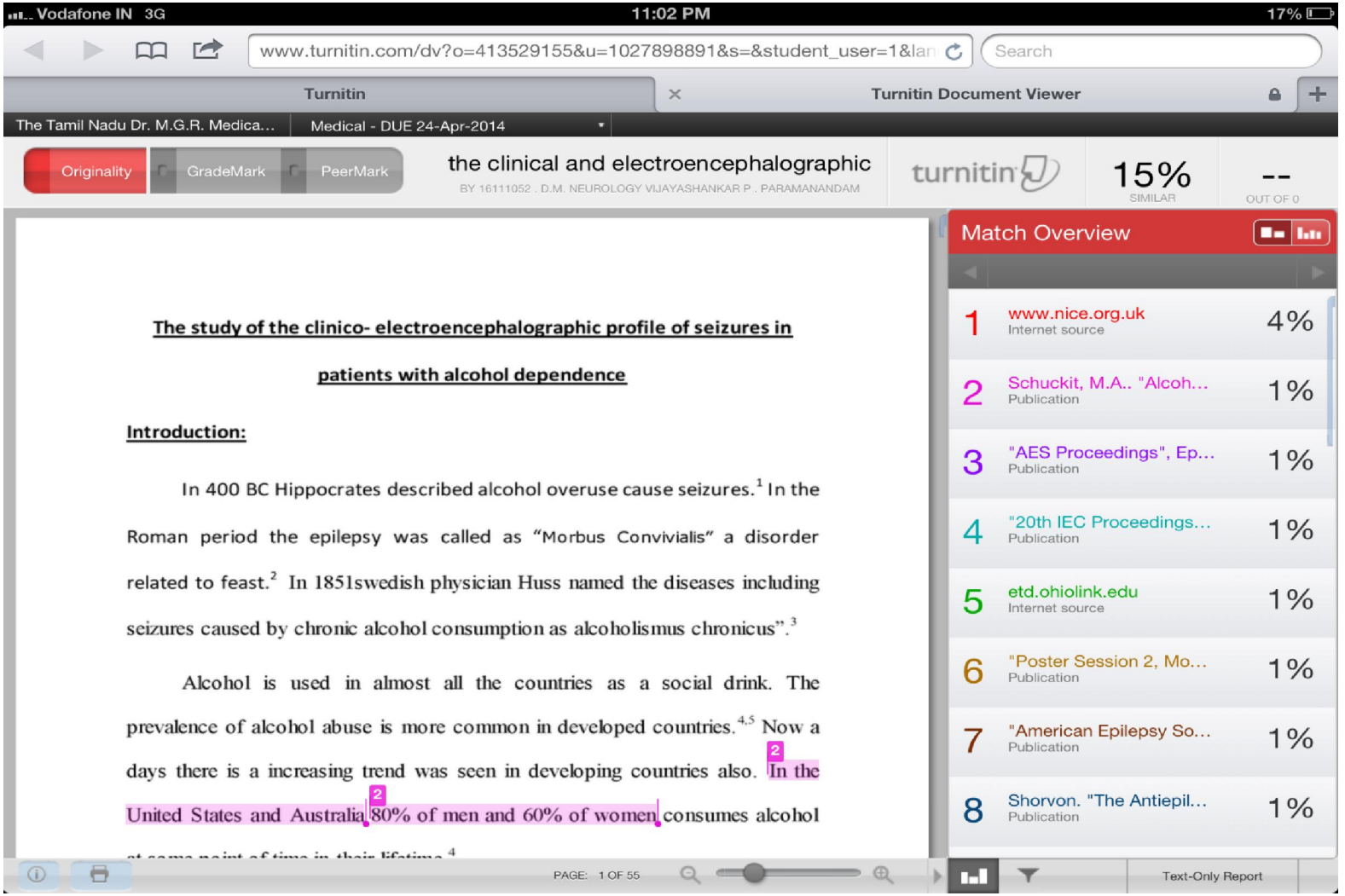
Medical events and others:

