PSYCHIATRIC MORBIDITY IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Dissertation submitted for partial fulfillment

of the rules and regulations

DOCTOR OF MEDICINE

BRANCH - XVIII (PSYCHIATRY)

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY,

CHENNAI,

TAMIL NADU

MAY 2019

CERTIFICATE

This is to certify that the dissertation titled, **"PSYCHIATRIC MORBIDITY IN PATIENTS WITH TRAUMATIC BRAIN INJURY"** is the bonafide work of **Dr. KIRUTHIKA.A**, in part fulfilment of the requirements for the M.D. Branch – XVIII (Psychiatry) examination of The Tamilnadu **Dr.M.G.R. Medical University,** to be held in May 2019. The period of study was from April 2017 – Sep 2017.

The Director,
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Chennai- 600003

CERTIFICATE OF GUIDE

This is to certify that the dissertation titled, **"PSYCHIATRIC MORBIDITY IN PATIENTS WITH TRAUMATIC BRAIN INJURY"** is the original work of **Dr. KIRUTHIKA. A**, done under my guidance submitted in partial fulfilment of the requirements for M.D. Branch – XVIII [Psychiatry] examination of The Tamilnadu Dr. M. G. R. Medical University, to be held in May 2019.

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DECLARATION

I, DR.A.KIRUTHIKA, solemnly declare that the dissertation titled **"PSYCHIATRIC MORBIDITY IN PATIENTS WITH TRAUMATIC BRAIN INJURY"** is a bonafide work done by myself at the Madras Medical College, Chennai, during the period from April 2017 – September 2017 under the guidance and supervision of **Prof. Dr. POORNACHANDRIKA MD, DCH**, Professor of Psychiatry, Madras Medical College. The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards part fulfilment for M.D. Branch XVIII (Psychiatry) examination.

Place: **Dr. A.KIRUTHIKA**

Date :

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CERTIFICATE OF APPROVAL

To

Dr.Kiruthika. A
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Dear Dr.Kiruthika.A,

The Institutional Ethics Committee has considered your request and approved your study titled **"PSYCHIATRIC MORBIDITY IN PATIENTS FOLLOWING TRAUMATIC BRAIN INJURY" - NO.21042017**

The following members of Ethics Committee were present in the meeting hold on **04.04.2017** conducted at Madras Medical College, Chennai 3

1.Prof.Dr.C.Rajendran, MD.,	:Chairperson
2.Prof.K.Narayanasamy, MD.,DM.,Dean(FAC), MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	:Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3	: Member
6.Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
7.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
8.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
9.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
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INTRODUCTION

Traumatic brain injury is one of the most common causes for high rates of mortality, morbidity and disability. It has been named as ‘silent epidemic’. India has the highest accident rates per vehicle in the world. The rehabilitation needs for it, is increasing. Apart from its physical effects, its psychiatric effects impair the social life and it is underdiagnosed and undertreated. It is therefore essential to look for it, for limiting the disability. It refers to the external force applied over the brain that causes permanent or temporary dysfunction. It is a universal health problem.

EPIDEMIOLOGY OF TRAUMATIC BRAIN INJURY

In India[1], nearly 1.5 to 2 million persons are injured and 1 million end to death every year. Road traffic accidents are the leading cause (60%) followed by falls (20-25%) and violence(10%).

TBI refers to the extracranial mechanical force to the brain that leads to any of the following:

1. Any period of loss of consciousness.
2. Any loss of memory for events immediately before or after the event.
3. Any alteration in mental state at the time of the Event.

Mild traumatic brain injury may affect the brain cells temporarily.

It can result in a variety of neuropsychiatric Disturbance from subtle deficits to severe intellectual and emotional disturbances and to severe vegetative states. Incidence over the rise in developing countries are due to high two wheeler usage and poor road safety conditions. Males are likely to have TBI than females with high cases over second and third decades. Falls more frequent in the elderly.

COMPARATIVE NOSOLOGY

InICD-10

"F06: Other mental disorders due to brain damage and dysfunction and to physical disease

F07 : Personality and behavioural disorder due to brain disease, damage and dysfunction

InDSM 5

Major Neurocognitive Disorder due to Traumatic brain injury

294.10: without behavioural disturbance

294.11: with behavioural disturbance

331.83: Mild Neurocognitive Disorder due to Traumatic brain injury"

REVIEW OF LITERATURE

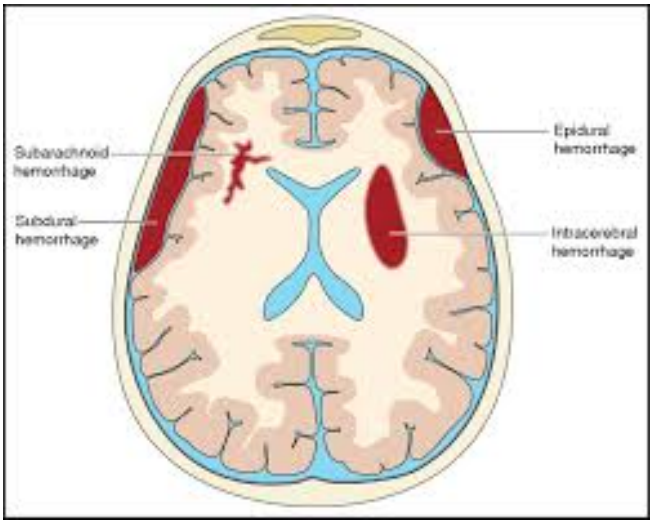
MAJOR TYPES OF HEAD INJURY

Hematoma

A hematoma is clotting of blood outside the blood vessels in the external space. The clotting can cause pressure building up inside the skull. This can cause loss of consciousness or result in permanent brain damage.

Hemorrhage

A hemorrhage is an uncontrolled bleeding. There can be bleeding in the space around your brain, called subarachnoid hemorrhage, and bleeding within your brain tissue, called intracerebral hemorrhage.Subarachnoid hemorrhages often cause headaches and vomiting. The severity of intracerebral hemorrhages depends on amount of bleeding, but any amount of blood can cause pressure buildup over the time.



Concussion

A concussion occurs when the impact on the head is severe enough to cause brain tissue injury. It's thought to be the result of the brain hitting against the skull or the forces of sudden acceleration and deceleration. The loss of function and consciousness associated with a concussion is temporary. However, repeated concussions can eventually lead to permanent damage.

Edema

Any type of brain injury can lead to edema or swelling. Many injuries cause swelling of the surrounding tissues, but injury to brain is more serious. The skull can't stretch to accommodate the swelling. This leads to building up of pressure in our brain, which causes the brain to press against the skull.

SYMPTOMS OF A HEAD INJURY:

Head has more blood vessels than any other part of our body, so bleeding anywhere related to brain is a serious concern in head injuries. But not all head injuries will cause bleeding. Symptoms must be closely monitored after a head injury.

Common symptoms of a minor head injury include:

- Headache
- Light Headedness
- Spinning Sensation
- Mild Confusion

- nausea
- temporary ringing in the ears

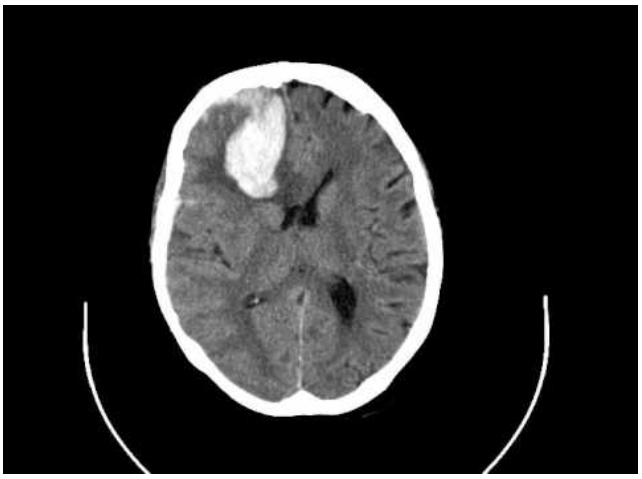
The symptoms of a severe head injury include many of the symptoms of minor head injuries. They can also include:

- loss of consciousness
- seizures
- vomiting
- balance or coordination problems
- disorientation
- abnormal eye movements
- loss of muscle control
- persistent or worsening headache
- memory loss
- inability to focus the eyes
- changes in mood
- leaking of clear fluid (CSF) from the ear or the nose

SYMPTOMS BASED ON LOCATION OF INJURY:

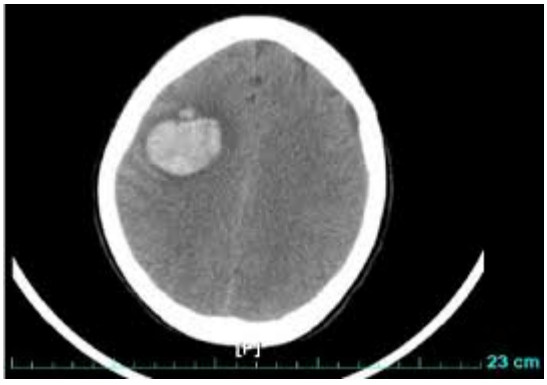
Patients with left parietotemporal brain lesions showed significant losses on the AGCT of general intelligence when pre- and post-traumatic scores were being compared. Among unilateral wounds, the left sided lesions were associated with more psychiatric disturbance than the right, particularly where dementias and psychoses were more concerned. Patients with parietal, occipital and cerebellar lesions were comparatively free from psychiatric problems.

Left hemisphere lesions to be more closely associated with overall psychiatric disability than right hemisphere lesions and patients with injury to the left temporal lobe is the most common with highest risk. However, intellectual disorders were found more commonly after left hemisphere lesion especially parietal and temporal lobes, while affective disorders, behavioural disorders and somatic complaints were more frequent after right hemisphere lesion. Intellectual disorders were especially associated with damage to the parietal and temporal lobes, whereas affective disorders, behavioural disorders and somatic complaints were more frequent after frontal lobe damage.

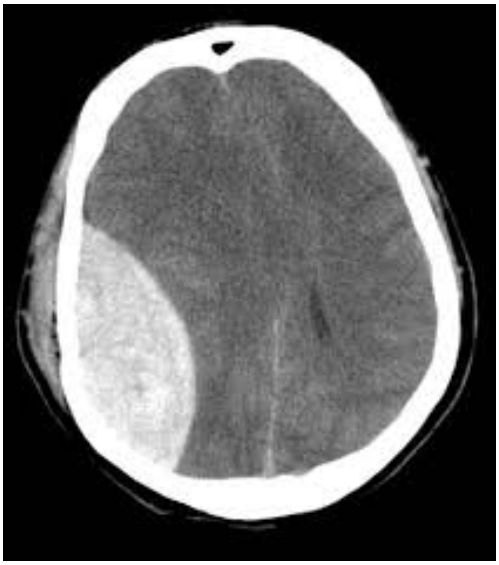


FRONTAL HEMORRHAGE

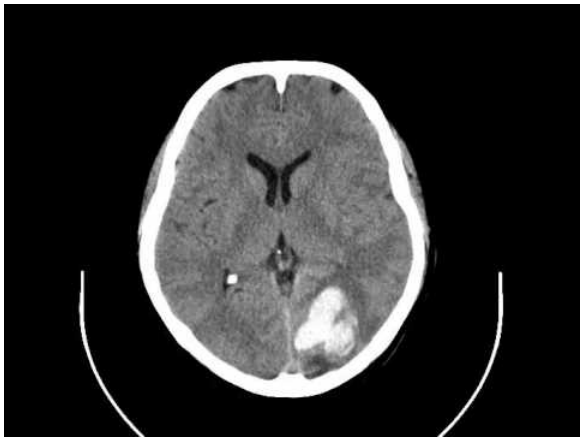
The frontal lobe controls high level cognitive functions such as planning, initiating, and organizing, as well as personality. When an individual suffers a concussion, or traumatic brain injury (TBI), the frontal lobe is often an area that suffers damage



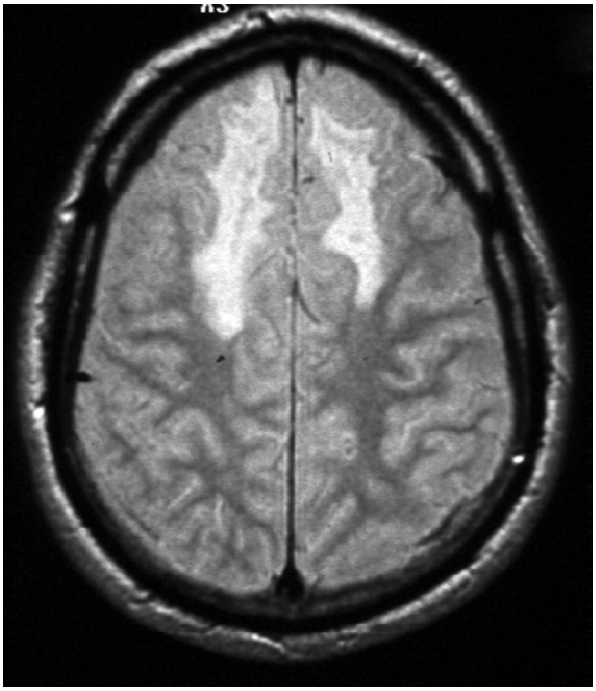
FRONTOPARIETAL HEMORRHAGE- Damage to the left **parietal lobe** can result in what is called "Gerstmann's Syndrome." It includes right-left confusion, difficulty with writing (agraphia) and difficulty with mathematics (acalculia). It can also produce disorders of language (aphasia) and the inability to perceive objects normally (agnosia)



TEMPORAL HEMORRHAGE-Left side lesions result in decreased recall of verbal and visual content, including speech perception. Right side lesions result in decreased recognition of tonal sequences and many musical abilities. Rightside lesions can also affect recognition of visual content (e.g. recall of faces). The temporal lobes are involved in the primary organization of sensory input

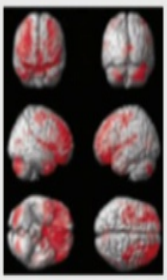


OCCIPITAL HEMORRHAGE-Located at the back of the brain, the occipital lobes are responsible for visual perception. Damage to them results in loss of visual capability, an inability to identify colours, and hallucinations. At times, patients experience severe vision loss or total blindness



DIFFUSE AXONAL INJURY- on impact there is little evidence of axons being torn apart, but the axolemma is damaged by the stretching and compression forces. This allows calcium to enter the axon, activating proteases that damage the intracellular structures i.e neurofilaments and microtubules necessary for axoplasmic transport.

Brain regions vulnerable to TBI and relationship to neurobehavioral sequelae



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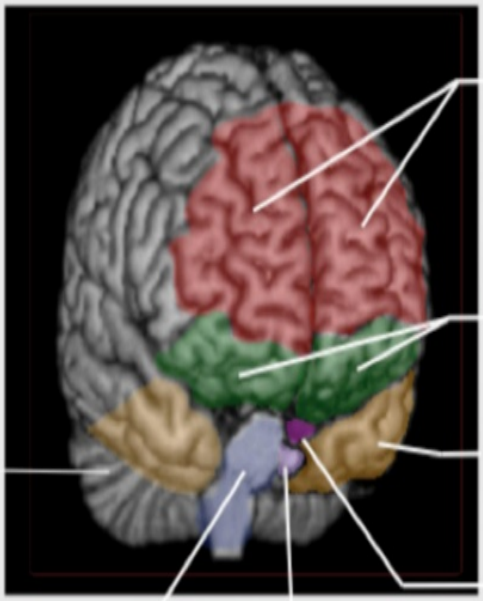


B

Dorsolateral prefrontal cortex
(executive function, working memory, sustained and complex attention, memory retrieval, abstraction, judgement, insight, problem solving)

Orbitofrontal cortex
(emotional and social responding, social comportment)

Temporal polar cortex
(memory retrieval, sensory-limbic integration)

Amygdala
(emotional learning and memory, fear conditioning)

Cerebellum
(coordination, working memory, mood regulation)

Ventral brain stem
(arousal, ascending modulatory neurotransmitter systems)

Entorhinal-hippocampal complex
(declarative memory, sensory gating, attention)

NEUROPSYCHIATRIC MANIFESTATIONS OF TBI:

Neuropsychiatric[2] aspects of TBI can be divided into-

1) Acute effects

2) Chronic Sequelae

Acute Effects of Head Injury –

1) Impairment / Loss of Consciousness

2) Post Traumatic Delirium / Confusional state

3) Post traumatic agitation

4) Post Traumatic Amnesia (PTA).

Chronic Sequelae of Head Injury –

Depends on multiple factors like mental constitution and premorbid personality, age at the time of injury, circumstances of the injury, compensation, secondary gain and attribution bias, development of epilepsy, amount of brain damage incurred, alcohol and drug abuse, location of brain damage incurred.

Post-head injury neuropsychiatric manifestations[2] include

1. COGNITIVE IMPAIRMENT
2. PERSONALITY CHANGES
3. AGGRESSION-predictors of aggression in TBI includes - alcohol, younger age at injury, being depressed, frontal injury, a pre-injury history of antisocial behaviour.