# MASTER CHART

| S.no | Name | Age | Sex | socio economic status | literacy | Residence | Age Onset of alcohol consumption | Duration of alcohol consumption | Quantity of alcohol consumption |
|---|---|---|---|---|---|---|---|---|---|
| 1 | Elango | 19 | male | iv | I - V standard | Urban | 18 | 1 | 360 |
| 2 | Siva | 29 | male | iv | VI -XII standard | Urban | 19 | 10 | 500 |
| 3 | Murugan | 35 | male | iv | I - V standard | Rural | 20 | 15 | 90 |
| 4 | Muruganandam | 41 | male | iii | Graduated | Rural | 26 | 15 | 180 |
| 5 | Karthik | 21 | male | iii | VI -XII standard | Urban | 17 | 4 | 360 |
| 6 | Arun | 42 | male | iii | VI -XII standard | Urban | 36 | 6 | 180 |
| 7 | Anandan | 33 | male | iv | I - V standard | Urban | 23 | 10 | 360 |
| 8 | Chandran | 37 | male | iv | Illiterate | Rural | 17 | 20 | 480 |
| 9 | Santhoshkumar | 24 | male | iv | VI -XII standard | Rural | 18 | 6 | 360 |
| 10 | Chinna durai | 42 | male | iv | I - V standard | Urban | 32 | 10 | 540 |
| 11 | Vinoth kumar | 23 | male | iv | VI -XII standard | Urban | 15 | 8 | 540 |
| 12 | Sudhakar | 34 | male | iii | Illiterate | Rural | 22 | 12 | 360 |
| 13 | Raji | 33 | male | iii | VI -XII standard | Rural | 30 | 3 | 360 |
| 14 | Ganesh | 53 | male | iv | I - V standard | Urban | 37 | 15 | 540 |
| 15 | Anand | 32 | male | iv | VI -XII standard | Urban | 21 | 11 | 360 |
| 16 | Kamalesh | 34 | male | iv | VI -XII standard | Urban | 14 | 20 | 360 |
| 17 | Vinoth | 25 | male | iii | VI -XII standard | Urban | 21 | 4 | 360 |
| 18 | Jeeva | 35 | male | iii | Illiterate | Urban | 15 | 20 | 520 |
| 19 | Rajendran | 40 | male | iv | Graduated | Rural | 25 | 15 | 180 |
| 20 | Kanakaraj | 31 | male | iii | I - V standard | Rural | 26 | 5 | 180 |
| 21 | Ramesh | 43 | male | iv | VI -XII standard | Urban | 23 | 20 | 360 |
| 22 | Srinivasan | 40 | male | iii | I - V standard | Rural | 32 | 8 | 360 |
| 23 | George | 37 | male | iv | VI -XII standard | Urban | 22 | 15 | 360 |
| 24 | Jaggiah | 46 | male | iii | Illiterate | Rural | 38 | 8 | 360 |
| 25 | Baskar | 45 | male | iii | I - V standard | Rural | 35 | 10 | 180 |
| 26 | Manogaran | 46 | male | iv | VI -XII standard | Urban | 38 | 18 | 360 |
| 27 | Suresh | 29 | male | iv | VI -XII standard | Urban | 19 | 10 | 360 |
| 28 | Madurai | 30 | male | iv | I - V standard | Rural | 25 | 5 | 360 |
| 29 | Saravanan | 45 | male | iv | VI -XII standard | Urban | 30 | 15 | 360 |
| 30 | Baskar | 40 | male | iv | Illiterate | Urban | 20 | 20 | 360 |
| 31 | Murugan | 35 | male | iv | I - V standard | Rural | 26 | 9 | 360 |
| 32 | Deva | 24 | male | iv | VI -XII standard | Urban | 15 | 9 | 360 |
| 33 | Soukath ali | 44 | male | iv | VI -XII standard | Rural | 38 | 7 | 360 |
| 34 | Narayanan | 35 | male | iii | I - V standard | Urban | 30 | 5 | 360 |
| 35 | Venkatesan | 50 | male | iv | Illiterate | Urban | 40 | 10 | 360 |
| 36 | Sathish | 29 | male | iv | VI -XII standard | Urban | 27 | 2 | 360 |
| 37 | Edward | 57 | male | iv | VI -XII standard | Rural | 37 | 20 | 240 |
| 38 | Jayakumar | 27 | male | iii | I - V standard | Rural | 18 | 9 | 720 |
| 39 | Chakravarthy | 45 | male | iv | I - V standard | Rural | 27 | 18 | 720 |
| 40 | Perumal | 33 | male | iv | Illiterate | Urban | 28 | 5 | 360 |
| 41 | Ravi | 40 | male | iv | VI -XII standard | Urban | 25 | 15 | 360 |
| 42 | Srinivasan | 30 | male | iii | I - V standard | Rural | 16 | 14 | 180 |
| 43 | Narendra kumar | 30 | male | iii | VI -XII standard | Urban | 21 | 9 | 360 |
| 44 | velu | 38 | male | iv | VI -XII standard | Urban | 26 | 9 | 180 |

| Type of Alcohol consumption | Epilepsy patients in the alcohol dependence group | Age of onset of seizures | Neurological deficits: | type of epilepsy in alcohol dependence patients | Type of Alcohol related seizures |
|---|---|---|---|---|---|
| Whiskey | Yes | 15 | No | Complex partial seizures | No |
| Brandy | NO | 25 | No | No | No |
| Whiskey | NO | 35 | No | No | Complex partial seizures |
| Brandy | NO | 40 | No | No | No |
| Mixed | NO | 21 | No | No | Generalized seizures |
| Whiskey | NO | 36 | No | No | No |
| Brandy | NO | 33 | No | No | Generalized seizures |
| Mixed | NO | 36 | No | No | No |
| Mixed | NO | 24 | No | No | Myoclonic epilepsy |
| Brandy | NO | 42 | No | No | No |
| Whisky | NO | 22 | No | No | No |
| Brandy | NO | 31 | No | No | No |
| Brandy | NO | 32 | No | No | No |
| Mixed | NO | 53 | No | No | Generalized seizures |
| Mixed | NO | 24 | No | No | No |
| Brandy | NO | 34 | No | No | Complex partial seizures |
| Brandy | NO | 24 | No | No | No |
| Brandy | NO | 34 | No | No | No |
| Brandy | NO | 40 | No | No | No |
| Brandy | NO | 31 | No | No | No |
| Mixed | NO | 15 | No | No | No |
| Rum | yes | 26 | No | Complex partial seizures | No |
| Brandy | NO | 34 | No | No | No |
| brandy | NO | 41 | No | No | No |
| Mixed | yes | 16 | No | No | No |
| Brandy | NO | 43 | No | No | No |
| Mixed | yes | 13 | No | Complex partial seizures | No |
| Mixed | NO | 27 | No | No | No |
| Whiskey | yes | 5 | No | Generalized seizures | No |
| Mixed | NO | 40 | No | Generalized seizures | No |
| Whiskey | yes | 13 | No | Complex partial seizures | No |
| Rum | NO | 17 | No | No | No |
| Brandy | yes | 28 | No | Complex partial seizures | No |
| Mixed | yes | 28 | No | Generalized seizures | No |
| Mixed | NO | 5 | No | No | Generalized seizures |
| Brandy | NO | 29 | No | No | Generalized seizures |
| Brandy | yes | 55 | No | Generalized seizures | No |
| Rum | NO | 2 | No | No | No |
| Mixed | yes | 27 | No | Complex partial seizures | No |
| Whiskey | NO | 1 | No | No | Generalized seizures |
| Mixed | NO | 5 | No | No | No |
| Brandy | NO | 4 | No | No | No |
| Mixed | NO | 13 | No | No | |
| Brandy | NO | 24 | No | Complex partial seizures | No |