4. ANXIETY DISORDERS
5. MOOD DISORDERS
6. PSYCHOSIS
7. POST TBI HEADACHE-Severe post TBI headache should raise suspicion of chronic SDH
8. PUNCH DRUNK SYNDROME-also called Dementia Pugilistica. progressive dementia due to diffuse injury to the cortex, basal ganglia
9. POST CONCUSSION SYNDROME-immediate and transient impairment of neural function, such as alteration of consciousness, disturbances of vision, equilibrium due to mechanical forces.
10. SUICIDE-Death by suicide occurs in about 1% of patients over the first 15 years after injury
11. RISK FACTOR FOR EPILEPSY -5% of closed injuries and around 30% in penetrating injuries
12. RISK FACTOR FOR DEMENTIA

ALCOHOL AND HEAD INJURY:

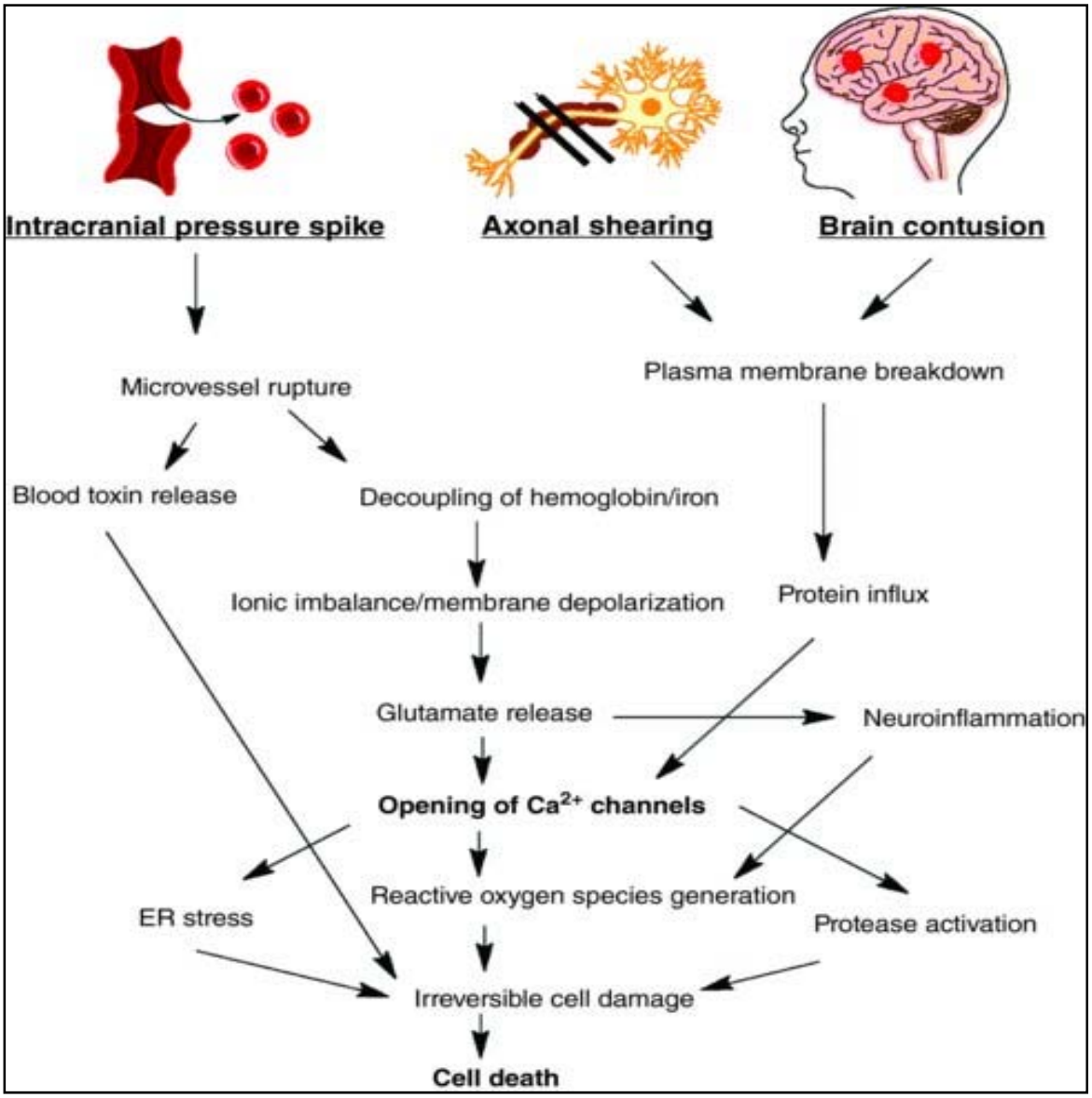
The ways in which acute alcohol intake can increase the extent of brain injury, and chronic intake can delay reparative processes within the nervous system, can be compared and learned from laboratory experimental studies. This may be because those who are intoxicated depending on the severity of traumatic brain injury, have deeper and more prolonged unconsciousness; their conscious level can be suppressed by the sedative effects of alcohol. Thus

when compared for injury severity using measures of consciousness level, intoxicated patients may have similar outcomes, despite potential deleterious effects of alcohol on outcome[3] The leading cause of trauma-related psychiatric disorders is traumatic brain injury and is associated with major public health problem[4,5]. Commonly studied disorders are Post traumatic stress disorder (PTSD) and depression after traumatic injury. Studies showed that 10%–20% of traumatic injury patients developed PTSD[6,7] and 9%–15% developed major depressive disorder[6,8].

Our knowledge about the psychiatric impact of traumatic injury is limited by many other factors. The main focus on PTSD and depression has led to neglect of many psychiatric disorders that can occur after traumatic injury. Few studies showed increased rates of anxiety and substance use disorders after traumatic injury[6,9,10], but most studies indicated that psychiatric disorders after trauma are typically comorbid with PTSD[11]. So there is a need to evaluate the full range of psychiatric disorder sequelae following traumatic injury. The psychiatric symptoms found in 120 persons with severe TBI included apathy (42%), irritability (37%), dysphoria / depressed mood (29%), disinhibition (28%), eating disturbances (27%), and agitation (24%)[12].

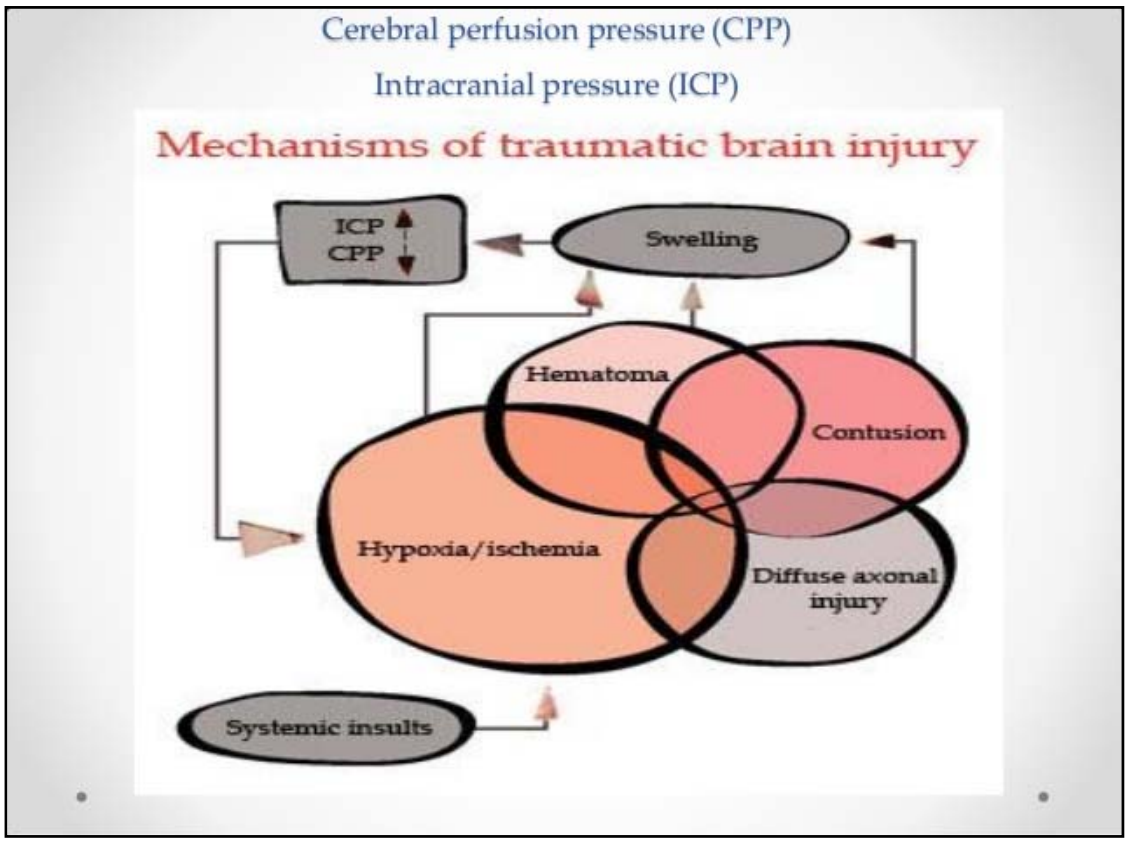
In 939 TBI patients the prevalence of any psychiatric illness in the 1st year was 49% following moderate to severe TBI and 34% following mild TBI. Whereas moderate to severe TBI is associated with a higher initial risk, mild TBI is associated with persistent psychiatric illness.

BIOLOGICAL MECHANISM: [13]Mechanical forces applied to the skull and conducted to the brain lead to focal or diffuse brain damage.



Focal injury usually result from a direct blow to the head and brain include laceration, contusion, subdural hemorrhage, subarachnoid and intracerebral hemorrhage, and ischemic infarct. Contusion which occurs directly underneath or opposite to the site of impact, commonly referred to as coup and contre-coup injury[14].

It is most common in the orbital–frontal area and the temporal regions, where acceleration and deceleration forces cause the brain to impact on the bony protuberances of the skull[15]. Diffuse brain injury can also result from the differential movement of the brain within the skull, causing shearing and stretching of the axons in the brain.



ASSESSING SEVERITY OF HEAD INJURY

Severity assessed based on combining

The initial Glasgow Coma Scale (GCS) score,

The duration of loss of consciousness (LOC), and

The duration of post traumatic amnesia (PTA).

Duration of PTA

- ✖ Assessed using the Galveston Orientation and Amnesia Test (GOAT)
- ✖ Duration of PTA- good predictor of the degree of disability, vocational outcome, and severity of personality change following TBI.

- **Mild TBI -** GCS of 13–15,
  LOC of less than 30 minutes
  and/or PTA of less than 1 hour.
- **Moderate TBI** - GCS of 9–12,
  LOC of 1–24 hours,
  and/or PTA of 30 minutes to 24 hours.
- **Severe TBI-** GCS of 8 or less,
  LOC of more 24 hours,
  and/or PTA of more than 1 day.

RISK FACTORS:

[16]The major risk factors for neuropsychiatric disturbances after head injury are old age, arteriosclerosis, and alcoholism. These factors can lead to the slowing of the reparative process of the central nervous system[17]. Premorbid personality can also play an important role in the process of rehabilitation, as mentioned by Symonds[18]. Similarly, factors such as marital discord, poor interpersonal relationships, problems at work, or financial instability are important contributors to the neuropsychiatric problems.

[19]Lishman hypothesized that many of the psychiatric symptoms following a head injury are precipitated initially by organic factors, but in some

patients they can be maintained by psychosocial factors for a long period of time[20]. Psychosocial factors may include the patient's socioeconomic status and premorbid personality. Although they could not adequately assess the premorbid personality of the patients in their study, history of psychiatric illness remained the most important prediction factor in precipitating a psychiatric illness 1 year after traumatic brain injury seemed to support Lishman's original hypothesis.

In the patient group, there were many other possible risk factors for the development of psychiatric illness that did not reach the level of statistical significance but showed a difference. These were male sex, severe head injury, and a family history of psychiatric illness.

However, psychiatric illness was more significantly common among the patients who showed unfavourable outcome according to the Glasgow Outcome Scale 1 year after traumatic brain injury. Interesting finding in this study was that premorbid factors such as lower social class and lower educational achievement, head-injury-related factors such as a low Glasgow Coma Scale score, and outcome-related factors such as the presence of disability and psychiatric "caseness" according to the Clinical Interview Schedule—Revised significantly influenced the rate and the pattern of neurobehavioral symptoms in the same study group[21]; as in this study, the rate of neurobehavioral symptoms was not related to the rate of compensation claims. It is now being accepted that the occurrence of a psychiatric illness following[22] traumatic brain injury depends on a complex interaction among

psychosocial and organic factors, and the findings of their study are broadly supportive of that notion.

[23]RISK FACTORS FOR POORER OUTCOME FOLLOWING TBI

Before injury	*At time of injury*	*After injury*
Age (older)	Acute symptom presentation (e.g., headaches, dizziness, or nausea in the emergency department)	Chronic pain conditions
Less education or lower levels of intelligence	Context of injury (e.g., stress, combat-related, traumatic)	Compensation
Low socioeconomic status	Lack of support system	Lack of support system
Mental health disorders (e.g., depression, anxiety, traumatic stress, substance use)		Less education
Neurologic conditions		Litigation (e.g., malingering, delayed resolution)
Sex (female)		Psychiatric disorders

RELATIONSHIP BETWEEN TIMING OF INJURY AND ONSET OF SYMPTOMS:

[19]The relationship between the time of the traumatic brain injury and the occurrence of the psychiatric symptoms is important. In this cross-sectional study, the course of psychiatric illness over a period of time was not assessed, which in the case of traumatic brain injury patients was likely to change. The lack of a properly matched control group was a drawback of the study. They found that patients with traumatic brain injury were often men of younger age coming from a lower socioeconomic background, misused alcohol and drugs, and possessed certain premorbid personality traits. These characteristics acts as a risk factor for developing psychiatric illness but at the same time made it

difficult to find a properly matched control group for them. These studies found it difficult to compare their findings with previous studies because of the relative lack of information on this subject.

Fenton and colleagues[24] assessed 41 consecutive admissions of head-injury patients and found that 39% had a psychiatric diagnosis when assessed 6 weeks after injury. Most of the other studies in this area reported mainly on psychiatric symptoms rather than a full spectrum of psychiatric syndromes. The reported rate of depression varied between 26% and 60% according to different studies[25-29].

[30]The finding that over 20% of injury survivors met diagnostic criteria for at least one psychiatric diagnosis at 12 months was disturbing given the frequency with which injury occurs. PTSD and depressive disorders were the most frequent diagnoses, accounting for 53% of all psychiatric diagnoses. Approximately half of those who were diagnosed with one psychiatric condition also met criteria for another diagnosis, with PTSD being most frequently comorbid with depression.

These findings were consistent with the epidemiological literature that showed a close comorbid relationship between PTSD and depression. The occurrence of PTSD and depression as single diagnoses (i.e., not comorbid with another disorder) was relatively unusual, which occurred in only approximately one-third of the cases.

These findings showed the importance of investigating comorbid diagnoses when assessing individuals after a trauma. The high prevalence of comorbid disorders following injury might have important implications for expected outcomes of treatment[31] and for better prognosis[3] The most common diagnosis when assessed at 12 months was depression (16.3%), followed by generalized anxiety disorder (11.1%), substance abuse (9.9%), PTSD (9.7%), agoraphobia (9.7%), social phobia (6.9%), panic disorder (5.9%), and obsessive-compulsive disorder (3.5%). Overall, 175 patients (22.2%) had a psychiatric diagnosis at 1 year without any previous diagnosis of psychiatric illness before injury (patients with mild TBI, 24.8%; patients with no TBI, 20.5%). The most common new diagnosis at 12 months following injury was depression (11.6%), followed by generalized anxiety disorder (9.5%), PTSD (7.0%), agoraphobia (6.5%), social phobia (5.0%), panic disorder (3.5%), obsessive-compulsive disorder (2.3%), and substance abuse (2.5%).

RELATIONSHIP OF TBI TO PSYCHIATRIC DISORDERS[32]

In addition to the changes in cognition, behaviour, and personality, various studies had given evidence that TBI results in an increased relative risk of developing various psychiatric disorders, including mood and anxiety disorders, substance abuse and psychotic syndromes [33-36]. For example, Kopenen et al[36] studied 60 individuals 30 years after their TBI and found that almost half (48%) developed a new Axis I psychiatric disorder after their injury that they never had before. The most common diagnoses were depression, substance abuse, and anxiety disorders consistently found in all studies. Rates

of lifetime and current depression (26%; 10%), panic disorder (8%; 6%), and psychotic disorders (8%; 8%) were significantly higher than base rates found in the Epidemiologic Catchment Area (ECA) study[37]. Hibbard et al[34] studied 100 adults on average 8 years after Traumatic Brain Injury. A significant number of individuals had Axis I disorders prior to injury. After TBI, the most frequent diagnoses were major depression and anxiety disorders (i.e., post-traumatic stress disorder (PTSD), obsessive-compulsive disorder and panic disorder). Almost half (44%) of individuals had two or more disorders that made a comorbid diagnosis. They also later reported a longitudinal study of 188 individuals enrolled within four years of injury and assessed at yearly intervals on at least two occasions[38].