| pe of Alcohol withdrawal seizu | Clusturs of seizures | Time of seizures | LFT | frequency of seizures | family history of seizures | EEG |
|---|---|---|---|---|---|---|
| No | No | 1 | normal | Once in six months | No | Spike and wave pattern |
| Complex partial seizures | No | 1 | elevated | once in a month | No | Normal study |
| No | No | 1 | elevated | once a year | No | Normal study |
| Generalized seizures | No | 1 | elevated | once in six months | No | Normal study |
| No | No | 1 | normal | once in a year | No | Normal study |
| Generalized seizures | No | 1 | elevated | once in six months | No | Normal study |
| No | yes | 2 | elevated | once in a year | No | Normal study |
| Complex partial seizures | No | 1 | elevated | once insix months | No | Normal study |
|  | No | 1 | elevated | once in a year | No | left fronto central spikes |
| Generalized seizures | No | 1 | elevated | Once in a month | No | Normal study |
| Generalized seizures | No | 1 | elevated | Once in a month | No | Normal study |
| Complex partial seizures | No | 1 | elevated | once in a month | No | Bilateral fronto central spkes |
| Generalized seizures | No | 1 | elevated | once in a year | No | Normal study |
| No | No | 1 | normal | once in a month | No | Normal study |
| Complex partial seizures | yes | 2 | elevated | once in a month | yes | Normal study |
| No | No | 1 | elevated | once in a month | No | Normal study |
| Complex partial seizures | yes | 2 | normal | once in six months | No | Normal study |
| Complex partial seizures | No | 1 | elevated | once in six months | yes | Normal study |
| Generalized seizures | No | 1 | elevated | once in a year | No | Normal study |
| Complex partial seizures | YES | 1 | normal | once in a year | No | Back ground slowing |
| Generalized seizures | No | 1 | elevated | once in six months | yes | Normal study |
| No | yes | 1 | normal | Once in six months | No | Normal study |
| Generalized seizures | No | 1 | elevated | Once in six months | No | Normal study |
| Generalized seizures | No | 1 | elevated | once in a month | No | Normal study |
| Generalized seizures | No | 1 | elevated | once in a month | yes | Normal study |
| Generalized seizures | No | 1 | elevated | once in a month | No | Normal study |
| No | No | 1 | elevated | once in a month | No | Normal study |
| Generalized seizures | No | 1 | normal | once in six months | No | Normal study |
| No | No | 1 | elevated | Once in six months | No | Normal study |
| No | No | 1 | elevated | Once in six months | No | Normal study |
| No | No | 1 | normal | Once in six months | No | Normal study |
| Complex partial seizures | No | 1 | normal | once in a month | No | Normal study |
| No | No | 1 | normal | once a month | yes | Normal study |
| No | No | 1 | normal | once in a month | No | Normal study |
| No | No | 1 | elevated | once a year | No | Normal study |
| No | No | 1 | normal | once in a year | No | Normal study |
| No | No | 1 | elevated | Once in a month | No | Normal study |
| Generalized seizures | No | 1 | normal | once in a month | No | Normal study |
| No | No | 1 | elevated | once in a month | No | Normal study |
| No | No | 1 | normal | once in a year | No | Normal study |
| Generalized seizures | No | 1 | normal | once in six months | No | Normal study |
| Complex partial seizures | No | 1 | elevated | once in a year | No | Normal study |
| Complex partial seizures | No | 1 | elevated | once in a year | No | Normal study |
| No | yes | 1 | elevated | once in a year | No | Normal study |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Introduction:
In 400 BC Hippocrates described alcohol overuse cause seizures.[1] In the Roman period the epilepsy was called as "Morbus Convivialis" a disorder related to feast.[2] In 1851swedish physician Huss named the diseases including seizures caused by chronic alcohol consumption as alcoholismus chronicus".[3]
Alcohol is used in almost all the countries as a social drink. The prevalence of alcohol abuse is more common in developed countries.[4,5] Now a days there is a increasing trend was seen in developing countries also. In the United States and Australia 80% of men and 60% of women consumes alcohol
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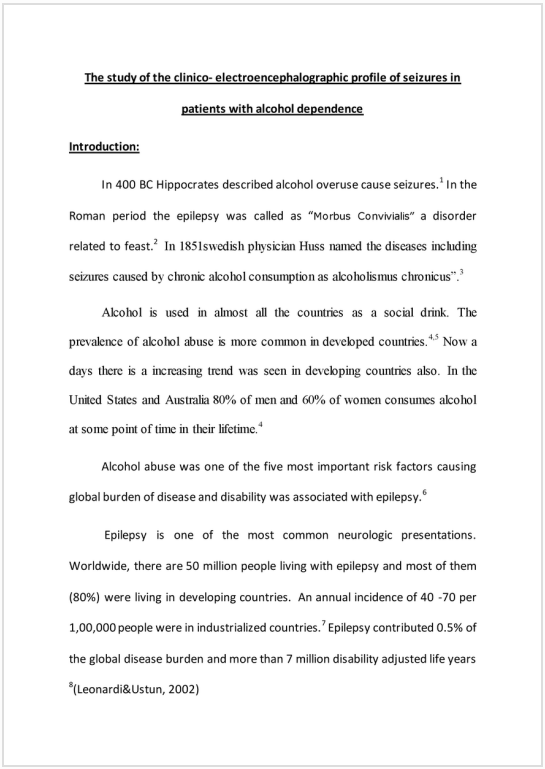

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**Introduction:**

In 400 BC Hippocrates described alcohol overuse cause seizures.[1] In the Roman period the epilepsy was called as "Morbus Convivialis" a disorder related to feast.[2] In 1851swedish physician Huss named the diseases including seizures caused by chronic alcohol consumption as alcoholismus chronicus".[3]

Alcohol is used in almost all the countries as a social drink. The prevalence of alcohol abuse is more common in developed countries.[4,5] Now a days there is a increasing trend was seen in developing countries also. In the United States and Australia 80% of men and 60% of women consumes alcohol at some point of time in their lifetime.[4]

Alcohol abuse was one of the five most important risk factors causing global burden of disease and disability was associated with epilepsy.[6]

Epilepsy is one of the most common neurologic presentations. Worldwide, there are 50 million people living with epilepsy and most of them (80%) were living in developing countries. An annual incidence of 40 -70 per 1,00,000 people were in industrialized countries.[7] Epilepsy contributed 0.5% of the global disease burden and more than 7 million disability adjusted life years [8](Leonardi&Ustun, 2002)