They again found elevated rates (compared to population base rates as reported in the ECA study) of psychiatric disorders (depression and substance abuse) prior to injury. Following TBI there were increased rates of depression, PTSD, and other anxiety disorders. This was particularly true of those with pre-injury psychiatric disorders. Furthermore, the rates were greatest at the initial assessment point within few months after injury and stabilized or decreased over years. Van Reekum et al[35] carefully reviewed the literature on the relationship of TBI to a variety of psychiatric disorders and, using the ECA data for baseline rates, concluded that TBI was associated with an increase in the relative risk for several psychiatric disorders. Others have also reported increased indicators of psychiatric illness after TBI and increased medical costs associated with those indicators[39,40].

LATERALITY OF LESIONS TO PSYCHIATRIC DISTURBANCES[41] :

	No. of cases (total 345)	Hemisphere		Lobe(s) (right, left or both)			
		Left	Right	Frontal	Parietal	Temporal	Occipital
Any intellectual disorder	117	*			*	*	?*
General intellectual impairment	32	*				*	?*
Dysphasia	24	*			*	*	
Impairment of memory	50	*			*		*
Difficulty in concentration	87		*				*
Any affective disorder	113		*	*			
Depression	58		*	*	*		
Anxiety	40						*
Irritability	72		*	*	*		
Aggression	10			*			
Apathy	35		*				
Euphoria	10			*			
Any behavioural disorder	40		*	*			
Crime or misdemeanours	5			*			
Sexual disturbance	8			*			
Lack of judgement, etc.	20			*			
Facile or childish behaviour	17		*	*			
Disinhibition	13			*			
Somatic complaints	71		*	*			
Headache or dizziness	62			*			
Fatigue	16				*		
Sensitivity to noise	24			*			
'Frontal lobe syndrome'	32		*	*			

DEPRESSION

Major depression[2] based on the location of the injury is quite common after injury. Those with depression recovered shortly within few months with higher rates found in the first year post injury. Psychosocial factors also contributed especially in late onset depression. The most common symptom was apathy which was found in depressed head injury patients than normal depressed patients. Others include fatigue, poor concentration and insomnia.

Rapoport et al[42] assessed 170 consecutive survivors of mild TBI using the SCID at their first follow-up appointment in a TBI clinic over an average period of 48 days following injury.

Patients with pre-existing major depression or bipolar disorder were excluded from that sample. In the sample, 15.3% of the patients met DSM-IV diagnostic criteria for major depressive disorder. Subjects with major depressive disorder had higher rates of psychosocial dysfunction on the Rivermead Head Injury Follow-up Questionnaire and psychosocial distress as measured on the General Health Questionnaire. Hibbard et al[43] evaluated 100 adults over an average period of 8 years following TBI using the SCID to identify Axis I psychiatric disorders. This study yielded a prevalence rate of 61% for major depressive disorder following TBI.

A separate analysis of major depressive disorder with onset following TBI led to a lower but still maintained at a substantial rate of 48%. This second prevalence estimate might be a more reliable indicator of depression with a higher likelihood of being caused by TBI. One limitation found in that study was that the sample was self-selected by participants responding to active recruitment through TBI newsletters and other means. This have led to an "enriched" sample of TBI patients with an excess of psychiatric symptomatology.

[13]Major depression occurred in approximately 25% of patients with head injury[44,45]. It was associated with cognitive dysfunction, negative affect and

prominent anxiety symptoms. Depression occurred more frequently with left dorsolateral frontal and left basal ganglia lesions. The mechanism of depression following head injury is probably due to disruption of biogenic amine-containing neurons as they pass through the basal ganglia or frontal-subcortical white matter. Feelings of loss, demoralization and discouragement seen soon after injury were often followed by symptoms of persistent dysphoria. Suicide potential should always be evaluated[46]. Fatigue, irritability, disinterest, and insomnia presented as an initial symptoms in a substantial number of patients 6–24 months or even longer after head injury[47,48].

Psychological impairments in excess of the severity of injury and poor cooperation with rehabilitation were strong indicators of a persistent depressive disorder. Depression commonly occurred during the period of recovery of other functions. The recovering patient must come to terms with his new physical and mental limitations, and may psychologically mourn those functions impaired or lost. The common symptoms of depression might be less pronounced in TBI patients due to overall personality flattening and cognitive problems and amotivation. Erratic or poor recovery, or worsening of a neurological deficit after initial recovery make us to think about possibility of depression. Poor premorbid levels of functioning and past history of psychiatric illness were major risk factors for depression[49].

The prevalence of depression in TBI[50] was approximately 30% across multiple time points up to and beyond a year; 27% met criteria for depression 3-6 months from injury; 32% at 6-12 months; and 33% beyond 12 months.

MIXED ANXIETY AND DEPRESSION[41]:

Post traumatic states with mixed anxiety and depression represent the commonest sequalae of head injury. Minor depression and states of tension and anxiety are frequently accompanied by irritability, phobia and social avoidance. Irritability is among the most common of emotional consequences of injury and show strong association with depression and anxiety.

MANIA:

Significant association between mania and the presence of posttraumatic seizures, predominantly of the partial complex type (temporal lobe epilepsy). There was no association, however, with family history of bipolar disorder among first-degree relatives. severity of brain injury are major determinants of recovery from TBI.Mood disorders have an independent deleterious effect on the recovery of TBI patients.

ANXIETY:

Anxiety disorders[13] are common in patients with TBI and range in frequency from 11%–70%[51,52]. All variants of anxiety disorders are seen like generalized anxiety disorder, panic disorder, phobic disorders, posttraumatic stress disorder, and obsessive–compulsive disorder. TBI patients often have generalized free-floating anxiety associated with persistent worry, tension, and fearfulness[53]. Panic disorder was the second most common diagnosis after depressive episode, and it was associated with other psychiatric illness like alcohol dependence syndrome and depressive episode. Increased activity of the

aminergic system and decreased activity of the GABA inhibitory network is the proposed mechanism[54]. Right-hemispheric lesions are more often associated with anxiety disorder than left-sided lesions[55]. Antidepressants such as SSRIs, opioid antagonists such as naltrexone[56], and buspirone[57] are promising in the treatment of anxiety disorders. Benzodiazepines[58] and antipsychotics[59] are to be avoided as they cause memory impairment, disinhibition and delayed neuronal recovery.

PSYCHOSIS:

Psychosis[49] secondary to head injury (PSTHI) occurred in 4% to 8.9% of individuals who sustained head trauma. Although rare, PSTHI got interest by clinicians and neuroscientists especially for three reasons:

1) There is usually a latency between the head injury and presentation of psychotic symptoms, thus the appearance of psychosis is often unexpected and puzzling;

2) There are diagnostic issues as some people who develop PSTHI have family histories of psychotic disorder, while many others do not; and

3) The disorder has conceptual relevance to understanding schizophrenia spectrum disorders. The mean latencies between head injury and onset of psychosis is between four to five years, but can range from a few days to over 20 years. Despite the wide range of latencies, studies suggest that about half of patients with PSTHI demonstrate symptoms

within the first year, and roughly 72% of patients had symptoms within the first five years[60].

Although PSTHI has been associated with many forms of delusions, including grandiose, referential, religious and Schneiderian symptoms, the most common symptoms are paranoid or persecutory delusions that are present in up to 80% of all patients. About 60% to 93% of patients presenting with auditory halucination. Visual hallucinations, negative symptoms and formal thought disorder are relatively rare, occurring in 8% to 32%, 15% to 22.2%, and 4.4% of patients, respectively[60,61]

Psychotic symptoms[13] are not uncommon in TBI patients. 0.7%–9.8 % of patients with TBI develop schizophrenia-like psychosis without a family history of schizophrenia[62]. Psychotic symptoms following TBI present as frank delusions, hallucinations, and illogical thinking. They are associated with symptoms of agitation, silly giggling, ideas of reference, grimacing, expression of odd ideas, regression, and impulsive aggressiveness. The psychotic features may be acute or chronic, transient or persistent, and may or may not be associated with mood disturbances. Both right[63] and left hemispheres[64] have been implicated in the genesis of psychotic symptoms. It is important to remember that psychosis is a symptom and not a diagnosis or etiology.

A rational approach[13] based on our knowledge of the neuropathology of TBI must be applied when choosing treatment options. For instance, when there is a suggestion of left-temporal involvement, there may be benefit from

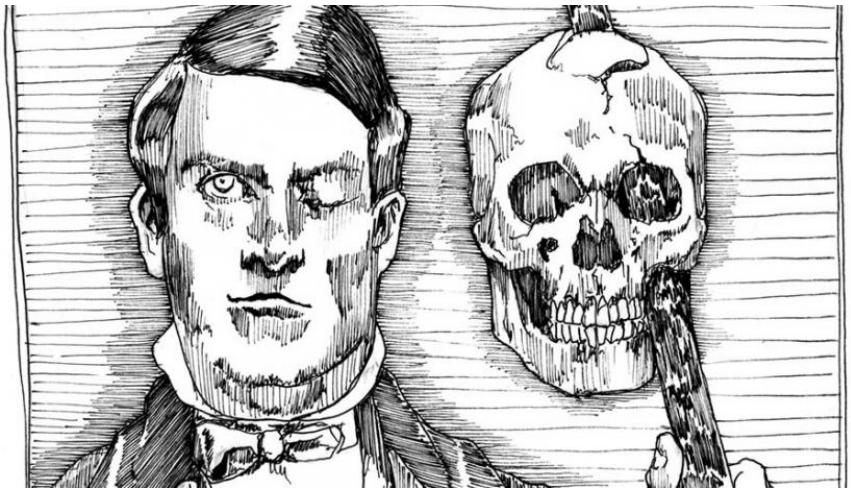
the use of an anticonvulsant. Delusional-type symptoms that seem more related to cognitive and behavioural impairments from frontal lobe dysfunction can benefit from dopaminergics. Neuroleptics, if administered, should be given in low doses, as animal studies showed impaired neuronal recovery[65,66]. PSTHI [50]occured in 4‑8.9% of individuals who sustained TBI. The mean latencies between TBI and onset of psychosis are between four to five years, but can range from a few days to over 20 years.

Despite the wide period of latencies, about half of patients with PSTHI demonstrate symptoms within the first year and roughly 72% of patients have symptoms within the first 5 years[67]. Duration of less than 1 year since injury have been associated with diffuse injuries, paranoid symptoms and visual hallucinations. Patients with longer latencies before the onset of symptoms were found to have localized damage to the temporal lobe and presence of epilepsy. Academic or vocational deterioration and social withdrawal were reported by roughly one third of the patients[67.68]. The most common symptom of PSTHI is persecutory delusions seen in up to 80% of all patients.

Auditory hallucinations are also common, present in 60-93% of patients. Visual hallucinations occurred in 8-32% of patients, negative symptoms in 15-22.2% and formal thought disorder in 4.4%[67,68] .Impairments in memory and executive functioning are Consistently found. Localization data consistently report abnormalities in the temporal areas[67,69], and less commonly, in the frontal lobes[69,70].

There is no consistent finding for hemispheric laterality of lesions. About 0.7‑9.8% of patients with TBI develop schizophrenia like psychosis[69]. childhood head injury might play a role in the expression of schizophrenia in families with a strong genetic predisposition.

PERSONALITY CHANGES:



In 1848, when Gage was just 25 years old, he sustained a terrible injury to his brain. His miraculous survival, and the effects of the injury upon his character, made Gage a curiosity to the public and an important case study for scientists hoping to understand more about the brain. The damage to Gage's frontal cortex caused by the iron rod seems to have resulted in a loss of social inhibitions. It indicates that the accident damaged the connections between the frontal cortex to the limbic system, which are involved in the regulation of emotions

Some localization of personality change[49] due to frontal lobe injury has been reported. Luria describes two main variants of frontally-mediated changes in personality and emotions:

(i) Pseudopsychopathic seen with lesions in the orbitobasal regions of the frontal lobes manifesting with euphoria, impulsiveness and inadequate actions, on a background of disinhibition, and

(ii) Pseudodepressed after lesions of the convexity regions of the frontal lobes manifesting with narrowing of interests and generalised emotional indifference, on a background of general inhibition and torpidity. Medial frontal syndrome is characterized by akinesia, sparse verbal output and incontinence.

Clinically a mixture of these syndromes is more commonly seen. Behavioural sequelae are very common and largely account for the distress to care givers much more than physical disabilities. A greater severity of behavioural sequelae is found in patients who have an abnormal EEG or compressed ventricles on early CT scans indicating significant brain edema.

Behavioural sequelae are known to correlate with severity of injury, extent and degree of disturbed behaviour during recovery, though again such relationship is not linear. Commonly reported changes include excessive tiredness, indifference, concentration and attention disorders, inflexibility, tendency towards perseveration, absence of ability to anticipate, behavioural disinhibition, irritability, a change in quality of relationship with more shallowness, and obsessive‑compulsive symptoms. van Reekum[71] reported that avoidant, borderline, and narcissistic personality disorders were the most common. The numbers however, are very low. In 60 patients assessed 30 years

after TBI 23.3% had at least one personality disorder. The most prevalent were avoidant (15.0%), organic (15.0%), paranoid (8.3%), and schizoid (6.7%) personality disorders[72].

Some localization of personality change due to frontal lobe injury been reported. Damage to the dorsolateral prefrontal cortex and its circuitry impairs executive functions such as working memory, decision making, problem solving and mental flexibility.

Damage to the orbitofrontal cortex and related nodal points impairs intuitive reflexive social behaviours and the capacity to self‐monitor and self‐correct in real time within a social context.

Damage to anterior cingulate and related circuitry impairs motivated and reward‐related behaviours. Damage to medial temporal regions impairs other aspects of memory and the smooth integration of emotional memory with current experience and real‐time assessment of stimulus salience. The frontal‐subcortical circuits responsible for these critical domains of higher intellectual function and empathic, motivated, nuanced human behaviour are highly vulnerable to injury in the typical TBI[17].

OBSESSIVE COMPULSIVE DISORDER:

Rigidity of thinking, obsessions, and ritualistic compulsions onset of obsessive–compulsive symptoms[2], however, is usually preceded by generalized

anxiety. Prevalence of (OCD) - 2 to 4 percent fronto-temporal cortex, caudate nucleus.

SUBSTANCE MISUSE:

Substance misuse[2] is one of the major risk factors and also act as a major determinant of the clinical and psychosocial outcome of TBI patient frequency of alcohol abuse and associated disorders decreased immediately after TBI but increased again at 1-year follow-up, although but not to preinjury levels. Although alcohol use can be significantly decreased during the first year after injury, about 25 percent of the patients reported alcohol-related problems combined effects of traumatic injury and the toxicity of chronic substance abuse (particularly alcohol) might produce high disruption of the neural circuits involved in mood regulation, motivation,and reward processing.

APATHY:

It denotes loss of motivation, initiation, interest and emotional responsiveness. Prevalence is 1/5 to 3/5 of TBI. It is not associated with low mood or sadness[2].

DISSOCIATIVE (CONVERSION) DISORDERS:

Dissociative (conversion) symptoms[50] including fits, dissociative fugues, dissociative amnesia, Ganser states, paralysis, anesthesia, and disturbance of speech, sight or hearing are common. A neurasthenic reaction may incapacitate the patient for months or even years.

SEXUALITY:

Sexual dysfunction[50] is a major ongoing problem in the TBI population, affecting 40‑60% of individuals. Limbic structures particularly amygdala, septal nuclei, hypothalamus, and various cortical areas which form part of the neuroanatomic and physiologic substrate of human sexual behavior can be damaged in TBI, resulting in impaired sexuality.

TREATMENT OF NEUROPSYCHIARIC SEQUALAE[13]

PSYCHOSTIMULANTS

Methylphenidate and dextroamphetamine are the commonly used psychostimulant which act by increasing catecholamine activity by blocking the reuptake of norepinephrine and dopamine[73]. Side effects includes paranoia, dysphoria, agitation, and irritability.

Anecdotal reports have demonstrated the efficacy of psychostimulants in the treatment of inattention, distractibility, disorganization, hyperactivity, impulsivity, hypoarousal, apathy, and hypersomnia[74,75,76].

DOPAMINERGIC AGONISTS

TBI is frequently associated with disturbances of dopamine transmission, which persists for many years after injury[77]**.** Amantadine was first used in the 1960s for treatment of influenza and was later found to have antiparkinsonian actions[78]. It enhances release of dopamine, inhibits reuptake, and increases activity at the postsynaptic receptors[79].

Levadopa and bromocriptine are both dopamine agonists. They have been studied in small, uncontrolled case studies and have been found to be effective in the treatment of mood, cognition, and behavior[80,81].

ANTIDEPRESSANTS

SSRIs are useful in the treatment of depression, mood lability and impulsivity[82].

ANTICONVULSANTS

Prophylactic use of AED – first 7 days reduces risk of seizures in severe TBI.USE beyond 7 days /mild to moderate injuries – avoided .Interferes with neuroplasticity

The role of anticonvulsants in the treatment of neuropsychiatric sequelae of TBI are multiple. They are used to treat seizure disorder, mood lability, mania, impulsivity, aggression and rage[83,84] Other Agents Studies have shown that naltrexone, an opioid antagonist, in doses of 50 mg–100 mg/day may be useful in treating self-injurious behavior[85].

Post-traumatic headache - [2]Valproate, Propranolol or Amitriptyline and Sumatriptan are useful. Non-addictive analgesics and Ergotamine preparations can be tried in vascular headache.Antidepressants can sometimes produce results.

Management of Insomnia - For nocturnal insomnia, good sleep hygiene measures helps. Treatment with hypnotics like benzodiazepine to be given

cautiously. The non-benzodiazepine hypnotics like zopiclone, zolpidem are probably a better starting point. Sedative antidepressants may be considered, possibly trazodone

REHABILITATION AND PSYCHOLOGICALTHERAPIES[2]:

Early Rehabilitation (Severe Injury) – To encourage physical activity and the value of early mobilisation recognised. Graduated exercises and games help to restore the patient's physical self-confidence.

Rehabilitation for Neurological Sequelae - The main areas that require evaluation are improving mobility, upper extremity function and improving communication. Hemiparesis, paraparesis or ataxia of gait requires physiotherapy. Occupational therapy has a special place in restoring useful function to the upper limbs. Speech therapy has role for the resolution of dysphasia or articulation difficulty.

Rehabilitation for Cognitive Impairment - The programme must be graded, with goals at any stage that are realisable, rational and acceptable to the patient. Self-esteem is to be boostered.

Components of Rehabilitation Programmes - These include assessment, formulation of plan, implementation of plan, review and modification of plan and discharge arrangement.

Core rehabilitation Team – It should include neurologists, psychiatrists, rehabilitation physicians, clinical psychologists, physiotherapists, occupational therapists, speech therapists and psychiatric social workers.

INVESTIGATIONS:

1. **Neuroimaging** - Computed Tomography (CT), magnetic resonance imaging (MRI), Functional MRI (fMRI), SPECT, Positron Emission Tomography (PET) (to show the level of cerebral metabolic activity)magnetic resonance spectroscopy (MRS) (to show the areas of cerebral neuronal loss and diffusion tensor imaging and fiber tractography (DTI/FT) which shows cerebral white matter tracts.

2. **Electrophysiological** - Electroencephalography (EEG) is used to assess for post traumatic epilepsy or encephalopathy. Quantitative EEG (QEEG) is sometimes used for slow wave abnormality following TBI and post traumatic temporal lobe epilepsy.

   Evoked response potentials (ERPs) and Video EEG monitoring or 24hrs ambulatory EEG is also useful.

   Polysomnography is helpful to assess atypical sleep disturbances following TBI like atypical night terror, sleep apnoea, nocturnal myoclonus.

3. **Neuropsychological Testing** : to assess cognitive functioning, language testing, tests of motivation and malingering, tests for premorbid functioning.

4. **Blood Biochemistry**

   Serum Electrolytes

   $Na+$, $K+$, Mg_2+, $Cl-$

Neuronal Protein – include both neuronal (e.g., neuron-specific enolase, creatine kinase-BB, cleaved Tau protein) and glial (e.g., myelin basic protein, S-100B) proteins. Most sensitive serum marker is S-100B

Neuro Endocrine Assessment – for pituitary hormone deficiencies, Diabetes insipidus, syndrome of growth hormone deficiency (GHD), central hypothyroidism, low cortisol levels and decreased gonadotropin secretion (e.g., history of alcohol and/or drug abuse, personality disorders), premorbid social functioning levels, the quality and extent of rehabilitation services and the availability of social and vocational support.

PROGNOSIS AND OUTCOME:

The long-term outcome of TBI patients is primarily related to severity of brain injury, type and location of intracranial lesion, patients' age, efficacy of acute medical and surgical treatment, socioeconomic status, educational level, previous psychiatric disorders

AIMS AND OBJECTIVES

1. To assess the prevalence of psychiatric disorders in patients with traumatic brain injury

2. To assess its correlation with radiological finding

MATERIALS & METHODS

SUBJECTS-100

STUDY PLACE-Department of neurosurgery, Chennai

DURATION-6months from the date of approval of ethical committee

STUDY DESIGN: CROSS SECTIONAL STUDY

INCLUSION CRITERIA

1. Patients attending outpatient and inpatient in neurosurgery department with history of head trauma
2. Patient showing radiological signs of brain damage through trauma
3. age >18 years
4. both men and women

EXCLUSION CRITERIA

1. Psychiatric illness before brain injury
2. Neurological disorders before brain injury
3. Patient having gross cognitive deficits making interview difficult

DATA COLLECTION AND METHODS

Patients attending outpatient and inpatient department of neurosurgery with history of traumatic brain damage confirmed through radiological finding are assessed cross-sectionally with **MINI international neuropsychiatric**

interview to screen for psychiatric disorders initially. Detailed history take from the patient regarding the illness**.** Then psychiatric morbidity is assessed using specific rating scales like

Hamilton rating scale for depression (HAM-D)

Hamilton rating scale for anxiety (HAM-A)

Positive and negative syndrome scale(PANSS)for psychotic disorders

DSM V and ICD-10

Then the findings are correlated with the radiological findings of the patient to know which region of the brain affected.

SEVERITY OF HEAD INJURY ASSESSED FROM GCS (glassgow coma scale)

EYE OPENING:

1-no eye opening

2-to pain

3-to speech

4-spontaneously

VERBAL RESPONSE:

1-None

2-incomprehensible sounds

3-inappropriate words

4-patient confused

5-oriented

MOTOR RESPONSE:

1-none

2-extensor response

3-flexion to painful stimulus

4-withdraws from pain

5-localises to painful stimulus

6-obeys commands

TOTAL SCORE

13-15-Mild

8-13-moderate

<8-severe

Positive and Negative Syndrome Scale

The Positive and Negative Syndrome Scale (PANSS) was developed in the late 1980s for the assessment of positive and negative symptoms of schizophrenia and other psychotic disorders.

The PANSS includes 30 items on three subscales: seven items covering positive symptoms, seven covering negative symptoms, and 16 covering general psychopathology.

Each item is scored on a seven-point, item-specific Likert scale with range from 1 to 7; thus, the positive and negative subscales each range from 7 to 49, and the general psychopathology scale ranges from 16 to 112 Its high

reliability and good coverage of both positive and negative symptoms make it excellent for this purpose.

Hamilton Rating Scale for Depression

The HAM-D was developed in the early 1960s to monitor the severity of major depression, with a focus on somatic symptomatology. The version in most common use has 17 items, although versions with different numbers of items are used. Items on the HAM-D are scored from 0 to 2 or from 0 to 4, with total score ranging from 0 to 50.

- Scores of 7 or less may be considered normal;
- 8 to 13 mild;
- 14 to 18, moderate;
- 19 to 22, severe; and
- 23 and above, very severe.

Hamilton Anxiety Rating Scale

The HAM-A was developed in the late 1950's to assess anxiety symptoms. There are 14 items, each of which is rated from 0 to 4 , with the total score ranging from 0 to 56.

A score of 14 has been suggested as the threshold for clinically significant anxiety.

14-17-mild anxiety

18-24-moderate anxiety

>25-severe anxiety

F06.1 Organic catatonic disorder

"A disorder of diminished (stupor) or increased (excitement) psychomotor activity associated with catatonic symptoms. The extremes of psychomotor disturbance may alternate. It is not known whether the full range of catatonic disturbances described in schizophrenia occurs in such organic states, nor has it been conclusively determined whether an organic catatonic state may occur in clear consciousness or whether it is always a manifestation of delirium, with subsequent partial or total amnesia. This calls for caution in making this diagnosis and for a careful delimitation of the condition from delirium. Encephalitis and carbon monoxide poisoning are presumed to be associated with this syndrome more often than other organic causes.

Diagnostic guidelines

The general criteria for assuming organic etiology, laid down in the introduction to F06, must be met. In addition, there should be one of the following:

a) Stupor (diminution or complete absence of spontaneous movement with partial or complete mutism, negativism, and rigid posturing);

b) Excitement (gross hypermotility with or without a tendency to assaultiveness);

c) Both (shifting rapidly and unpredictably from hypo- to hyperactivity).

Other catatonic phenomena that increase confidence in the diagnosis are:

- Stereotypies,
- Waxy flexibility, and
- Impulsive acts"

F07.0 Organic personality disorder

"This disorder is characterized by a significant alteration of the habitual patterns of premorbid behaviour. The expression of emotions, needs, and impulses is particularly affected. Cognitive functions may be defective mainly or even exclusively in the areas of planning and anticipating the likely personal and social consequences, as in the so called frontal lobe syndrome. However, it is now known that this syndrome occurs not only with frontal lobe lesions but also with lesions to other circumscribed areas of the brain.

Diagnostic guidelines

In addition to an established history or other evidence of brain disease, damage, or dysfunction, a definitive diagnosis requires the presence of two or more of the following features:

a. Consistently reduced ability to persevere with goal-directed activities, especially those involving longer periods of time and postponed gratification;

b. Altered emotional behavior, characterized by emotional lability, shallow and unwarranted cheerfulness (euphoria, inappropriate jocularity), and easy change to irritability or short-lived outbursts of anger and aggression; in some instances apathy may be a more prominent feature;

c. Expression of needs and impulses without consideration of consequences or social convention (the patient may engage in dissocial acts, such as stealing, inappropriate sexual advances, or voracious eating, or may exhibit disregard for personal hygiene);

d. Cognitive disturbances, in the form of suspiciousness or paranoid ideation, and/or excessive preoccupation with a single, usually abstract, theme (e.g. religion, "right" and "wrong");

e. Marked alteration of the rate and flow of language production, with features such as circumstantiality, over-inclusiveness, viscosity, and hypergraphia;

f. Altered sexual behavior (hyposexuality or change of sexual preference).

Includes:

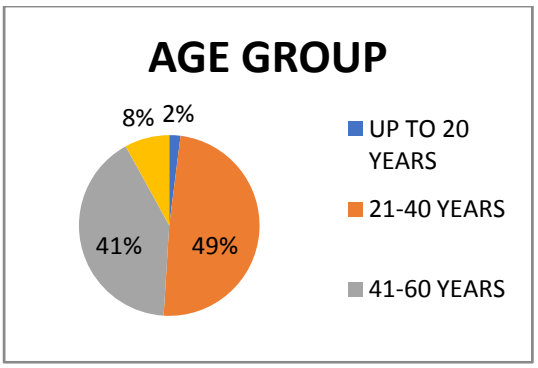
- frontal lobe syndrome
- limbic epilepsy personality syndrome
- lobotomy syndrome
- organic pseudopsychopathic personality
- organic pseudoretarded personality
- postleucotomy syndrome”

RESULTS

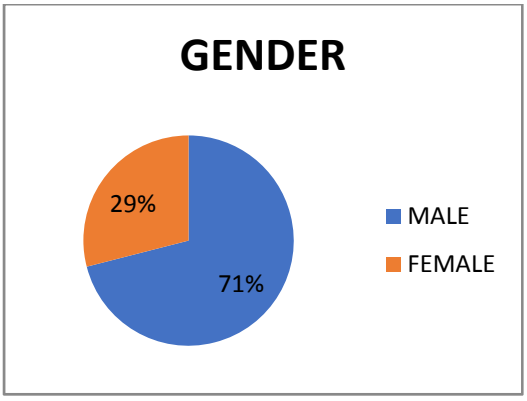
The protocol was approved by institutional ethics committee. Written informed consent was obtained from both the patients and the accompanying person. After applying the inclusion and exclusion criteria, total of 100 patients were taken by random sampling. Socio demographic details were obtained initially. They were assessed for the presence of psychiatric illness with MINI plus scale and when they fit into any of the psychiatric complaint scales were given to assess the severity of illness.

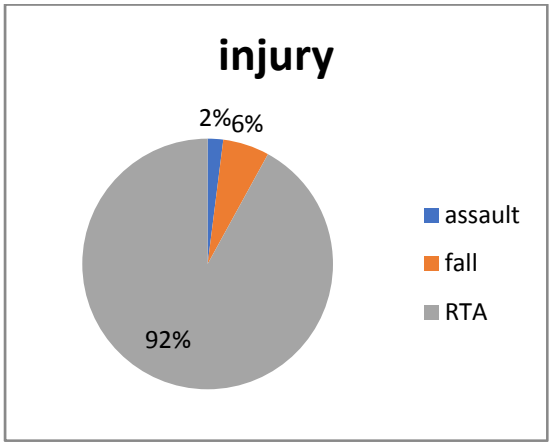
SOCIO-DEMOGRAPHIC CHARACTERISTICS

There was no significant difference among age, sex, socioeconomic background, marital status, type of injury among the patients.

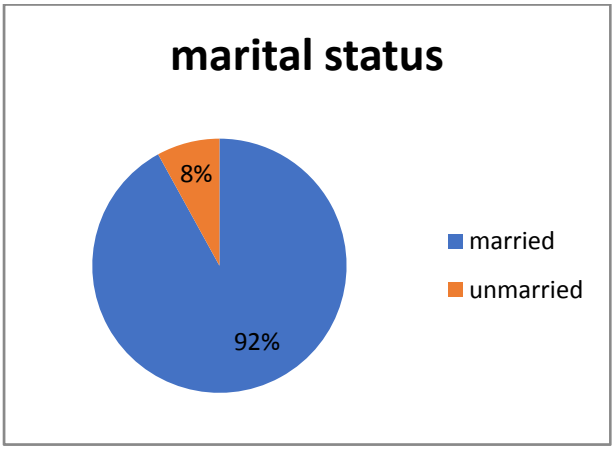


Patients from age group 18-60 years were taken and 71 male patients and 29 female patients were taken. Most of our patients had brain injury through road traffic accidents (92%) and the rest by accidental fall (6%) or assault(2%).

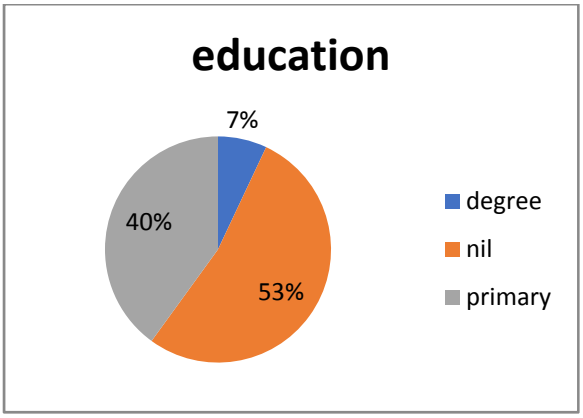




Among them, 3 patients had a positive family history of psychiatric illness.2 0% patients had substance use disorders. All belong to lower socioeconomic status.



92% of the persons were married and 8% unmarried

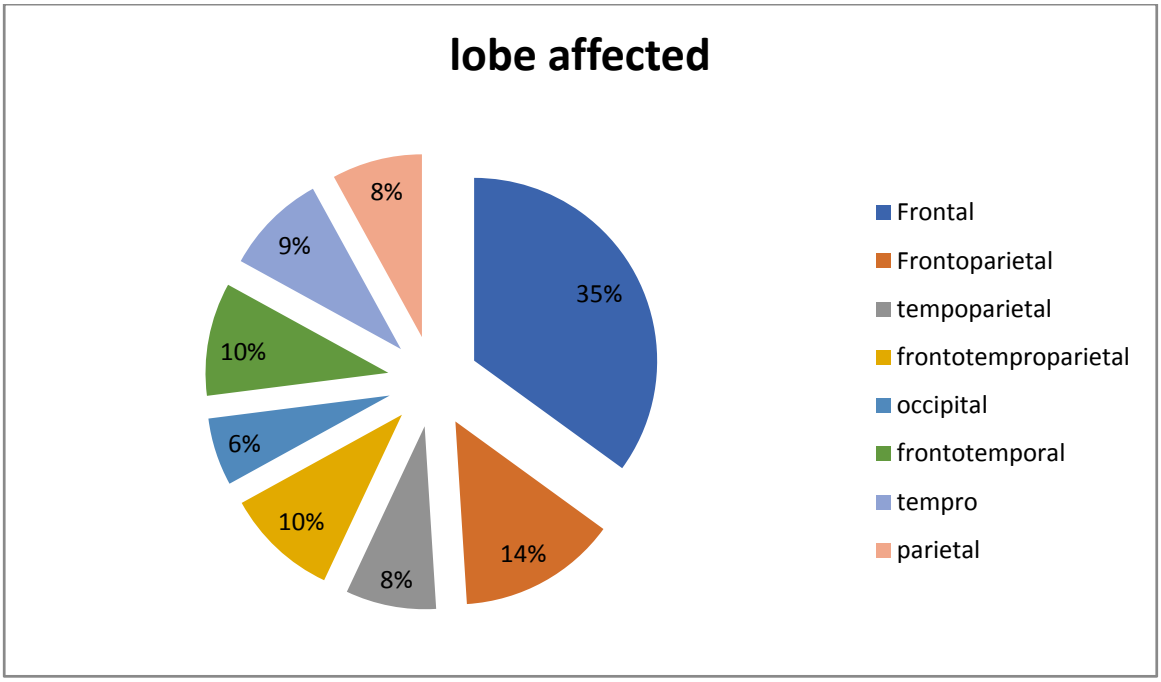


40% had primary school of education and 7% had completed their degree

DURATION SINCE INJURY TO ILLNESS

Duration	Frequency	Percent
Less than 6 months	82	82.0
6months to 1year	7	7.0
1-3 years	9	9.0
More than 3years	2	2.0
Total	100	100.0

Most of the patients were interviewed in acute phase following injury within 6 months both during patient admission or during follow-up. There were patients who had been in treatment for months and during follow-up found to have comorbid psychiatric illness and referred for psychiatric consultation.



Most of the patients have contusion or haemorrhage involving the frontal lobe(35%) next is frontoparietal(14%), frontotemperoparietal(10%) frontotemporal(10%), temperoparietal(8%), occipital(6%), temporal(9%) and parietal(8%).

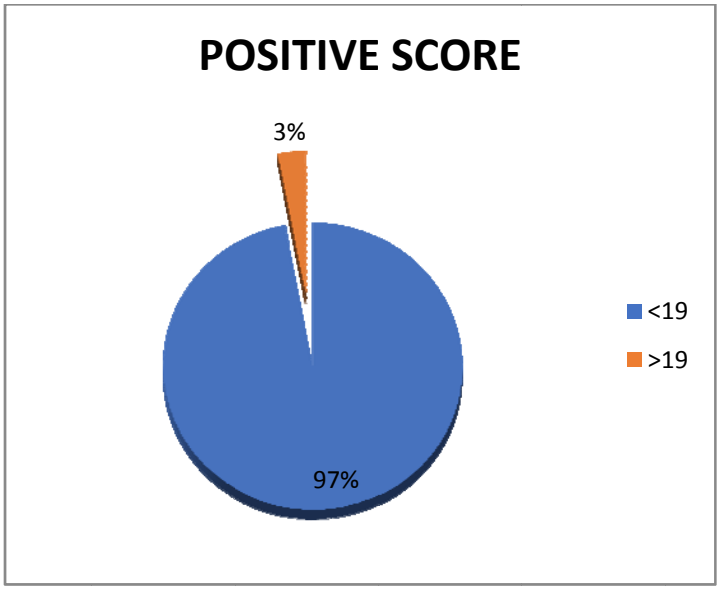
SIDE OF BRAIN AFFECTED:

Side of brain affected	Frequency	Percent
Bilateral	12	12.0
Left	47	47.0
Right	41	41.0
Total	100	100.0

Left hemispheric lesions was found in 47% of patients followed by right sided lesions in 41% and bilateral lesions was found in 12% of patients.

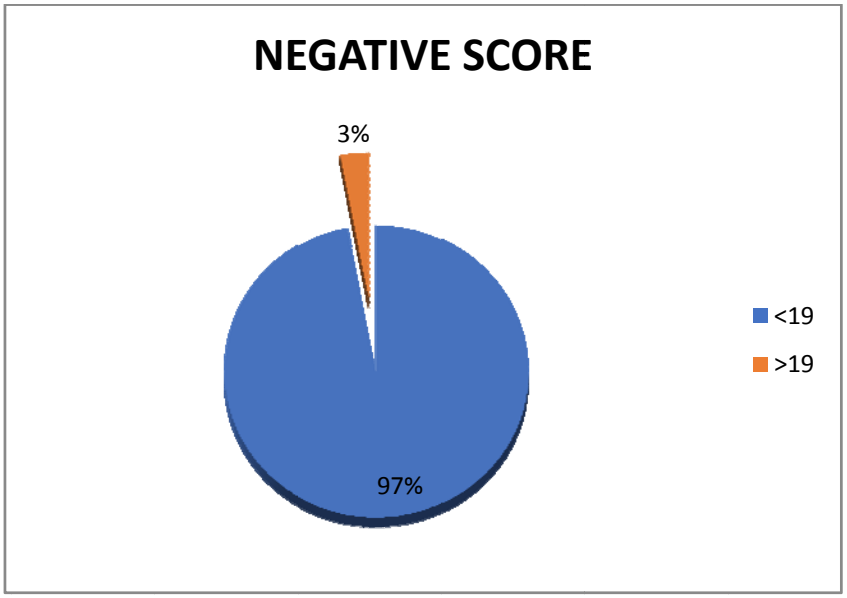
PREVALENCE OF PSYCHIATRIC SYMPTOMS

POSITIVE SYMPTOMS OF PSYCHOSIS



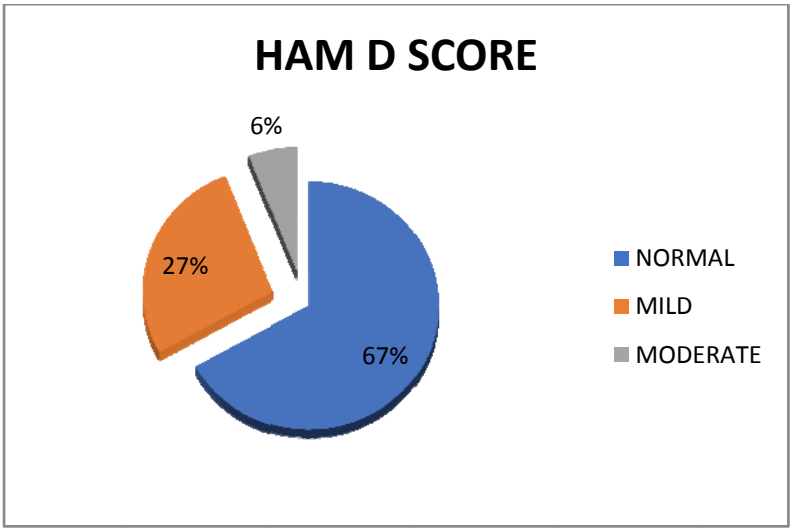
3% were exhibiting positive symptoms of psychosis as per PANSS score not satisfying the criteria for schizophrenia.

NEGATIVE SYMPTOMS



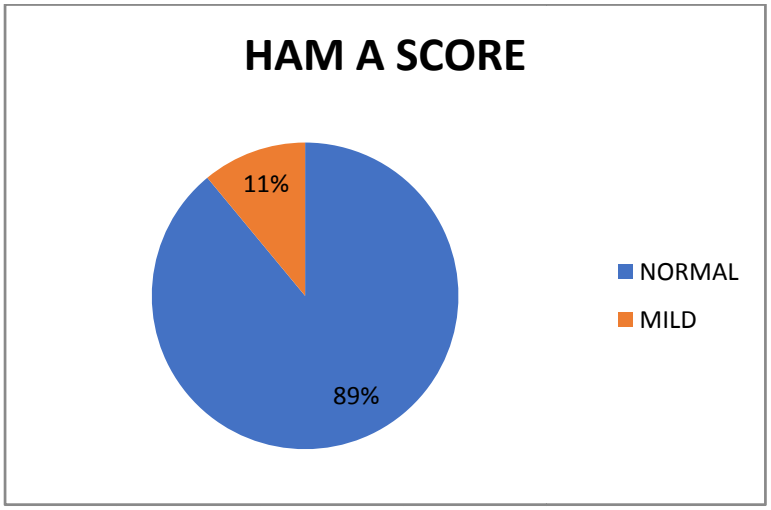
3% were having negative symptoms in significant scores.

DEPRESSION



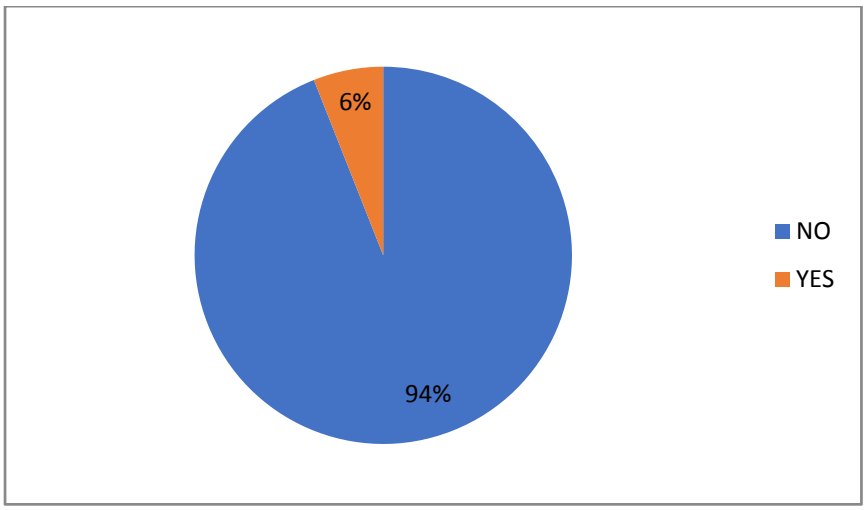
33% patients were having depressive symptoms in significant score with 27% were having mild symptoms and 6% were having moderate symptoms.

ANXIETY



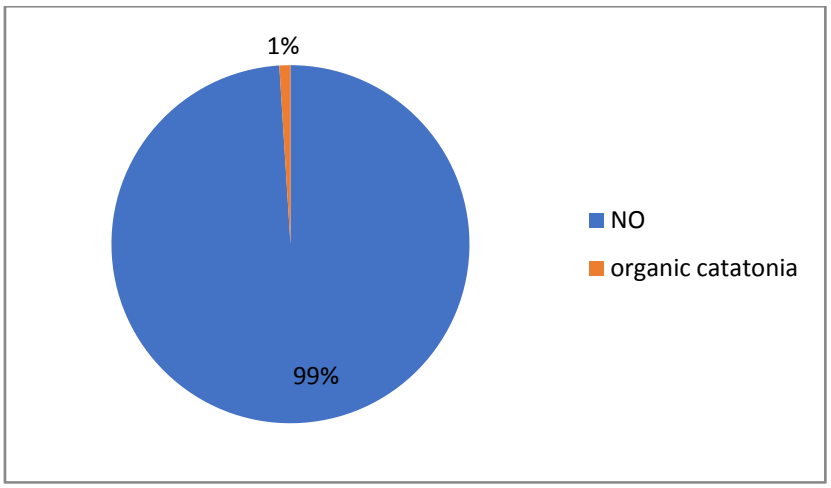
11% were having anxiety symptoms with mild severity.

PERSONALITY CHANGES



6% patients were having disinhibition, behavioural disturbances not in touch with their premorbid personality and satisfied the criteria of organic personality disorder as per ICD-10 criteria.

ORGANIC CATATONIA



1% patient were satisfying the criteria for organic catatonia as per ICD-10 criteria, onset of symptoms with temporal relationship with head injury.

OVERALL PREVALENCE OF PSYCHIATRIC MORBIDITY:

		Count	Table N %
POSITIVE_SCORE	<19	97	97.0%
	>19	3	3.0%
NEGATIVE_SCORE	<19	97	97.0%
	>19	3	3.0%
HAM_D_SCORE	NORMAL	67	67.0%
	MILD	27	27.0%
	MODERATE	6	6.0%
	SEVERE	0	.0%
HAM_A_SCORE	NORMAL	89	89.0%
	MILD	11	11.0%
	MODERATE	0	.0%
	SEVERE	0	.0%
PERSONALITY CHANGES	NO	94	94%
	ORGANIC PERSONALITY	6	6%
ICD_10	NO	99	99.0%
	ORGANIC CATATONIA	1	1.0%

Table 1.1-COMPARISON BETWEEN DURATION AND POSITIVE SYMPTOMS

			Duration				
			<6 months	**6-12 months**	**1-3 years**	**above 3 years**	**Total**
POSITIVE SCORE	<19	Count	79	7	9	2	97
		% within duration	96.3%	100.0%	100.0%	100.0%	97.0%
	>19	Count	3	0	0	0	3
		% within duration	3.7%	.0%	.0%	.0%	3.0%
Total		Count	82	7	9	2	100
		% within duration	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=0.679 p= 0.878

No significant difference was found between the duration of illness and the positive symptoms of psychosis.

Table 1.2: COMPARISON BETWEEN DURATION AND NEGATIVE SYMPTOMS

			Duration				
			<6 months	**6-12 months**	**1-3 years**	**above 3 years**	**Total**
NEGATIVE SCORE	<19	Count	80	7	8	2	97
		% within duration	97.6%	100.0%	88.9%	100.0%	97.0%
	>19	Count	2	0	1	0	3
		% within duration	2.4%	.0%	11.1%	.0%	3.0%
Total		Count	82	7	9	2	100
		% within duration	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=2.402

p= 0.493

Among 100 patients 3 had a score >19 for negative symptoms and there was no significant difference between duration and negative symptoms.

Table 1.3:COMPARISON BETWEEN DURATION AND DEPRESSION SCORE

			<6 months	6-12 months	1-3 years	above 3 years	Total
HAM_D SCORE	NORMAL	Count	52	6	7	2	67
		% within duration	63.4%	85.7%	77.8%	100.0 %	67.0%
	MILD	Count	24	1	2	0	27
		% within duration	29.3%	14.3%	22.2%	.0%	27.0%
	MODERATE	Count	6	0	0	0	6
		% within duration	7.3%	.0%	.0%	.0%	6.0%
Total		Count	82	7	9	2	100
		% within duration	100.0 %	100.0 %	100.0 %	100.0 %	100.0 %

Pearson Chi-Square= 3.513 p= 0.742.

Among 100, 33 patients satisfied the criteria for depression and 27 were having mild symptoms and 6 were having moderate symptoms. No significant difference was found between duration since injury and depression score.

Table1.4: COMPARISON BETWEEN DURATION AND ANXIETY SYMPTOMS

			duration				Total
			<6 months	**6-12 months**	**1-3 years**	**above 3 years**	
HAM_A SCORE	NORMAL	Count	77	1	9	2	89
		% within duration	93.9%	14.3%	100.0%	100.0%	89.0%
	MILD	Count	5	6	0	0	11
		% within duration	6.1%	85.7%	.0%	.0%	11.0%
Total		Count	82	7	9	2	100
		% within duration	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=43.286**

p< 0.001

11 were having anxiety features and it was seen more during the initial period up to 6 months following injury.

TABLE 1.5-COMPARISON BETWEEM DURATION AND PERSONALITY CHANGES (ORGANIC PERSONALITY DISORDER)

Crosstab							
			duration				Total
			<6 months	6-12 months	1-3 years	above 3 years	
Personality changes		Count	80	6	6	2	94
		% within duration	97.6%	85.7%	66.7%	100.0%	94.0%
	Yes	Count	2	1	3	0	6
		% within duration	2.4%	14.3%	33.3%	.0%	6.0%
Total		Count	82	7	9	2	100
		% within duration	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=14.745**

p< 0.0012

Significant differences was found between duration since injury and personality changes with 2 cases in first 6months after injury and 1 case after 6months and 3 cases was diagnosed 1 year after injury.

TABLE 1.6-COMPARSION BETWEEN DURATION AND ORGANIC CATATONIA

Crosstab							
			duration				Total
			<6 months	6-12 months	1-3 years	above 3 years	
ICD_10		Count	82	7	8	2	99
		% within duration	100.0%	100.0%	88.9%	100.0%	99.0%
	organic catatonia	Count	0	0	1	0	1
		% within duration	.0%	.0%	11.1%	.0%	1.0%
Total		Count	82	7	9	2	100
		% within duration	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=10.213*

p= 0.017

Significant difference was found between duration and organic catatonia where the patient had symptoms 1year after the injury.

Table 2.1: COMPARISON BETWEEN SEVERITY OF HEAD INJURY AND POSITIVE SYMPTOMS OF PSYCHOSIS:

			Severity			Total
			mild	moderate	Severe	
POSITIVS SCORE	<19	Count	34	58	5	97
		% within GCS	94.4%	100.0%	83.3%	97.0%
	>19	Count	2	0	1	3
		% within GCS	5.6%	.0%	16.7%	3.0%
Total		Count	36	58	6	100

Pearson Chi-Square=6.453 * p= 0.040

Severity is assessed in our study based on the GCS score during the time of injury and the symptoms. Significant difference was found between patients who had severe injury and showing positive symptoms.

Table 2.2: COMPARISON BETWEEN SEVERITY OF HEADINJURY AND NEGATIVE SYMPTOMS:

			SEVERITY			Total
			MILD	MODERATE	SEVERE	
NEGATIVE SCORE	<19	Count	34	57	6	97
		% within GCS	94.4%	98.3%	100.0%	97.0%
	>19	Count	2	1	0	3
		% within GCS	5.6%	1.7%	.0%	3.0%
Total		Count	36	58	6	100
		% within GCS	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=1.318 p= 0.517

No significant difference between severity of head injury and negative symptoms.

Table 2.3: COMPARISON BETWEEN SEVERITY AND DEPRESSION SCORE:

			SEVERITY			Total
			MILD	MODERATE	SEVERE	
HAM_D SCORE	NORMAL	Count	31	33	3	67
		% within GCS	86.1%	56.9%	50.0%	67.0%
	MILD	Count	5	22	0	27
		% within GCS	13.9%	37.9%	.0%	27.0%
	MODERATE	Count	0	3	3	6
		% within GCS	.0%	5.2%	50.0%	6.0%
Total		Count	36	58	6	100
		% within GCS	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=31.170**

$p < 0.001$

Significant difference was found between the severity of head injury and depressive symptoms of 33 patients with depression 5patients had mild TBI, 25 patients had moderate injury and 3 had severe head injury.

Table 2.4: COMPARISON BETWEEN SEVERITY AND ANXIETY SCORE:

			SEVERITY			
			MILD	**MODERATE**	**SEVERE**	**Total**
HAM_A SCORE	NORMAL	Count	36	50	3	89
		% within GCS	100.0%	86.2%	50.0%	89.0%
	MILD	Count	0	8	3	11
		% within GCS	.0%	13.8%	50.0%	11.0%
Total		Count	36	58	6	100
		% within GCS	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=14.233 p< 0.001**

Similar significant difference was found between severity and anxiety symptoms. Of 11 patients with anxiety symptoms, 8 had moderate head injury and 3 had severe head injury.

Table 2.5: COMPARISON BETWEEN SEVERITY AND

PERSONALITY CHANGE:

			SEVERITY			Total
			MILD	MODERATE	SEVERE	
personality changes		Count	33	55	6	94
		% within GCS	91.7%	94.8%	100.0%	94.0%
	Yes	Count	3	3	0	6
		% within GCS	8.3%	5.2%	.0%	6.0%
Total		Count	36	58	6	100
		% within GCS	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=0.801 p< 0.970.

No significant difference was found between severity and personality change.

TABLE 2.6-COMPARISON BETWEEN SEVERITY OF INJURY AND ORGAIC CATATONIA:

Crosstab

			SEVERITY			
			MILD	**MODERATE**	**SEVERE**	**Total**
ICD_10		Count	36	58	5	99
		% within GCS	100.0%	100.0%	83.3%	99.0%
	organic catatonia	Count	0	0	1	1
		% within GCS	.0%	.0%	16.7%	1.0%
Total		Count	36	58	6	100
		% within GCS	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=15.825**

$p < 0.001$

Significant difference was found between severity and catatonia as the patient had severe head injury.

Table 3.1: COMPARISON BETWEEN LOBE INVOLVED AND POSITIVE SYMPTOMS:

			POSITIVS_SCORE		
			<19	>19	Total
lobe_affected_group	Frontal	Count	34	1	35
		% within lobe_affected_group	97.1%	2.9%	100.0%
	Frontoparietal	Count	14	0	14
		% within lobe_affected_group	100.0%	.0%	100.0%
	Tempoparietal	Count	7	1	8
		% within lobe_affected_group	87.5%	12.5%	100.0%
	Frontotemproparietal	Count	10	0	10
		% within lobe_affected_group	100.0%	.0%	100.0%
	Occipital	Count	6	0	6
		% within lobe_affected_group	100.0%	.0%	100.0%
	Frontotemporal	Count	10	0	10
		% within lobe_affected_group	100.0%	.0%	100.0%
	Tempro	Count	8	1	9
		% within lobe_affected_group	88.9%	11.1%	100.0%
	Parietal	Count	8	0	8
		% within lobe_affected_group	100.0%	.0%	100.0%
Total		Count	97	3	100
		% within lobe_affected_group	97.0%	3.0%	100.0%

Pearson Chi-Square= 6.003 p=0.539

No significant difference was found between lobes affected and positive symptoms. Of 3 patients with positive symptoms, 1 had frontal injury,1 had temporal injury and 1 had temperoparietal injury.

3.2 NEGATIVE SYMPTOMS

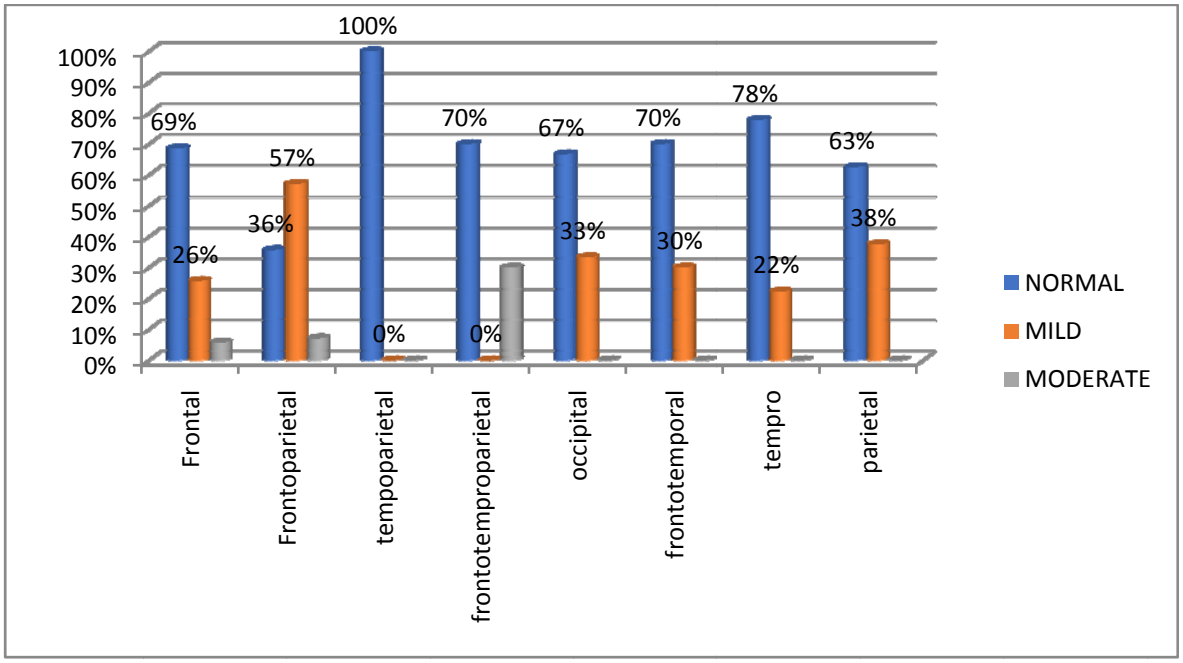
			NEGATIVE_SCORE		
			<19	>19	Total
lobe_affected_group	Frontal	Count	34	1	35
		% within lobe_affected_group	97.1%	2.9%	100.0%
	Frontoparietal	Count	14	0	14
		% within lobe_affected_group	100.0%	.0%	100.0%
	Tempoparietal	Count	8	0	8
		% within lobe_affected_group	100.0%	.0%	100.0%
	Frontotemproparietal	Count	10	0	10
		% within lobe_affected_group	100.0%	.0%	100.0%
	Occipital	Count	6	0	6
		% within lobe_affected_group	100.0%	.0%	100.0%
	Frontotemporal	Count	9	1	10
		% within lobe_affected_group	90.0%	10.0%	100.0%
	Tempro	Count	9	0	9
		% within lobe_affected_group	100.0%	.0%	100.0%
	Parietal	Count	7	1	8
		% within lobe_affected_group	87.5%	12.5%	100.0%
Total		Count	97	3	100
		% within lobe_affected_group	97.0%	3.0%	100.0%

Pearson Chi-Square=5.621

p=0.585

No significant difference between lobe affected and negative symptoms. Of 3 patients with negative symptoms, 1 had frontal injury ,1 had parietal injury,1 had frontotemporal injury.

3.3-DEPRESSION SCORE



Pearson Chi-Square=39.944**

p= 0.005.

Significant difference was found between the brain region affected and depressive symptoms. Patients with frontal, frontoparietal, occipital, frontotemporal, temporal and parietal regions had mild symptoms of depression and persons with frontal, frontoparietal, frontotemporoparietal had moderate symptoms of depression.

3.3. ANXIETY SCORE

4.			HAM_A_SCORE		Total
			NORMAL	MILD	
lobe_affected_group	Frontal	Count	33	2	35
		% within lobe_affected_group	94.3%	5.7%	100.0%
	Frontoparietal	Count	12	2	14
		% within lobe_affected_group	85.7%	14.3%	100.0%
	Tempoparietal	Count	7	1	8
		% within lobe_affected_group	87.5%	12.5%	100.0%
	Frontotemproparietal	Count	6	4	10
		% within lobe_affected_group	60.0%	40.0%	100.0%
	Occipital	Count	5	1	6
		% within lobe_affected_group	83.3%	16.7%	100.0%
	Frontotemporal	Count	10	0	10
		% within lobe_affected_group	100.0%	.0%	100.0%
	Temporal	Count	9	0	9
		% within lobe_affected_group	100.0%	.0%	100.0%
	Parietal	Count	7	1	8
		% within lobe_affected_group	87.5%	12.5%	100.0%
Total		Count	89	11	100
		% within lobe_affected_group	89.0%	11.0%	100.0%

Pearson Chi-Square=12.326

p=0.090

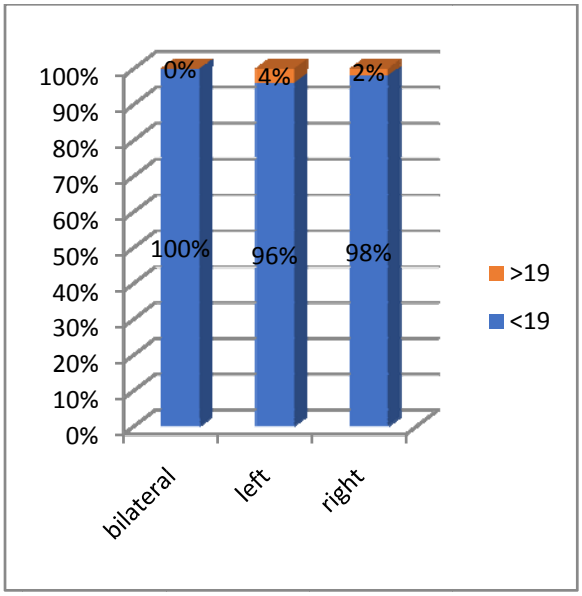
No significant difference was found between specific lobe affected and anxiety symptoms.

No significant difference was found between lobe involved and personality changes and catatonic changes.

TABLE 4. CORRELATION BETWEEN SIDE OF INJURY AND SYMPTOMS:

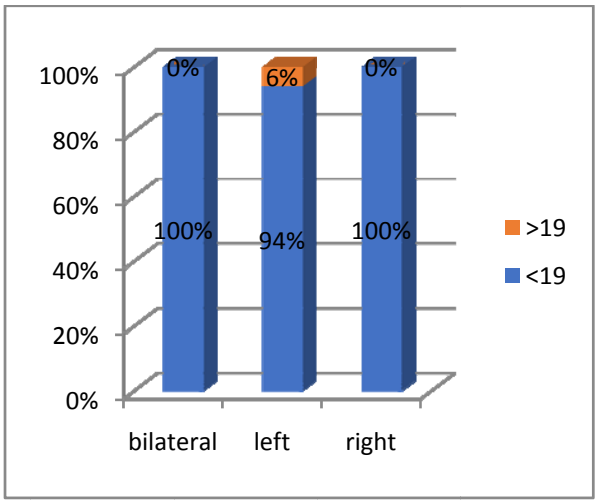
No significance between side affected and positive, negative and anxiety scores

POSITIVE SCORE



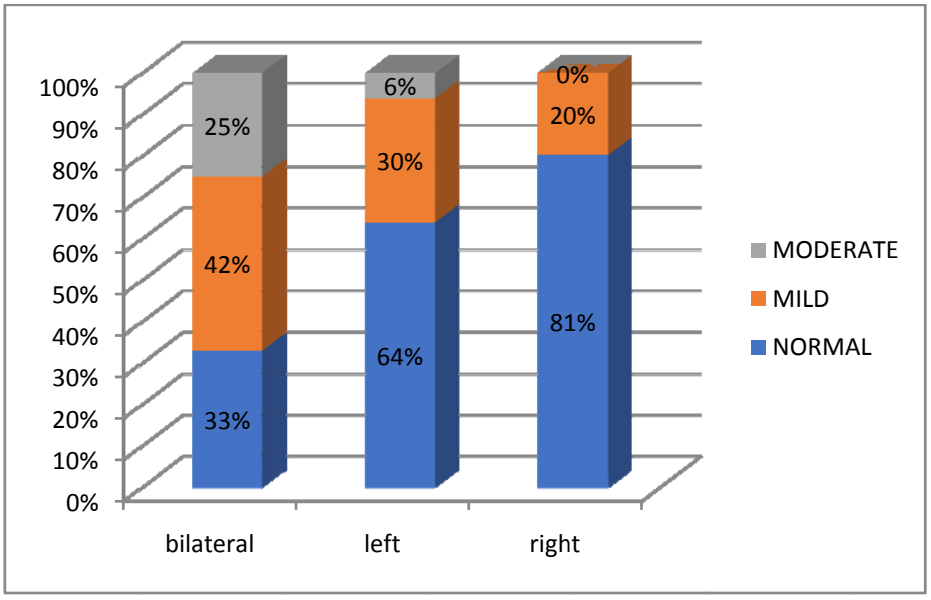
Pearson Chi-Square=0.670 P= 0.715

NEGATIVE SCORE



Pearson Chi-Square=3.488 P= 0.175

DEPRESSION SCORE:

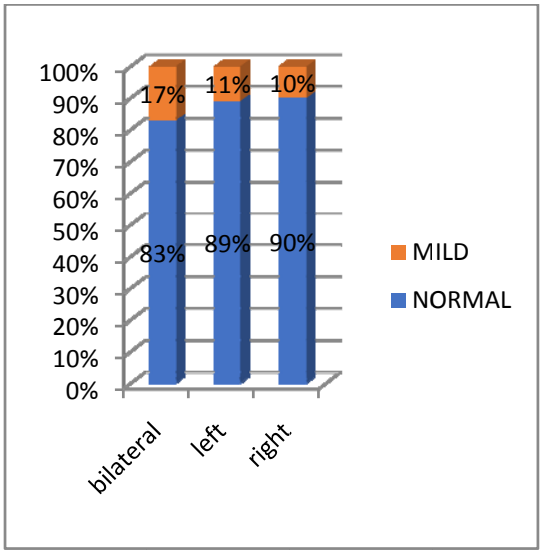


Pearson Chi-Square=14.848**

P= 0.005.

Significant difference was found between side affected and depression score. Of 33 patients with depression,8 had bilateral injury,17 had left side injury and 8 had right side injury.

ANXIETY SCORE



Pearson Chi-Square=0.465 P= 0.793

DISCUSSION

Head injury was defined with the presence of a radiological finding in CT or MRI suggestive of injury with a hemorrhage or contusion or edema or contusion in the brain parenchyma. Like any potentially disabling condition, persons with TBI report a variety of symptoms in different domains, but not all of the symptoms rise to the level of a disorder. Our study dealt with the psychiatric morbidity in head injury patients using validated scales to assess the severity of various symptom dimensions for a disorder. Neurocognitive disturbances was not assessed and our study dealt mainly with common psychiatric diagnosis postinjury.

As much possible, this study inclusion and exclusion ruled out the possibility of past history ,alcohol dependence and other high risk factors for developing the illness premorbidly and took patients with only temporal relationship with head injury. Other than the road traffic accidents it also included injury due to assault and fall. Therefore it assessed brain injury as opposed to head injury .most of the cases taken from inpatients and details of severity of injury was able to be taken from their medical records. All the psychiatric illness was found to have incidence more than the general population. In a cross sectional study like this it was difficult to assess the course of illness over a period of time, which on assessing the course we can be able to assess the symptom reduction or enhancement over the years.

The most common diagnosis in my study is depression after traumatic brain injury. This is one of the study which showed a high prevalence of depression among patients with traumatic brain injury. 33% of patients had significant depressive scores in Hamilton rating scale for depression and others having symptoms like sleep disturbance, fatigue alone not significant enough to satisfy the criteria. Our study also shows significant differences between left side injury and depressive scores which are consistent with the review by Vani et al[6] where left frontal lesions was associated with an increased probability of developing major depression. Insomnia, low mood, fatigability, somatic symptoms were the common presenting complaints. Depressive features can be accounted for by the presence of risk factors like psychosocial stressors, increased age, lack of work and fear of job loss, poor recovery, worsening of a neurological deficit after initial recovery which has not been accounted for in our study.

Our study don't have any incidence of post traumatic manic illness unlike in our previous studies which showed the incidence of mania . Bipolar illness with past history of psychiatric illness before injury was found and no new case of mania detected after injury

There was no significant difference between duration since injury and the depression symptoms unlike other previous studies which show rate of psychiatric illness is more in the first year after injury and subsides over the course of illness. Ours is a cross sectional study which studied only the incidence at a time and not over the course of illness.

The second most common diagnosis was the presence of anxiety symptoms. [5]Anxiety symptoms were more common in the first 6 months post injury. Severity of head injury has also significant difference with anxiety symptoms with 3cases in moderate severity and 8 cases in patients with severe head injury. Literature shows various forms of anxiety like phobia, panic disorder, PTSD common in TBI and range from 11-70% but our study more towards having anxiety symptoms not satisfying to the level of a disorder like panic, phobia or obsessive compulsive disorder. Right hemispheric lesions are more often associated with anxiety but our study showed no significant differences among laterality of lesions.

Personality changes and psychotic (positive and negative)symptoms was present in 6% of patients each .Among patients with psychosis, previous literature shows high incidence of psychosis up to 20% where in our study it has low incidence about 6% (3% had positive symptoms and 3% had negative symptoms as per PANSS scale)where delusions, hostility, apathy, emotional withdrawal, lack of spontaneity predominates. Our study had significant findings between severity and positive symptoms like other review studies with 2% patients with positive and 2%patients with negative symptoms was found in patients with mild severity and 1%negative symptoms in patient with moderate severity and 1% positive symptom in patient with severe head injury. No consistent finding for hemispheric laterality of lesions and lobar involvement. Associated cognitive impairment act as a risk factor for psychosis but not been accounted in our study.

Personality changes are found more among frontal lesions but not statistically significant. van Reekum[4] reported that avoidant, borderline, and narcissistic personality disorders were the most common. In 60 patients assessed 30 years after TBI 23.3% had at least one personality disorder. The most prevalent were avoidant (15.0%), organic (15.0%), paranoid (8.3%), and schizoid (6.7%) personality disorders. Our study has found only organic personality disorder in about 6% of patients who have been presented both during the initial months and also chronically. Aggression and behavioural disinhibition acts very difficult for the caregivers to manage. Statistical difference was found between patients with personality changes and duration since injury with 2%cases found before 6months and 1%case after 6month and 3 cases diagnosed after 1 year. No statistical difference with hemispheric laterality of injury.

There have been studies with organic stupor or mutism after injury but there was no studies showing organic catatonia as per icd-10 classification and our study is the first one to have a finding on it. It had a incidence of 1% in our study. It has significance with duration since injury with the case found 1year after injury. And significant with the severity of lesion with the case found in patient with severe injury.

CONCLUSION

In the treatment of head injury, apart from the treatment of physical symptoms, It is also important to treat psychiatric symptoms because psychiatric comorbidity delays the social and functional outcome of the patient. Most of it had been missed by clinicians and it affects the prognosis of the patient, so it is essential to identify the risk factors, understanding the clinical features for better treatment of the patient.

Not a single can predict the risk of psychiatric comorbidity and also found it has no relationship with the severity and laterality of lesions consistently. Treatment of the illness involves a multi disciplinary approach with the psychiatrist in liaison with neurosurgeon and family . Treatment should follow from a clearly articulated diagnostic scheme and should be time-limited and re-evaluated in the presence of poor or incomplete response.

LIMITATIONS

- It is a cross sectional study-difficult to assess the course of illness over a period of time, which in case of a TBI is likely to change.
- Severity assessed only based on GCS score and loss of consciousness and post traumatic amnesia not accounted for in our study.
- Other risk factors and the relation between them and quality of life for them which have indirectly contributed to their depressive symptoms is missed.
- Lack of control group is a drawback of study.
- Post trauma substance use disorders not diagnosed.
- Depression associated with disability caused due to head injury rather than the primary organic cause can't be ruled out.
- Relationship between premorbid personality and the psychiatric diagnosis after injury can't be seen.

FUTURE DIRECTIONS

PSYCHOSIS:

Future research is necessary

1. To clarify the diagnostic criteria for psychosis due to traumatic brain injury. Specifically, this would require:

    a. To define and validate core symptoms that distinguish "psychoticism" from cognitive or perceptual disturbances. Because the negative symptoms may prove nonspecific or look similar in their diagnostic validity in the presence of amotivational frontal symptoms.

    b. To define a minimum and maximum time frame between injury and onset of psychosis. And a exclusionary criteria that would differentiate TBI-related psychoses from idiopathic psychotic disorders.

2. To identify genetic, neuropsychological, and clinical correlates that are predictive of the development of post-TBI psychosis.

3. To clarify the pathophysiological mechanism of posttraumatic psychosis compared with that of schizophrenia.

DEPRESSION

Future research is necessary to:

1. Diagnostic criteria: Develop standardized categorical and dimensional criteria for post-TBI depression that have clinical and discriminative validity.

2. Long-term effects of post-TBI depression: Describe the long-term psychosocial, functional, and physical impact of post-TBI depression, particularly the impact of early recognition and treatment on longitudinal outcome. Once standardized criteria for diagnosis are developed, both prospective and retrospective studies using case control methodologies could facilitate this objective.

3. Predictors of post-TBI depression: Identify biological, psychosocial, and cognitive risk factors for developing post-TBI depression. Such variables could include family history of mood disorder, preinjury psychological trauma, or post-TBI executive cognitive impairment.

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PROFORMA

PSYCHIATRIC MORBIDITY IN PATIENTS WITH TRAUMATIC BRAIN INJURY

NAME: AGE: SEX:

MARITAL STATUS: EDUCATION:

RELIGION:

ADDRESS:

PHONE NUMBER:

SOCIO ECONOMIC STATUS:

INFORMANT:

RELATIONSHIP WITH THE PATIENT:

FAMILY HISTORY:

COMORBID ILLNESS:

PAST HISTORY OF PSYCHIATRIC ILLNESS:

SUBSTANCE USE:

HEAD INJURY:

DURATION SINCE INJURY:

IMAGING STUDY:

CT-

MRI-

SCALES:

MINI PLUS-

Hamilton rating scale for depression (HAM-D)

Hamilton rating scale for anxiety (HAM-A)

Positive and negative syndrome scale (PANSS)

DSM V/ICD-10 for diagnosing personality disorders

MEDICATIONS ON:

PART OF BRAIN AFFECTED (SIDE/LOBE):

Information to Participants

Title: Psychiatric morbidity in patients following traumatic brain injury

Principal Investigator: Dr. KIRUTHIKA A

Name of Participant:

Site: Department of Neurosurgery, Madras Medical College, Chennai

You are invited to take part in this research. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

What is the purpose of research?

The prevalence of psychiatric disorders in those with traumatic brain injury is higher and it impairs their social life. If these comorbidities are not treated, it accounts for high morbidity. Timely intervention will improve the patient's condition in hopes of limiting disability.

We have obtained information from the Institutional Ethics committee

The study design

Study will be done at Department of Neurosurgery, Madras medical college. 100 patients who are physically stable, giving their consent will be taken up for the study. Prevalence of psychiatric disorders will be assessed in patients with traumatic brain injury.

Study Procedures

Study will include a population of patients diagnosed with traumatic brain injury attending outpatient and inpatient of Department of Neurosurgery. They are chosen for study if they are physically stable after taking an informed consent. Later, standard assessment tools, are used to assess the Prevalence of psychiatric disorder. The results are correlated with radiological imaging of the patient. Various data obtained will be analyzed statistically to get the results.

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Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel, Institutional Ethics Committee and any person or agency required by law like the Drug Controller General of India to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not loose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to discontinuing form the study.

Signature of Investigator Signature of Participant

Date Date

Signature of the witness

Date:

INFORMED CONSENT FORM

Title : " Psychiatric morbidity in patients following traumatic brain injury"

Name of the Participant:

_____.

Name of the Principal (Co-Investigator): Dr.KIRUTHIKA.A

Documentation of the informed consent

I _____ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in

"Psychiatric morbidity in patients following traumatic brain injury"

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.
7. I have not participated in any research study within the past _____month(s).
8. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
9. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
10. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
11. I have understand that my identity will be kept confidential if my data are publicly presented
12. I have had my questions answered to my satisfaction.
13. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____ Signature_____
Date_____

Name and Signature of impartial witness (required for illiterate patients):
Name _____ Signature_____
Date_____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:
Name _____ Signature_____
Date_____

ஆய்வு தகவல் தாள்

ஆய்வின் தலைப்பு : மூளை சிதைவு ஏற்பட்டவர்களின் மன நோயுற்ற தன்மை

ஆய்வாளரின் பெயர் : மரு.அ.கிருத்திகா

பங்கு கொள்பவரின் பெயர்

மருத்துவ நிலையம் : மூளை அறுவை சிகிச்சை பிரிவு
சென்னை மருத்துவகல்லூரி
சென்னை

ஆய்வின் நோக்கம்

மூளை சிதைவு ஏற்பட்டவர்களுக்கு மனநோயின் தாக்கம் அதிகமாக உள்ளது மற்றும் அது அவர்களின் சமூகவாழ்க்கையை பாதிக்கின்றது. இதை குணப்படுத்தாவிட்டால் அது நோயின் தாக்கத்தை அதிகரிக்கும். தகுந்த நேரத்தில் சிகிச்சை அளித்தால் இயலாமையை குணப்படுத்தலாம். இந்த ஆய்வு நடத்துவதற்கு நெறிமுறை குழுவிடம் இருந்து ஒப்புதல் பெறப்பட்டு இருக்கிறது.

செய்முறை விளக்கம்

மூளை சிதைவு ஏற்பட்டபவர்களுக்கு மனநோயுற்ற தன்மை தகுந்த கோல்கள் மூலம் அறியப்படும். இந்த தகவல் நோயாளிகளின் நரம்பு படவியலுடன் இணையுறவு செய்யப்படும்.

தகவலின் ரகசியத் தன்மை

தங்களுடைய சுய விளக்கம் மருத்துவ குறிப்புகள் மற்றும் மருத்துவ சோதனை அறிக்கை அனைத்தும் ரகசியமாக வைப்பதற்கும் தனிஉரிமை அளிக்கப்படும். இதன் முடிவுகளை வெளியிடும்போதோ அல்லது ஆய்வின் போதோ தங்களின் பெயரையோ அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆய்வில் தாங்கள் பங்கேற்காவிட்டாலும் தங்களுடைய மருத்துவ உதவியில் எந்த ஒரு பின்விளைவுகளும் ஏற்படாது.

இந்த ஆய்வில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பெயரில் தான் மேலும் நீங்கள் எந்த நேரமும் இந்த ஆய்வில் இருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறேன்.

ஆய்வாளரின் கையொப்பம்

நாள் :

பங்கேற்பாளர் கையொப்பம்

நாள் :

ஆய்வு ஒப்புதல் படிவம்

ஆய்வின் தலைப்பு : மூளை சிதைவு ஏற்பட்டவர்களின் மன நோயுற்ற தன்மை

ஆய்வாளரின் பெயர் : மரு.அ.கிருத்திகா

பங்கு கொள்பவரின் பெயர் :

மருத்துவ நிலையம் : மூளை அறுவை சிகிச்சை பிரிவு
சென்னை மருத்துவகல்லூரி
சென்னை

_____எனும் நான் எனக்கு கொடுக்கப்பட்ட தகவல் தாளினை படித்து புரிந்துகொண்டேன். நான் 18 வயதை கடந்திருப்பதால் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த ஆய்வில் என்னைச் சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

நான் எனக்கு கொடுக்கப்பட்ட தகவல் தாளினை படித்து புரிந்துகொண்டேன்.

எனக்கு இந்த ஆய்வின் ஒப்புதல் படிவம் விளக்கப்பட்டது.

எனக்கு இந்த ஆய்வின் நோக்கமும், விவரங்களும் விளக்கப்பட்டது.

எனக்கு என்னுடைய உரிமைகளை பற்றி விளக்கப்பட்டது.

நான் இதற்கு முன்பு எடுத்துக்கொண்ட அனைத்து மருத்துவ முறைகளைப் பற்றி தெரிவித்திருக்கிறேன்.

இந்த ஆய்வில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

என்னை பற்றிய எந்த தகவல்களும் அடையாளமும் வெளியிடப்படமாட்டாது என்பதை நான் புரிந்துகொண்டேன்

என்னுடைய முழு சுதந்திரத்துடன் இந்த ஆய்வில் என்னைச் சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

பங்கேற்பாளர் பெயர் மற்றும் கையொப்பம் ..... தேதி.....

ஆய்வாளரின் பெயர் மற்றும் கையொப்பம் ..... தேதி.....

THE HAMILTON RATING SCALE FOR DEPRESSION

(to be administered by a health care professional)

Patient's Name

Date of Assessment

To rate the severity of depression in patients who are already diagnosed as depressed, administer this

questionnaire. The higher the score, the more severe the depression.

For each item, write the correct number on the line next to the item. (Only one response per item)

1. DEPRESSED MOOD (Sadness, hopeless, helpless, worthless)

0= Absent

1= These feeling states indicated only on questioning

2= These feeling states spontaneously reported verbally

3= Communicates feeling states non-verbally—i.e., through facial expression, posture,

voice, and tendency to weep

4= Patient reports VIRTUALLY ONLY these feeling states in his spontaneous verbal and nonverbal

communication

2. FEELINGS OF GUILT

0= Absent

1= Self reproach, feels he has let people down

2= Ideas of guilt or rumination over past errors or sinful deeds

3= Present illness is a punishment. Delusions of guilt

4= Hears accusatory or denunciatory voices and/or experiences threatening visual

hallucinations

3. SUICIDE

0= Absent

1= Feels life is not worth living

2= Wishes he were dead or any thoughts of possible death to self

3= Suicidal ideas or gesture

4= Attempts at suicide (any serious attempt rates 4)

4. INSOMNIA EARLY

0= No difficulty falling asleep

1= Complains of occasional difficulty falling asleep—i.e., more than 1/2 hour

2= Complains of nightly difficulty falling asleep

5. INSOMNIA MIDDLE

0= No difficulty

1= Patient complains of being restless and disturbed during the night

2= Waking during the night—any getting out of bed rates 2 (except for purposes of voiding)

6. INSOMNIA LATE

0= No difficulty

1= Waking in early hours of the morning but goes back to sleep

2= Unable to fall asleep again if he gets out of bed

7. WORK AND ACTIVITIES

0= No difficulty

1= Thoughts and feelings of incapacity, fatigue or weakness related to activities; work or

hobbies

2= Loss of interest in activity; hobbies or work—either directly reported by patient, or

indirect in listlessness, indecision and vacillation (feels he has to push self to work or

activities)

3= Decrease in actual time spent in activities or decrease in productivity

4= Stopped working because of present illness

8. RETARDATION: PSYCHOMOTOR (Slowness of thought and speech; impaired ability

to concentrate; decreased motor activity)

0= Normal speech and thought

1= Slight retardation at interview

2= Obvious retardation at interview

3= Interview difficult

4= Complete stupor

9. AGITATION

0= None

1= Fidgetiness

2= Playing with hands, hair, etc.

3= Moving about, can't sit still

4= Hand wringing, nail biting, hair-pulling, biting of lips

10. ANXIETY (PSYCHOLOGICAL)

0= No difficulty

1= Subjective tension and irritability

2= Worrying about minor matters

3= Apprehensive attitude apparent in face or speech

4= Fears expressed without questioning

11. ANXIETY SOMATIC: Physiological concomitants of anxiety, (i.e., effects of autonomic

overactivity, "butterflies," indigestion, stomach cramps, belching, diarrhea, palpitations,

hyperventilation, paresthesia, sweating, flushing, tremor, headache, urinary frequency).

Avoid asking about possible medication side effects (i.e., dry mouth, constipation)

0= Absent

1= Mild

2= Moderate

3= Severe

4= Incapacitating

12. SOMATIC SYMPTOMS (GASTROINTESTINAL)

0= None

1= Loss of appetite but eating without encouragement from others. Food intake

about normal

2= Difficulty eating without urging from others. Marked reduction of appetite and

food intake

13. SOMATIC SYMPTOMS GENERAL

0= None

1= Heaviness in limbs, back or head. Backaches, headache, muscle aches. Loss of energy

and fatigability

2= Any clear-cut symptom rates 2

14. GENITAL SYMPTOMS (Symptoms such as: loss of libido; impaired sexual performance;

menstrual disturbances)

0= Absent

1= Mild

2= Severe

15. HYPOCHONDRIASIS

0= Not present

1= Self-absorption (bodily)

2= Preoccupation with health

3= Frequent complaints, requests for help, etc.

4= Hypochondriacal delusions

16. LOSS OF WEIGHT

A. When rating by history:

0= No weight loss

1= Probably weight loss associated with present illness

2= Definite (according to patient) weight loss

3= Not assessed

17. INSIGHT

0= Acknowledges being depressed and ill

1= Acknowledges illness but attributes cause to bad food, climate, overwork, virus, need

for rest, etc.

2= Denies being ill at all

18. DIURNAL VARIATION

A. Note whether symptoms are worse in morning or evening. If NO diurnal variation, mark none

0= No variation

1= Worse in A.M.

2= Worse in P.M.

B. When present, mark the severity of the variation. Mark “None” if NO variation

0= None

1= Mild

2= Severe

19. DEPERSONALIZATION AND DEREALIZATION (Such as: Feelings of unreality;

Nihilistic ideas)

0= Absent

1= Mild

2= Moderate

3= Severe

4= Incapacitating

20. PARANOID SYMPTOMS

0= None

1= Suspicious

2= Ideas of reference

3= Delusions of reference and persecution

21. OBSESSIONAL AND COMPULSIVE SYMPTOMS

0= Absent

1= Mild

2= Severe

Total Score _____

Hamilton Anxiety Rating Scale (HAM-A)

Below is a list of phrases that describe certain feeling that people have. Rate the patients by finding the answer which best describes the extent to which he/she has these conditions. Select one of the five responses for each of the fourteen questions.

0 = Not present, 1 = Mild, 2 = Moderate, 3 = Severe, 4 = Very severe.

---

1 Anxious mood [0] [1] [2] [3] [4]

Worries, anticipation of the worst, fearful anticipation, irritability.

2 Tension [0] [1] [2] [3] [4]

Feelings of tension, fatigability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.

3 Fears [0] [1] [2] [3] [4]

Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.

4 Insomnia [0] [1] [2] [3] [4]

Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors.

5 Intellectual [0] [1] [2] [3] [4]

Difficulty in concentration, poor memory.

6 Depressed mood [0] [1] [2] [3] [4]

Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.

7 Somatic (muscular) [0] [1] [2] [3] [4]

Pains and aches, twitching, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone.

8 Somatic (sensory) [0] [1] [2] [3] [4]

Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation.

9 Cardiovascular symptoms [0] [1] [2] [3] [4]

Tachycardia, palpitations, pain in chest, throbbing of vessels, fainting feelings, missing beat.

10 Respiratory symptoms [0] [1] [2] [3] [4]

Pressure or constriction in chest, choking feelings, sighing, dyspnea.

11 Gastrointestinal symptoms [0] [1] [2] [3] [4]

Difficulty in swallowing, wind abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation.

12 Genitourinary symptoms [0] [1] [2] [3] [4]

Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.

13 Autonomic symptoms [0] [1] [2] [3] [4]

Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.

14 Behavior at interview [0] [1] [2] [3] [4]

Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, etc.

POSITIVE AND NEGATIVE SYNDROME SCALE (PANSS) RATING CRITERIA

GENERAL RATING INSTRUCTIONS

Data gathered from this assessment procedure are applied to the PANSS ratings. Each of the 30 items is accompanied by a specific definition as well as detailed anchoring criteria for all seven rating points. These seven points represent increasing levels of psychopathology, as follows:

1- absent

2- minimal

3- mild

4- moderate

5- moderate severe

6- severe

7- extreme

In assigning ratings, one first considers whether an item is at all present, as judging by its definition. If the item is absent, it is scored 1, whereas if it is present one must determine its severity by reference to the particular criteria from the anchoring points. The highest applicable rating point is always assigned, even if the patient meets criteria for lower points as well. In judging the level of severity, the rater must utilise a holistic perspective in deciding which anchoring point best characterises the patient's functioning and rate accordingly, whether or not all elements of the description are observed.

The rating points of 2 to 7 correspond to incremental levels of symptom severity:

- A rating of 2 (minimal) denotes questionable or subtle or suspected pathology, or it also may allude to the extreme end of the normal range.
- A rating of 3 (mild) is indicative of a symptom whose presence is clearly established but not pronounced and interferes little in day-to-day functioning.
- A rating of 4 (moderate) characterises a symptom which, though representing a serious problem, either occurs only occasionally or intrudes on daily life only to a moderate extent.
- A rating of 5 (moderate severe) indicates marked manifestations that distinctly impact on one's functioning but are not all-consuming and usually can be contained at will.
- A rating of 6 (severe) represents gross pathology that is present very frequently, proves highly disruptive to one's life, and often calls for direct supervision.
- A rating of 7 (extreme) refers to the most serious level of psychopathology, whereby the manifestations drastically interfere in most or all major life functions, typically necessitating close supervision and assistance in many areas.

Each item is rated in consultation with the definitions and criteria provided in this manual. The ratings are rendered on the PANSS rating form overleaf by encircling the appropriate number following each dimension.

PANSS RATING FORM

		absent	minimal	mild	moderate	moderate severe	severe	extreme
P1	Delusions	1	2	3	4	5	6	7
P2	Conceptual disorganisation	1	2	3	4	5	6	7
P3	Hallucinatory behaviour	1	2	3	4	5	6	7
P4	Excitement	1	2	3	4	5	6	7
P5	Grandiosity	1	2	3	4	5	6	7
P6	Suspiciousness/persecution	1	2	3	4	5	6	7
P7	Hostility	1	2	3	4	5	6	7
N1	Blunted affect	1	2	3	4	5	6	7
N2	Emotional withdrawal	1	2	3	4	5	6	7
N3	Poor rapport	1	2	3	4	5	6	7
N4	Passive/apathetic social withdrawal	1	2	3	4	5	6	7
N5	Difficulty in abstract thinking	1	2	3	4	5	6	7
N6	Lack of spontaneity & flow of conversation	1	2	3	4	5	6	7
N7	Stereotyped thinking	1	2	3	4	5	6	7
G1	Somatic concern	1	2	3	4	5	6	7
G2	Anxiety	1	2	3	4	5	6	7
G3	Guilt feelings	1	2	3	4	5	6	7
G4	Tension	1	2	3	4	5	6	7
G5	Mannerisms & posturing	1	2	3	4	5	6	7
G6	Depression	1	2	3	4	5	6	7
G7	Motor retardation	1	2	3	4	5	6	7
G8	Uncooperativeness	1	2	3	4	5	6	7
G9	Unusual thought content	1	2	3	4	5	6	7
G10	Disorientation	1	2	3	4	5	6	7
G11	Poor attention	1	2	3	4	5	6	7
G12	Lack of judgement & insight	1	2	3	4	5	6	7
G13	Disturbance of volition	1	2	3	4	5	6	7
G14	Poor impulse control	1	2	3	4	5	6	7
G15	Preoccupation	1	2	3	4	5	6	7
G16	Active social avoidance	1	2	3	4	5	6	7

s.no	age	sex	socioeconomic	op/ip	subsance use	education	marital status	family history	injury	duration	side of brain affected	lobe affected	GCS	positive	negative	general psychopathology	HAM D	HAM A	DSM V(personality)	ICD 10
1	33	m	l	ip	use	nil	married	nil	RTA	1	bilateral	f,t,o	2	7	7	22	4	4		
2	30	m	l	op	nil	nil	married	nil	RTA	4	left	t,p	2	14	10	24	5	5		
3	29	m	l	ip	nil	nil	married	nil	RTA	1	right	f	2	8	7	20	2	2	yes	
4	45	f	l	ip	nil	primary	married	nil	fall	1	left	f,t,p	2	10	7	17	5	16		
5	57	m	l	ip	use	nil	married	nil	RTA	1	left	f,t,p	2	7	8	25	4	6		
6	19	m	l	ip	nil	primary	unmarried	nil	RTA	1	right	t,p	2	8	7	18	5	7		
7	32	m	l	ip	nil	nil	married	nil	RTA	1	left	f,t,p	2	9	7	20	6	3		
8	45	m	l	ip	nil	nil	married	nil	RTA	1	left	t	2	12	9	21	7	2		
9	45	m	l	ip	nil	primary	married	nil	RTA	1	right	t	1	13	7	16	6	4		
10	35	m	l	ip	nil	nil	married	nil	RTA	1	right	p	1	9	7	18	5	3		
11	38	m	l	ip	nil	degree	married	nil	RTA	1	right	p	1	8	7	19	8	4		
12	40	m	l	ip	nil	nil	married	nil	RTA	1	right	t	2	10	8	20	4	11		
13	42	m	l	ip	nil	nil	married	nil	RTA	4	left	f,t,p	2	8	7	19	2	3		
14	30	m	l	ip	nil	degree	married	nil	TTA	3	right	f,t,p	3	12	9	22	3	6		organic catatonia
15	36	m	l	ip	use	nil	married	nil	assault	1	left	f,t	1	10	7	23	1	8	yes	
16	75	f	l	ip	nil	primary	married	nil	RTA	1	bilateral	f	2	7	9	27	16	2		
17	45	f	l	ip	nil	nil	married	nil	RTA	1	left	f, BG	2	8	8	26	17	4		
18	47	m	l	ip	nil	nil	married	nil	RTA	1	right	f	2	9	7	21	9	12		
19	44	f	l	ip	nil	primary	married	nil	RTA	1	left	t	1	19	12	20	5	6		
20	41	m	l	ip	use	primary	married	nil	RTA	1	right	f	1	11	9	18	3	8		
21	65	m	l	ip	nil	primary	married	nil	fall	1	bilateral	f,t,p	2	9	8	25	20	15		
22	37	m	l	ip	nil	primary	married	nil	RTA	1	right	t	1	8	9	18	4	8		
23	38	m	l	op	nil	nil	married	nil	RTA	3	left	f,p	1	12	10	19	4	7	yes	
24	33	m	l	ip	nil	nil	married	nil	RTA	1	left	p	1	7	11	20	5	7		
25	38	m	l	ip	nil	degree	married	nil	RTA	1	left	f,t	2	9	8	17	2	6		
26	32	m	l	ip	nil	nil	married	nil	RTA	1	left	f,p	1	7	9	18	5	8		
27	30	m	l	ip	nil	primary	married	nil	RTA	1	bilateral	f,p	2	8	10	20	12	5		
28	29	m	l	ip	nil	primary	married	nil	RTA	1	left	t,p	1	11	12	21	4	9		
29	43	m	l	ip	nil	nil	married	yes	RTA	1	left	t,p	3	22	9	19	3	12		
30	38	m	l	ip	use	primary	married	nil	RTA	1	left	f	2	10	10	19	4	6		
31	46	f	l	ip	nil	primary	married	nil	RTA	1	bilateral	f,t	2	8	7	22	8	4		
32	49	f	l	ip	nil	nil	married	nil	RTA	1	left	f,t	1	7	9	20	6	9		
33	45	f	l	ip	nil	nil	married	nil	RTA	2	right	o	2	10	8	19	3	17		
34	37	f	l	ip	nil	primary	married	nil	RTA	1	left	f,p	2	11	13	17	4	7		
35	42	f	l	ip	nil	primary	married	nil	RTA	1	bilateral	o	1	8	11	19	5	14		
36	34	m	l	ip	nil	primary	married	nil	RTA	1	right	t,o	2	16	7	21	2	3		
37	47	m	l	ip	nil	primary	married	nil	RTA	1	right	f	2	9	9	18	6	8		
38	22	m	l	ip	use	nil	unmarried	nil	RTA	2	bilateral	f	2	14	8	19	5	11	yes	
39	48	f	l	ip	nil	nil	married	nil	RTA	1	right	f,p	2	11	7	20	7	10		
40	39	m	l	ip	use	primary	married	nil	RTA	1	left	t	2	10	11	21	9	8		
41	20	m	l	ip	use	nil	unmarried	nil	RTA	1	right	f	1	8	10	19	3	9		
42	35	f	l	ip	nil	primary	married	nil	RTA	2	right	t,p	2	9	12	17	5	16		
43	47	m	l	ip	nil	nil	married	nil	RTA	1	right	f,t	1	7	7	21	7	8		
44	35	m	l	ip	use	nil	married	nil	RTA	1	left	f,BG	2	7	8	18	14	8		
45	42	f	l	ip	nil	primary	married	nil	RTA	1	left	p	1	7	19	17	6	9		
46	42	f	l	ip	nil	nil	married	nil	RTA	2	left	p	2	8	10	19	4	16		
47	33	m	l	ip	nil	nil	married	nil	RTA	1	right	f	1	10	7	21	5	7		
48	37	m	l	ip	nil	nil	married	nil	RTA	1	right	p	1	7	7	20	4	5		
49	66	m	l	ip	use	nil	married	yes	fall	1	left	f,p	3	8	14	28	21	16		
50	61	m	l	ip	nil	nil	married	nil	fall	1	right	f	2	7	9	19	5	4		
51	36	m	l	ip	nil	degree	married	nil	RTA	1	right	t	2	10	15	18	5	5		
51	47	m	l	ip	use	nil	married	nil	RTA	1	left	f	2	7	8	20	4	3		
53	58	m	l	ip	nil	nil	married	nil	RTA	1	left	t	2	8	9	21	5	5		
54	38	f	l	ip	nil	primary	married	nil	RTA	1	left	f	2	10	8	27	17	7		
55	51	f	l	ip	nil	nil	married	nil	RTA	1	right	o	1	7	7	19	6	4		
56	58	f	l	ip	nil	nil	married	nil	RTA	1	right	f,p	2	8	9	18	4	14		
57	57	m	l	ip	use	nil	married	nil	RTA	1	left	f,p	2	9	7	17	8	9		
58	33	m	l	ip	nil	primary	married	nil	RTA	1	right	t,o	2	7	9	22	7	10		

59	38	m	l	ip	nil	primary	married	nil	RTA	1	right	t,p	1	7	7	21	5	6		
60	44	f	l	ip	nil	primary	married	nil	RTA	1	bilateral	f,p	1	7	11	20	9	8		
61	43	m	l	ip	use	primary	unmarried	nil	RTA	3	bilateral	f	2	12	9	18	6	9	yes	
62	49	m	l	ip	nil	primary	married	nil	RTA	1	left	p	2	9	8	17	7	5		
63	58	m	l	ip	nil	primary	married	nil	RTA	1	bilateral	f,t,p	3	8	12	25	19	17		
64	52	f	l	ip	nil	primary	married	nil	RTA	1	right	f	1	10	9	17	6	4		
65	61	m	l	ip	nil	nil	married	nil	fall	1	left	f	2	8	7	16	5	7		
66	58	m	l	ip	nil	nil	married	nil	RTA	1	right	f,p	2	10	14	18	12	4		
67	55	f	l	ip	nil	nil	married	nil	RTA	1	right	t,p	1	8	9	17	4	6		
68	21	m	l	ip	use	primary	unmarried	nil	RTA	1	left	f	2	8	8	21	5	3		
69	36	m	l	ip	nil	primary	married	nil	RTA	2	right	f	2	9	9	22	3	15		
70	44	m	l	ip	nil	nil	married	nil	RTA	1	left	t,p	2	7	8	16	2	5		
71	28	m	l	ip	use	primary	married	nil	assault	3	right	f	1	13	10	18	6	8	yes	
72	39	f	l	ip	nil	primary	married	nil	RTA	1	left	p	2	10	7	17	14	6		
73	34	m	l	ip	nil	primary	married	nil	RTA	2	left	f	2	8	9	16	4	16		
74	67	m	l	ip	nil	primary	married	nil	RTA	1	left	f,t	2	7	8	22	3	12		
75	33	f	l	ip	nil	primary	married	nil	RTA	1	left	f	2	7	20	21	9	4		
76	49	m	l	ip	nil	nil	married	nil	RTA	1	right	f,p	1	8	7	16	4	10		
77	35	m	l	ip	nil	nil	married	nil	RTA	1	bilateral	f	1	10	9	17	8	5		
78	29	f	l	ip	nil	degree	unmarried	nil	RTA	1	right	f	1	9	11	16	3	3		
79	34	m	l	op	use	nil	married	nil	RTA	3	left	f,p	2	7	10	19	11	5		
80	42	m	l	ip	nil	primary	married	nil	RTA	1	right	f,t,p	3	9	8	18	6	17		
81	39	m	l	ip	nil	primary	married	nil	RTA	1	left	f,t	1	8	9	16	4	7		
82	72	f	l	ip	nil	primary	married	nil	fall	1	right	f,p	2	7	7	18	10	4		
83	44	f	l	ip	nil	primary	married	nil	RTA	1	right	f	1	20	11	17	2	5		
84	57	m	l	ip	use	nil	married	nil	RTA	3	right	f	2	7	9	16	5	8		
85	32	m	l	op	nil	nil	married	nil	RTA	3	left	f	2	8	9	17	4	6		
86	46	f	l	ip	nil	nil	married	nil	RTA	1	left	f,t	2	8	7	19	7	9		
87	44	m	l	ip	nil	nil	married	nil	RTA	1	left	f	2	10	7	20	5	6		
88	57	m	l	ip	nil	nil	married	nil	RTA	1	bilteral	f,t,p	3	8	10	18	17	7		
89	42	f	l	ip	nil	nil	married	nil	RTA	1	left	f	2	7	8	16	12	5		
90	41	m	l	ip	nil	nil	married	nil	RTA	1	right	f	1	7	9	17	3	8		
91	36	m	l	ip	nil	nil	married	nil	RTA	3	left	t,o	2	7	13	19	13	5		
92	34	f	l	ip	nil	nil	married	nil	RTA	3	left	f,t	1	7	24	18	2	12		
93	33	m	l	ip	nil	nil	married	nil	RTA	1	left	f	2	8	9	17	10	4		
94	28	m	l	ip	use	nil	unmarried	nil	RTA	1	right	f	1	7	12	20	5	7		
95	65	f	l	ip	nil	nil	married	nil	RTA	2	left	f,p	2	7	9	23	14	15		
96	33	m	l	ip	use	degree	married	nil	RTA	1	right	f,t	1	7	11	16	6	6		
97	39	m	l	ip	nil	primary	married	nil	RTA	1	right	f	1	7	10	18	9	5		
98	31	m	l	ip	use	nil	married	nil	RTA	1	right	f	1	7	9	17	4	12		
99	38	f	l	ip	nil	degree	married	nil	RTA	1	left	f	2	8	8	22	15	6		
100	27	m	l	ip	nil	primary	unmarried	yes	RTA	1	left	t	1	7	9	16	5	3		